This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at http://books.google.com/

COUNTWAY LIBRARY
HC 32L1 D

LIBRARY OF
S. K. HARRIMAN, M. D
NATICK.

A.G. 1891

Harvard University Med
School

LIBRARY OF THE
PATHOLOGICAL LABORA
THE GIFT OF
Miss H. J. Ewing

TEXT-BOOK

OF THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

Cl Mel 1891.2

Presented by Miss H. J. Ewing.

TEXT-BOOK

OF THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

BY THE LATE

CHARLES HILTON FAGGE, M.D., F.R.C.P.

PHYSICIAN TO, AND LECTURER ON PATHOLOGY IN, GUY'S HOSPITAL; EXAMINER IN MEDICINE IN THE UNIVERSITY OF LONDON

AND

PHILIP HENRY PYE-SMITH, M.D., F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, PHYSICIAN TO, AND LECTURER ON MEDICINE IN, GUY'S HOSPITAL

Third Edition

VOL. I

LONDON

J. & A. CHURCHILL

11, NEW BURLINGTON STREET

1891

6.G. 1891.1

PREFACE.

THE First Edition of the present work, published in January, 1886, consisted of the MS. left by the late Dr Fagge, arranged and edited by the present writer, with numerous additions in the Second Volume, of which the most important were some sections on the Diseases of the Heart (by Dr Wilks) and all the chapters on Diseases of the Skin.

In the Second Edition, published in 1888, the whole book was revised: some parts (chiefly transcriptions of cases from other authors) were omitted, and the following chapters were added or re-written —those on Pyæmia, Enteric Fever, Febricula, Vaccinia, Rubeola, Actinomycosis, Peripheral Neuritis, Friedreich's and Thomsen's Diseases, Insular Sclerosis, and the chapter on Insanity by Dr Savage; while large additions were made to the sections on Diseases of the Heart, Pneumonia, Intestinal Obstruction, Diseases of the Liver, Anæmia, and Diseases of the Joints.

In the present Edition every page has been carefully revised; and, by occasional excision and more frequent compression, room has been made for such additions as the progress of medicine seemed to render desirable.

The Introductory Chapter has been re-written, and the Pathological Section which follows has been re-arranged and added to. The treatment of Enteric Fever and Diphtheria, and the pathology of Cholera and Ague, have been considerably expanded.

The great advances made in our knowledge of the anatomy and diagnosis of Diseases of the Nervous System led to considerable alterations and additions in this part of the book in the Second Edition, and similar though less extensive changes will be found in the present one.

The chapters on the Diseases of the Heart, which were something of a patchwork, have been revised and in great part re-written, so as to make as complete and homogeneous an account of this important subject as the writer's ability and the space which it seemed reasonable to assign to this section would allow.

The chapter on Phthisis has been condensed in some parts, and expanded in others. It had been already sent to press some time before the publication (at once too late and too early) of the method of treatment by inoculation introduced by the eminent discoverer of the essential nature of this disease, as well as (in all probability) of that of Cholera. The circumstances, however, of the announcement and practice of this method were such, and the disappointment of the

hopes at first existed has been so great, that the omission is perhaps not to be regretted.

The chapter on Influenza has been amplified from a historical compilation to a description based on experience of the epidemic of the last two years.

The additions and corrections in the sections on Diseases of the Alimentary Tract, of the Liver and of the Kidneys have been numerous, but on the whole less important than in the Second Edition. In the chapter on Diabetes an attempt has been made to present the problems involved from a point of view somewhat different from that taken by Dr Fagge, and perhaps more in accordance with recent physiological knowledge. The time, however, has not yet come for a satisfactory theory of this interesting and tantalising region of pathology.

The subject of Anæmia had been re-cast in the Second Edition, and the plan then followed has been adhered to, with the addition of some fresh clinical and pathological facts.

The chapters on Gout and Rheumatism, on Gonorrhœal Synovitis and Osteo-arthritis, and that on Rickets, have been expanded in several particulars.

Lastly, the section devoted to Diseases of the Skin, for which the writer was entirely responsible in the First Edition, has now been subjected to careful revision and correction, and such additions have been made as will, it is hoped, render it more complete than before.

New Indices, both of subjects and authors, have been constructed for this Edition, and, by a somewhat different arrangement of material, room has been gained for introducing the former one into the First Volume; so that each volume now contains a complete Index to the subject-matter of both.

I must again thank various correspondents who have favoured me with criticisms or references to new facts, and reviewers in home and foreign journals for their friendly and useful comments.

Special acknowledgments are due to my friend and colleague Dr L. E. Shaw, and to our late Medical Registrar at Guy's Hospital, Dr E. W. Goodall, for the help repeatedly given in statistical work.

Lastly, I must express my deep obligation to my friend Dr Cavafy, of St George's Hospital, who most kindly undertook the tedious task of reading the proof sheets, and to whose keen eye and accurate knowledge I am indebted for the correction of many errors.

P. H. PYE-SMITH.

HARLEY STREET;
July, 1891.

CONTENTS OF THE FIRST VOLUME

PAGES

INTRODUCTORY CHAPTER

Definition of medicine and of disease—Nosology: organic and functional diseases—Pathological anatomy and clinical characters as bases of nosology—Diagnosis—Symptoms and diseases—Symptoms and physical signs—General ætiology: exciting and predisposing causes of disease—General prognosis and therapeutics—Arrangement of the present work . . . 1—10

INFECTION

Transmission of diseases—Contagion and miasm—Theory of infection—Contagium vivum—Microbes—Their origin and life—Their exclusive action in producing specific diseases—Their mode of action—Immunity from contagion—Protective inoculation—Modes of transference of miasmata and contagia—Theory and practice of disinfection . . . . . 11—26

FEVER

Pyrexia—Thermometry—Clinical course of pyrexia—Hyperpyrexia—Theory of fever—Possibly beneficial effect of pyrexia—Idiopathic and symptomatic fever—Raised temperature without fever—The physiological stages of fever—Concomitant phenomena of pyrexia: nutrition: pulse: respiration: muscles—Subnormal temperature . . . . . 27—42

INFLAMMATION

Historical review: Virchow: William Addison, Waller and Cohnheim: Goodsir—Direct observation of inflammation in animals—Redness, swelling, pain, heat, pyrexia—Exudation: membranous, catarrhal, purulent, diphtheritic, gangrenous inflammation—Repair—Causes of inflammation: irritants and predisposition—Chronic inflammation—Granuloma . . . 43—60

PYÆMIA

Theories of pyæmia—Metastasis—Phlebitis—Septicæmia—Embolism—Infection—Microphytes—Practical application—External or traumatic and internal pyæmia—Channels of infection—Local distribution of secondary abscesses—Idiopathic pyæmia—Slighter forms—Diagnosis: especially from enteric fever and tuberculosis—Prognosis—Treatment . . . . 61—69

TUMOURS

New growths—Innocent and malignant tumours—Virchow's characters of malignancy—Malignancy not an attribute of structure—Histological classification of tumours—Malignant osteoid tumour—Sarcoma: round, spindle, and giant cells; hæmorrhagic and alveolar sarcoma: melanotic sarcoma—Multiple sarcomata as a general disorder—Osteitis deformans—Lymphoma—Carcinoma—Heredity—Theory of new growths . . . 70—97

PAGES

SPECIFIC INFECTIOUS DISEASES

AFFECTING THE WHOLE BODY AND ACCOMPANIED BY FEVER

Nosology — Bases of classification — General and local diseases — Specific fevers—Tabular conspectus . . . . . 98—102

TYPHUS

History, nomenclature, and geographical distribution—Incubation—Course: first week, exanthem; second week, crisis and convalescence: death and post-mortem appearances—Complications and sequelæ—Ætiology; contagion of typhus: its supposed spontaneous origin—Diagnosis—Prognosis—Treatment . . . . . 103—123

RELAPSING FEVER

History, nomenclature, and distribution—Incubation, onset and course—Relapse—Convalescence — Death and morbid anatomy — The spirillum—Famine as a predisposing cause—Diagnosis—Prognosis—Treatment 124—133

ENTERIC FEVER

History of the recognition of enteric fever and of its distinction from typhus—Its extent—Nomenclature—Incubation—Clinical course and symptoms—Recovery—Abortive cases—Fatal cases—Complications—Sequelæ—Relapses—Protection—Diagnosis — Morbid anatomy—Ætiology—A contagious disease, always produced from a previous case—Methods of contagion: by air, water, &c.—Predisposing causes: soil-water, climate, age — Prognosis — Treatment—Food, stimulants, drugs—Cold baths and antipyretics—Treatment of other symptoms—Convalescence—FEBRICULA . . 134—176

MEASLES

History and nomenclature—Ætiology—Incubation—Onset—Course and eruption—Varieties—Complications—Sequelæ—Protection—Diagnosis — Prognosis—Treatment . . . . . 177—183

SCARLATINA

History and nomenclature—Distribution—Contagion and transference—Surgical and puerperal scarlatina—Incubation—Onset and course—The throat—The rash—Abortive and malignant varieties; scarlatina anginosa—Complications and sequelæ — Anatomy—Prognosis — Protection — Prophylaxis—Treatment . . . . . 184—193

RUBEOLA

Recognition and nomenclature—Its characteristics—Discrepancies and probable explanation—Incubation—Onset and course—Diagnosis — Protection—Prognosis and treatment . . . . . 194—197

SMALLPOX

Name and history—Contagion and spread—Incubation—Onset—Early rashes—Specific eruption—Varieties of the exanthem—Course and symptoms—Discrete, confluent, and modified smallpox—Complications and sequelæ—Prognosis and treatment.

VACCINIA—Inoculation—Introduction of vaccination—Course of the eruption—Its protective power—Its drawbacks—Its relation to variola . 198—213

VARICELLA

History of the recognition of chicken-pox—Its distinction from smallpox—Incubation and onset—Characters of the eruption—Symptoms and course—Sequelæ—A disease of children—Diagnosis—Prognosis . . 214—218

THE PLAGUE

History and geographical distribution—Incubation, onset, and symptoms—Diagnosis—Anatomy—Fatality—Treatment—Conditions of contagion and causes of the decline of the plague.

THE SWEATING SICKNESS—History—Symptoms and Pathology . 219—224

PAGES

CHOLERA

History—Name—Onset and early symptoms—Stage of collapse—Stage of reaction: the urine at this stage—So-called "cholera-typhoid"—Cholerine—Complications—Post-mortem conditions—Pathology—Ætiology—Theory of contagion—The Comma Bacillus—Propagation of the disease—Diagnosis—Prognosis—Treatment . . . . . 225—245

ERYSIPELAS

Definition of the disease—Its contagion—A specific febrile exanthem—Microbe—Relation of the phlegmonous form to facial idiopathic erysipelas—Incubation and onset—The eruption—Its histology—The symptoms and course——Recurrent erysipelas—Complications—Sequelæ—Diagnosis—Prognosis—Treatment . . . . . 246—255

DIPHTHERIA

History of the recognition of the disease—Its definition—Its anatomy and pathology—Relation to croup and to "diphtheritic inflammation"—Microbes—Ætiology—Contagion—Course and symptoms—Varieties—Complications and sequelæ—Albuminuria—Paralysis—Prognosis—Treatment . 256—275

SYPHILIS

History of the disease—Its present extent—Nomenclature—Its place among specific exanthems—Origin and incubation—Primary lesion—Infecting and soft sores—Premonitory stage—Secondary lesions of skin, throat and mouth, eyes, &c.—Tertiary lesions of skin, tongue, palate, bones, and testes—Diagnosis—Conditions of infection—Special questions in syphilitic pathology—Prognosis and protection—Treatment.

Congenital syphilis—Its transmission to the child—Syphilitic placenta—Local manifestations—Its further transmission—Its treatment—Later effects 276—304

TUBERCULOSIS

Theory of tuberculosis—Anatomy and histology of tubercle: views of Laennec, Bayle, and Louis: of Virchow, Schüppel, and Ziegler—Tubercle a product of modified inflammation—Infiltrating tubercle—Caseous and fibroid transformations of tubercle—Ætiology—Koch's tubercle-bacillus—Result of experiments on animals: inoculation of tubercle: Perlsucht: infection by the mouth and by the lungs—Predisposing causes—Invasion and spread of tubercle in the body—Course of acute tuberculosis—Varieties of chronic tuberculosis: phthisis pulmonum, laryngis et ilei; tuberculous inflammation of serous membranes; genito-renal tuberculosis; Addison's disease; tuberculosis of the lymph-glands and spleen; tuberculous disease of joints: multiple tubercle generally—Concluding historical retrospect . 305—321

AGUE

Intermittent fever—Its pathology, history, and distribution—Incubation—The cold, hot, and sweating stages—Regular and irregular varieties—Remittent fever—Malarial cachexia—The spleen, liver, and other organs in ague—Melansemia—Nature and laws of malarious contagion—Microphytes and microzoa—Diagnosis—Treatment . . . . . 322—338

YELLOW FEVER

History—Incubation, course, and events—Morbid anatomy—Ætiology and pathology—Question of its contagious or miasmatic nature—Diagnosis—Prognosis—Preventive and curative treatment . . . 339—345

DENGUE

History—Course and symptoms—The fever and the interval—The exanthem—Sequelæ—Pathology—Contagion—Diagnosis—Prognosis—Treatment 346—349

PAGES

EPIZOOTIC DISEASES TRANSFERRED TO MAN

ANTHRAX—Nomenclature—Distribution—Varieties in animals—The bacillus—Modes of infection—Varieties in man: charbon, intestinal and thoracic anthrax, their anatomy, prognosis, and treatment—Pathology—Prophylaxis.

GLANDERS—History and nomenclature—Origin in horses—The bacillus—Transfer of contagium—Acute form—eruption, ozæna, pyrexia, fatality—Chronic form—its treatment—Anatomy.

FOOT-AND-MOUTH DISEASE—An epidemic disorder among cattle—Bacillus—Its conveyance to man—Its symptoms, diagnosis, and treatment.

ACTINOMYCOSIS—Its occurrence in cattle—in the human subject—The microphyte—its transference—Pathology—Varieties and course—Treatment.

HYDROPHOBIA—Incubation, symptoms, course, and event—The disease in animals—Its ætiology, anatomy, and pathology—Diagnosis—Treatment—Prophylaxis by Pasteur's method of inoculation . . 350—379

DISEASES OF THE NERVOUS SYSTEM

Local or regional diseases compared with general and specific diseases—Principles of their classification and nomenclature—Order followed.

Nervous diseases—Peculiarities of this group—Arrangement adopted . 380—382

AFFECTIONS OF THE NERVE-TRUNKS

NEURALGIA—Definition—Pathology and general characters—Trifacial neuralgia—Neuralgia of arm and trunk—Sciatica—Treatment of neuralgia.

PERIPHERAL PARALYSIS—Causes—Motor palsies of vertebral nerves in particular—The reaction of degeneration—Sensory paralysis—Pain—Causalgia—Glossy skin and other trophic results—Diagnosis, prognosis, treatment—Paralysis of cranial nerves—Motor, of the portio dura, hypoglossal, and oculomotor—Sensory, of the trifacial and olfactory—Treatment.

PERIPHERAL NEURITIS—Its history and symptoms—Histology—Causes—Prognosis and treatment . . . . . 383—426

AFFECTIONS OF THE SPINAL CORD

PARAPLEGIA

Arrangement of diseases of the cord proposed.

Symptoms of paraplegia—Localisation—Sensory symptoms—Physiological interpretation of symptoms—Incomplete paraplegia—Reflex movements—Knee-jerk and clonus—Muscular atrophy and rigidity—The bladder and urine in paraplegia—The rectum and genitalia—Bedsores.

INTRINSIC PARAPLEGIA—Myelitis—Pathology and histology of acute myelitis—Of chronic myelitis or sclerosis—Ætiology—Course and symptoms of acute myelitis—Prognosis—Diagnosis—Treatment.

Paraplegia due to hæmorrhage and tumours within the cord—Anæmia and congestion of the cord, Divers' palsy—Concussion of the cord.

Acute ascending paralysis of Landry—Alcoholic and syphilitic paraplegia.

Reflex and hysterical paraplegia—Neurasthenia spinalis—Rachialgia.

HEMIPARAPLEGIA—Its pathology and symptoms, origin and course.

PAGES

EXTRINSIC PARAPLEGIA—Compression of the cord from vertebral caries or cancer—from aneurysm, hydatid cyst, or meningeal tumour—Symptoms, diagnosis, prognosis, and treatment of paraplegia from compression.

SPASTIC PARAPLEGIA—In adults—In children—Primary and secondary—Anatomy—Mixed forms—Symptoms—Prognosis and treatment—Cases.

MENINGEAL AFFECTIONS—Hæmorrhage—Acute and chronic spinal meningitis. 427—487

PARALYSIS ATTENDED WITH MUSCULAR WASTING

General characters of this group of diseases of the cord.

SUDDEN ATROPHIC SPINAL PARALYSIS—In infants—Its course, sequelæ, anatomy, and treatment—In adults.

SUBACUTE ATROPHIC SPINAL PARALYSIS—Its course and anatomy.

CHRONIC ATROPHIC PARALYSIS—PROGRESSIVE MUSCULAR ATROPHY—Symptoms—Pathology—Primary and secondary forms—Spastic atrophic paralysis—Pachymeningitis cervicalis—Diagnosis—Treatment.

BULBAR PARALYSIS—Progressive muscular atrophy in children—Hereditary form.

PSEUDO-HYPERTROPHIC PARALYSIS—Primary (myopathic) muscular atrophy.

LEAD PALSY—Distribution—Diagnosis—Tremors—Treatment.

Summary of other results of plumbism—Effects upon the nervous system of other metallic poisons—Mercurial tremors, brassfounders' ague, &c. 488—523

TABES

Histology and nomenclature—Ataxic symptoms—Anæsthesia—Paraplegic symptoms—Lightning-pains—Visceral pains—Loss of knee-jerk—Disorders of the eyes—Atrophy of joints—Perforating ulcer—Other complications—Course and event—Anatomical seat and histology of tabes—Relation between the ataxy, dysæsthesia, and other symptoms—Ætiology—Diagnosis—Prognosis and treatment.

Hereditary tabes: Friedreich's disease, its symptoms and pathology 524—540

INSULAR SCLEROSIS

History—Anatomy—Symptoms—Ætiology—Diagnosis—Prognosis and treatment—Comparative frequency of diseases of the spinal cord in hospital practice . . . . . 541—547

DISEASES OF THE BRAIN

CEREBRAL DISORDERS DUE TO ARTERIAL LESIONS

Anatomical effects of obstruction of the cerebral arteries by embolism, thrombosis, syphilis, red and white softening—Effects of hæmorrhage—Renal disease, atheroma, miliary aneurysms, as causes of cerebral hæmorrhage.

HEMIPLEGIA—Its characters and extent—The local lesion—Course and sequelæ.

APHASIA—Amnesia—Agraphia—Their characters, seat, and physiology.

APOPLEXY—Symptoms, onset, and course—Determination of locality—Its diagnosis from injury, narcotic poison, alcoholic intoxication, pyæmia, uræmia, epilepsy—Diagnosis of the anatomical causes of apoplexy.

Treatment of apoplexy and hemiplegia . . . . . 548—596

TUMOURS AND ABSCESS OF THE BRAIN

CEREBRAL TUMOURS—Tubercle—Syphilitic gumma—Meningeal growths—Glioma and other neoplasms—Symptoms: headache and other cephalic symptoms—Vomiting—Temperature, pulse, and respiration—Optic neuritis—Localising symptoms: for the base, the cerebellum, the motor area, and other parts—Diagnosis from hysteria, meningitis, &c.—Diagnosis between the several kinds of tumour—Prognosis—Treatment.

PAGES

CEREBRAL ABSCESS—Ætiology—Anatomy—Symptoms: general and localising—Diagnosis—Course and event—Treatment.

Red softening—Histology—Causes—seldom or never primary . 597—633

DISEASES OF THE MENINGES AND VENTRICLES

TUBERCULAR MENINGITIS—History—Morbid anatomy—Relation of the tubercles to the inflammation—Their occasional absence in basal meningitis—Ætiology—Sex and age—Onset—Course—Later and final stages—Tubercular meningitis in adults—Diagnosis—Prognosis.

Acute Non-tubercular Meningitis—Anatomy—Varied causes—Secondary and idiopathic—Symptoms and course—Diagnosis—Prognosis.

Epidemic Meningitis—History—Morbid anatomy—Symptoms and course—Varieties, complications, and sequelæ—Ætiology—Diagnosis.

Treatment of meningitis generally.

HYDROCEPHALUS—In children—Origin—Pathology and anatomy—Effect on the skull—Symptoms, course, and event—Treatment—Adult Hydrocephalus.

Adhesive thrombosis of the cerebral sinuses—Infective thrombosis of the sinuses.

Pachymeningitis hæmorrhagica . . . . . 634—679

GENERAL PARALYSIS, ETC.

GENERAL PARALYSIS OF THE INSANE—History—Ætiology—Symptoms, stages, and course—Anatomy—Diagnosis—Prognosis—Treatment.

Other forms of cerebral atrophy—Senile—Alcoholic—Saturnine.

Chronic diffused inflammation of the brain—Hypertrophy of the brain 680—692

FUNCTIONAL NERVOUS DISORDERS

THE SPASMODIC NEUROSES

Arrangement of the Neuroses and particularly of Spasmodic Neuroses.

LOCAL SPASMODIC NEUROSES—Facial or Histrionic Spasm—Spasmodic Wryneck.

Reflex and Fatigue Spasms: Saltatorial and Salaam Spasm—Spasmus nictitans—Singultus—Athetosis, &c.—Scriveners' Palsy, Hammer Palsy, and other handicraft-spasms.

Tetanilla—Description—Relation to Trismus neonatorum, Laryngismus, and Carpo-pedal contractions, and of all to Rickets—Ætiology—Treatment.

Thomsen's Disease—Its history, characters, and symptoms.

PARALYSIS AGITANS—History—Symptoms—Pathology—Diagnosis—Ætiology—Treatment.

TETANUS—Symptoms—Varieties—Ætiology—Pathology—Diagnosis—Events—Acute and chronic course—Mortality and Prognosis—Treatment.

CHOREA—Nomenclature—Symptoms and course—Chorea gravis—Morbid anatomy—Statistics—Pathology and Ætiology—Relation to Rheumatism and Cardiac disease—Embolic theory—Treatment.

Congenital choreiform spasms of cerebral origin—Hereditary chorea in adults—Myoclonus multiplex—Dubini's disease . . . 693—746

PAGES

THE PAROXYSMAL NEUROSES

Introductory remarks—Relation to other neuroses and to each other.

EPILEPSY—Definition—E. minor—E. major: aura: fit: sequelæ—Pathology, seat, and causation—Diagnosis—Treatment of the fit and of the intervals.

Other forms of eclampsia—Puerperal, uræmic, &c.

Paroxysmal vertigo—Auditory vertigo and Menière's disease—Somnambulism—Catalepsy—Night terrors.

MIGRAINE—Nomenclature—Symptoms—Pathology and causation—Treatment 747—786

NEUROSES OF THERMIC AND TOXIC ORIGIN

SUNSTROKE, AND THE EFFECTS OF DRINK AND OPIUM

HEAT-STROKE—Origin and pathology—Symptoms of the cardiac and cerebro-spinal forms—Prognosis—Sequelæ—Diagnosis—Treatment.

ALCOHOLIC NEUROSES—Delirium tremens—its symptoms and course—Prognosis and treatment—Chronic alcoholic poisoning—its symptoms and course—Antecedents—Diagnosis and treatment.

NARCOTIC NEUROSES—Opium-eating—The morphia habit—Symptoms and results—Treatment—Neuroses due to other narcotic drugs . 787—800

HYSTERIA AND HYPOCHONDRIASIS

HYSTERIA—A real and distinct disease—Mental and moral perversions—Hysterical affections of sensation and of movement—Contractures—Globus hystericus — Flatulence — The hysterical fit—Ætiology and Pathology of hysteria—Diagnosis—Prophylaxis—Treatment.

Catalepsy—Trance—Sleep-walking—Ecstasy—Dancing-convulsions—Mania—Hystero-epilepsy—Anorexia and marasmus—Hysteria in men and boys.

HYPOCHONDRIASIS—Distinctions from hysteria—The supposition of imaginary diseases, commonly abdominal—Distinction from melancholia—May mask real disease—Treatment—Prognosis . . . . . 801—825

PSYCHOSES, OR DISORDERS AFFECTING THE MIND

(By G. H. SAVAGE, M.D.)

Insanity as a (1) disorder of functions of brain; (2) an expression by the nervous system of bodily disease; (3) the expression of disease of the brain—Classification—Causation.

Acute delirious mania—Acute and other forms of ordinary mania—Melancholia with bodily complaints—Hypochondriasis with mental complaints—True Melancholia—Stupor, active and passive—Delusional insanity—Hallucinations of the senses—States of mental weakness—by defect—by instability—Specially named varieties of insanity: puerperal, alcoholic, moral, syphilitic, gouty, plumbic, febrile, phthisical, asthmatic, cardiac, renal and diabetic—Epileptic insanity—Paralytic and tabid insanity.

Testamentary capacity—Certificates and other medico-legal points.

Idiocy and imbecility . . . . . 826—858

CRETINISM AND ENDEMIC BRONCHOCELE

Cretinism as an endemic form of idiocy—Its anatomy—Endemic bronchocele—Sporadic cretinism in England—Relation of goitre to cretinism—The cretinoid condition in adults (myxœdema)—The condition which follows removal of a bronchocele (cachexia strumipriva) in man—and the effects of removal of the thyroid in animals—Acromegaly . . . . . 859—865

PAGES

DISEASES OF THE RESPIRATORY SYSTEM

AFFECTIONS OF THE LARYNX AND TRACHEA

The laryngoscope and its use—Arrangement of the subjects of this chapter.

LARYNGEAL PARALYSIS—Of one or both recurrent nerves—Of the abductor muscles alone—Of the adductors—Aphonia and other disorders of the voice.

LARYNGISMUS STRIDULUS—Nomenclature—Symptoms and Diagnosis—Pathology—Ætiology—Prognosis and treatment.

LARYNGITIS—Acute plastic inflammation—Croup—Its relation to Diphtheria and to Spurious Croup—Its symptoms and causes, anatomy, diagnosis, and treatment—Acute and chronic catarrh of the larynx—Tubercle—Lupus—Syphilis of the larynx—Œdematous laryngitis—Perichondritis.

TUMOURS OF THE LARYNX—Papilloma—Polypus, &c.—Sarcoma and Carcinoma—Laryngeal malformations—Foreign bodies in the larynx.

OBSTRUCTION OF THE TRACHEA—External compression from enlarged thyroid, aneurysm, &c.—Intrinsic stenosis from syphilis, &c.—Obstruction by impaction of a foreign body—Symptoms, diagnosis, and treatment 866—924

DISEASES OF THE LUNGS

SYMPTOMS AND PHYSICAL SIGNS

PERCUSSION—History—Methods—Terminology—Physical theory—Tympanitic resonance—Amphoric and cracked-pot sounds—Diagnostic significance.

AUSCULTATION—History—Methods—The respiratory murmur—Bronchial and tubular breathing—Physical explanation—Râles and rhonchi—The vocal resonance in health—Bronchophony—Pectoriloquy—Ægophony.

Palpation—Tactile vibration—Inspection and measurement of the chest.

COMMON SYMPTOMS—Dyspnœa—Varieties—Phrenic dyspnœa—Cheyne-Stokes breathing—Cough: reflex, faucial, gastric, and cerebral—Pleurodynia 925—950

BRONCHITIS

General symptoms: cough, dyspnœa, pain—Physical signs: rhonchus, sibilus, râles—Morbid anatomy.

Acute Bronchitis—Capillary form—Its symptoms and prognosis—Pulmonary collapse—Its production and relation to pneumonia.

Chronic Bronchitis—Varieties—Sequelæ—Emphysema—Its anatomy and origin—Atrophic emphysema—Symptoms and signs of emphysema—Bronchiectasis, uniform and saccular—Fœtid bronchitis—Ætiology, prognosis, and treatment of bronchitis generally.

Plastic Bronchitis—Its rarity—Anatomy, course, and symptoms—Treatment. 951—984

PNEUMONIA AND INFLAMMATION OF THE LUNGS

PNEUMONIA—History and definition—Anatomy and histology: stages, locality, events—Complications—Signs—Symptoms—Ætiology—Pathology—Prognosis—Mode of death—Convalescence—Origin and nature—Treatment.

Secondary pneumonia—Hypostatic pneumonia—Acute pulmonary congestion—Chronic lobar pneumonia.

BRONCHO-PNEUMONIA, or Pulmonary Catarrh—Causes in children and in adults—Anatomy—Symptoms and course—Prognosis and treatment—Vesicular, caseous, and pysemic pneumonia—Inflammatory œdema of the lungs.

CHRONIC INTERSTITIAL PNEUMONIA, or Cirrhosis of the Lung—Anatomy—Origin—Symptoms.

PAGES

SYPHILITIC INFLAMMATION OF THE LUNGS—Cases—Diagnosis—Anatomy—Acquired and hereditary forms.

GANGRENOUS INFLAMMATION OF THE LUNGS—Anatomy—Origin—Fœtor and other symptoms—Treatment . . . . . 985—1017

DISEASES OF THE PLEURA

PLEURISY—Its anatomy—Its physical signs—Pleuritic effusion—Compressed and carnified lung—Dulness—Ægophony—Symptoms—Course and events—Empyema—Diagnosis—of dry pleurisy—of serous effusion—of empyema—Hydrothorax—Hæmothorax—Pleuritic effusion in children—Complications of pleurisy—Its ætiology and relation to tubercle—Prognosis—Treatment of pleurisy and of empyema—Paracentesis and its results.

PNEUMOTHORAX—Origin and pathology—post-mortem characters—Physical signs—Diagnosis and symptoms—Prognosis—Treatment . . 1018—1052

PHTHISIS

History and definition—Pathology—unity of phthisis—localisation—phthisis always tubercular—Histology: miliary, caseous, and infiltrating tubercle—Tubercular pneumonia and ulceration—vomicæ—adhesions: involution—Symptoms: wasting, pyrexia, cough, sputum—The bacillus—Hæmoptysis—Physical signs of the three stages and of involution—Diagnosis—Course and duration—mode of death—recovery—Prognosis—Ætiology—the question of contagion—hereditary taint—diathesis and conformation—overcrowding, &c.—inhalation of dust—damp soil—Age and sex—Distribution—Treatment: preventive, curative, and palliative—diet and hygiene—climate—drugs.

ACUTE PULMONARY TUBERCULOSIS—Distinction from phthisis—Morbid anatomy—Physical signs—Clinical symptoms and course—Diagnosis by concomitant tuberculosis, especially of the choroid—Ætiology—Prognosis 1053—1120

CATARRHAL DISORDERS OF THE AIR-PASSAGES, WHOOPING-COUGH AND ASTHMA

CORYZA, or Nasal Catarrh—its symptoms, causes and treatment—Feverish colds.

INFLUENZA, or Epidemic Catarrh—history—symptoms—events—ætiology and pathology—diagnosis—prognosis and treatment—comparison of 1762 and 1890.

OZENA, or fœtid coryza—origin in atrophy of the nasal tissues—minor forms—treatment—Epistaxis—ætiology—course—treatment.

HAY FEVER, or Summer Catarrh—onset and course—ætiology—distribution among persons and places—treatment.

WHOOPING-COUGH—History and names—Symptoms and course—Events—Complications and sequelæ—Prognosis—Pathology—Treatment.

ASTHMA—The term—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Prognosis and treatment . . . . . 1121—1148

INDEX TO SUBJECTS . . . . . 1149—1186

THE

PRINCIPLES AND PRACTICE OF MEDICINE

INTRODUCTORY CHAPTER

ἰατρεύει (ὁ ἰατρὸς) καθ' ἕκαστα.—ARISTOTLE.

Definition of medicine and of disease—Nosology: organic and functional diseases—Pathological anatomy and clinical characters as bases of nosology—Diagnosis—Symptoms and diseases—Symptoms and physical signs—General Ætiology: exciting and predisposing causes of disease—General Prognosis and Therapeutics—Arrangement of the present work.

MEDICINE—*Ars medendi*, *Ars valetudinis*—is the art of detecting, of relieving, and of preventing diseases.

Preventive Medicine, in its general object of preserving the health of the community, forms a separate branch best called HYGIENICS, and in its relation to government, is known as State Medicine. In its particular object of preserving individuals from diseases it will find its place in several parts of the present work under the name of Prophylaxis. It depends upon our knowledge of the causes and origin of each malady.

Curative Medicine, or THERAPEUTICS, includes not only the application of means which entirely remove the disease and without which it would continue, but also the far more frequent cases in which without directly overcoming the malady, its course is guided in the most favourable manner towards recovery, and those moreover in which, although the disorder cannot be arrested, its progress is retarded and its pain relieved. *La médecine guérit quelquefois, elle soulage souvent, elle console toujours.*

This art of mitigating the sufferings and in more or less direct ways saving the lives of the sick is, like all arts, empirical and individual; but it rests on a scientific basis: first on knowledge of the several disorders of the human body and of the natural functions from which they are deviations—Pathology; and, secondly, on knowledge of the various means, mechanical, physical, chemical, and vital, by which the morbid structures and functions may be influenced—Pharmacology, in the most extended sense of the word. Inasmuch as our knowledge on both sides is very imperfect, we depend on the result of experience to correct our conclusions and to regulate our future methods.

The practice of medicine, then, is an art, controlled by experience and guided by individual skill and insight, but resting upon the science of

pathology, or the natural history of diseases, as its principles—on an exact acquaintance with their origin, immediate and remote, their physiological course, and their natural termination.

The art of medicine has been compared to the art of watch mending, to the art of war, and to the art of education; but perhaps the best illustration of its scope and methods is the art of navigation, which depends on scientific principles, but consists in the application of science to individual and varying conditions, guided by natural acumen, and controlled by long experience.

The present work attempts to deal with both branches of Medicine, its principles and its practice. By an arbitrary but useful convention external injuries and diseases which are chiefly curable by operations are separately dealt with as Surgical Pathology and Therapeutics; and the diseases peculiar to women, as well as those incident to the eye and ear, are also treated of in modern times by special treatises.

DISEASE.—Many attempts have been made to frame a satisfactory definition of *disease;* but no better result has been reached than that it is the opposite of *health*, a derangement of the body in whole or in part, which causes pain or shortens life.

In truth both Disease and Health are incapable of precise definition, because they are not scientific terms. They are descriptive, popular, and subjective.

"Disease" is "discomfort." Whatever causes bodily uneasiness or whatever by experience will sooner or later cause it, whatever interferes with our bodily functions, whatever tends to death, is disease. Health is the opposite condition, of comfort, ease, and ability to eat, sleep, move, and perform the other functions of life.

Diseases have only this in common, that they all interfere with comfort or shorten life. There is no common cause for the pains of inflammation, of colic and of mechanical injury.

No line can be drawn between health and disease. Pathology is only physiology under various disturbing causes. Decay and death are as much physiological events as birth and life.

All diseases imply two things—an exciting cause, *quidquid irritans*, mechanical, thermal, chemical, parasitic, infective, or of unknown nature; and a reacting, living organism, *quidquid irritabile.* Stone in the bladder is not a disease; the disease is the reaction of the body. The severest injuries, the most violent poisons, produce no disease in a corpse.

There is a tendency after disturbance to return to the previous condition, if the equilibrium has not been too violently upset. This tendency has been called *Vis medicatrix Naturæ;* but there is no such force, and the so-called "efforts of Nature" often aggravate instead of curing the mischief. Our mortal bodies are not made to last for ever.

It is clear that if disease is not a single state nor the result of a single cause, it cannot be removed by any single method, or on any universal principle.

Hence all "Systems" of Medicine, like all "Universal remedies," are of necessity false. Iatro-mechanical and iatro-chemical schools, Brunonian and Antiphlogistic theories, Allopathy and Homœopathy, are all equally unreasonable; not wrong solutions of a scientific problem, but ignorant answers to an absurd question.

The art of medicine, therefore, is not concerned with Disease in the

abstract, but with separate diseases, with pains and discomforts, the cause, seat, and origin of which the physician seeks to detect, and, if possible, to cure in each case—*ἰατρεύει γὰρ καθ' ἕκαστα.*

NOSOLOGY.—Since diseases are of diverse nature, so that they are not mutually comparable, it is clear that a scientific classification of them is impossible. Hence there is little left of the importance formerly attached to Nosology, or the right nomenclature, definition, and arrangement of diseases.

It may, however, still be asked—and the inquiry is not without a practical bearing—whether in naming and classifying diseases we should do so according to their *causes*, their character as physiological *processes*, the *structural changes* they produce, or the *symptoms* by which we recognise them during life.

The first is the most satisfactory basis, for it is fundamental. When we define scabies as the effects of the presence of an acarus in the skin, or "dropt wrist" as paralysis from lead, we know the most important points at once, the leading indications for prevention and for cure. But our knowledge is far too imperfect to render an ætiological classification possible.

A pathological arrangement is almost as hopeless, for in many cases we are ignorant of the physiological derangements which are taking place, and the long list of "inflammations" includes very diverse diseases.

What has in modern times been called a pathological nosology is not one of processes, but of results. It is really based, not on morbid physiology, but on morbid anatomy. During the present century the utmost zeal has been devoted to the search for organic lesions in the dead body, and to tracing the relations between such changes after death and states of ill-health during life. Many anatomical lesions have been thus discovered, which were unknown to the physicians of former ages; and many of these can be recognised without difficulty by skilled observers, though the patient is unaware of their existence.

On the other hand, there is a not inconsiderable number of disorders which force themselves into notice by the pain or discomfort they occasion, and which may in some cases destroy life; and yet after death all the organs and tissues seem to have their structure unaltered even when they are examined with the highest powers of the microscope and by every chemical test that can be devised.*

At first sight it appears as though there were a fundamental distinction

* By many writers, the occurrence of functional diseases is altogether denied. They believe that if our inquiries could be carried far enough we should discover some morbid changes to account for every disorder or complaint, even the slightest. They say that they "cannot conceive" how any of the vital processes should fail to be carried on naturally so long as every part of the body retains an absolutely normal structure; and they seem to suppose that no further argument is needed to establish their opinion. But, as is well known, the history of philosophy is full of lessons which show how fallacious it is to assume that things are impossible because when submitted to the scrutiny of our faculties they seem to be inconceivable. The question is not whether an effect can arise without a cause, but whether the cause must necessarily be of one particular kind because we know of no other.

However, physiology suggests an analogy that may not perhaps be without its application to pathology; the case of muscular fatigue. This was formerly supposed to be the result of the consumption of contractile tissue in the performance of work. It is now known to be due to the accumulation in the tissues of those waste products which arise out of the contractile material so consumed. But the nerves and nerve-centres are also liable to exhaustion; and it seems not unlikely that the *neuroses*—as functional nerve disorders are called—may depend upon similar conditions.—C. H. F.

between *organic* or *structural* diseases attended with recognisable morbid changes, and *functional* diseases in which no such lesions can be found. But the progress of science is continually transferring maladies from the latter to the former class. So that the distinction between functional and organic disorders is, though convenient, not fundamental.

In both cases the "disease" is the *condition* of the living patient, the pathological *process*, the *derangement* of the functions, and this includes the "symptoms" (which are generally the most obvious phenomena in the living patient and which used themselves to be called diseases) and the anatomical lesion, if known, which underlies them.

Hence, as knowledge improves, what was a "disease" becomes a "symptom," and what was an anatomical lesion becomes the evidence of a pathological process. Dropsy, jaundice, paralysis, are terms which now denote only the first, not the final stage of diagnosis. Phthisis, which at first meant "wasting," afterwards meant a special anatomical lesion of the lungs, and now means the whole physiological process, including structural, functional, and symptomatic disorders which are the result of infection by a microscopic plant.

It is not necessary that every disease should be defined alike; in *scabies* the fundamental character is the presence of a special cause, in *aneurysm* it is an anatomical lesion, in *chorea* it is a series of disordered functions. Whenever a group of clinical cases can be traced to a single structural change, this should be regarded as the most essential element in the definition of the disease, and it should, if possible, furnish the name. But if the effects of several lesions are identical, so that they are clinically undistinguishable from one another, it is better to retain a common designation. Such an arrangement may be regarded as provisional, but it is likely to be needed for many years to come.

DIAGNOSIS.—Until we have traced a disease to a definite organic lesion we can seldom be sure that cases which seem to be identical are really so; and upon their real identity depends all certainty of knowledge as to their cure.

Again, until we know the origin of the structural lesion we cannot tell how to guard against its production, *i. e.* how to prevent the whole process which we call the disease.

The process by which we give a name to a case is one of analysis; and in some cases we can carry this process further than in others. A complete diagnosis, such as "variola," "syphilitic iritis," "scabies," includes recognition of the characteristic symptoms of the anatomical lesion in its seat and structure, and of the origin of the whole morbid process. Nevertheless, to say that a patient has "hepatic ascites," though an incomplete diagnosis is sometimes more accurate than to say that he has "cirrhosis of the liver," for we cannot always exclude perihepatitis, or cancer, or gummata. To define a cerebral case as one of "hemiplegia" is better than to call it "softening of the brain," if the lesion is but little less likely to be a clot of effused blood, or a tumour, or an abscess. Yet there are cases in which we can be sure that cirrhosis of the liver, or that softening of the brain is present; and it is obviously better to name such cases according to their respective lesions.

The diagnosis of "hemiplegia" or of "ascites" is a provisional but not a wrong diagnosis: the phenomena which are commonly spoken of as sym-

ptoms are parts of the disease to which they belong, no less than the lesion, or the specific cause, or whatever else is taken as its main characteristic. It is true that, when they afford the means by which we infer the existence of a hidden morbid change, they bear to it the relation of effects to a cause; but this only shows that the term symptom is a relative one. So long as we are unable, either during life or after death, to discover any cause for a set of clinical phenomena, they certainly are not symptoms, for they indicate nothing. There can be no symptom until the disease of which it is a symptom is discovered. As the process of analysing a case or a group of cases advances, the name which we apply is rightly changed at every step; and each "disease" becomes in its turn a "symptom" of the disease which succeeds it. Itching was once hardly distinguished from the itch; afterwards, when the characters of the eruption of scabies were made out, itching was looked on as a symptom, and the eruption was the disease; now that the *sarcoptes* has been discovered, the eruption is itself reduced to a symptom.

If, however, the analysis is always arrested at a certain point, it is absurd to say that the ultimate fact in such cases is "only a symptom." So far from hesitating to classify clinical symptoms as diseases when we are unable to trace them to their causes, it is to be wished that we had a more complete supply of suitable names denoting functional disturbance. It is probable, as Dr Moxon argued in the 'Guy's Hospital Reports' for 1870, that a good "clinical nomenclature" would be quickly followed by a great advance in our knowledge of functional disorders.

Even when we understand the whole pathology of a case, and when the clinical phenomena which characterise it are rightly called symptoms, the distinction between them and the anatomical lesions is often arbitrary. Thus, in acute tuberculosis, if the ophthalmoscope discovers a tubercle in the choroid membrane of the patient's eye, it is called a symptom; but the disease itself (anatomically) is but a multitude of precisely similar tubercles. So, again, the enlarged liver of mitral disease, the nodes of syphilis, the swollen joints of rheumatism belong in strictness to the pathological anatomy of these several complaints. Nor, if we would be logical, can we stop even here; the same thing must be said of all cutaneous eruptions, down to the rose-rash of enteric fever.*

On the other hand, in the case of a functional disorder, to speak of the clinical phenomena as symptoms is really to reduce the "disease" itself to a mere name.

Practically, however, we agree to call all indications which help us to a diagnosis, symptoms, whether they respect structures or functions.

SYMPTOMS AND SIGNS.—Symptoms, so defined, naturally divide themselves into two distinct classes. Some of them are "subjective;" that is, they are appreciable by no one but the patient, so that our knowledge of them rests solely on his statements. Others are "objective;" that is, they can be directly observed by other persons; indeed, they can be investigated when the patient himself is unconscious.

Now, subjective symptoms are open to fallacy. They can often be feigned by one who desires to deceive. Moreover, their severity depends

* In that part of 'Ziemssen's Cyclopædia' which is devoted to the exanthemata, the appearances seen on the skin are in fact described in some instances (but not in all) apart from the "symptoms," and under the head of "anatomical changes."

upon the nervous susceptibility of the individual in whom they occur: a hearty labouring man will take no notice of a pain which would drive a delicate lady to her bed for weeks. In the former case one must be careful not to overlook, in the latter not to assume, the presence of disease. Yet constant attention must be paid to subjective symptoms, for they often yield information which can be obtained in no other way.

The physician elicits objective symptoms by examining the patient's body with hands, eyes, and ears. Until the time of Auenbrugger, Corvisart and Laennec, he could do little more than feel the pulse and look at the tongue. Then came the discoveries of percussion and of auscultation, which gave to the diagnosis of diseases a precision undreamt of before. It was natural that those who introduced the new methods should endeavour to mark their value by giving a special name to the indications afforded by them. And thus it became usual to speak of "physical signs" as opposed to "symptoms." In reality, however, the distinction is untenable. A raised temperature, an internal squint, albumen in the urine—are all "physical signs," no less than a cardiac murmur or a dull percussion-note; and small crepitation is as much a symptom as rusty sputa. Lastly, it is misleading to call a particular symptom "pathognomonic," as if it bore so close a relation to the disease that, whereas all the rest of the symptoms afford only presumptive indications of its presence, this one proves it absolutely.

ÆTIOLOGY.—Our knowledge of the *causes* of diseases is, as a rule, exceedingly fragmentary and imperfect. In many instances there is little difficulty in perceiving that an illness has been preceded by some definite change in the outward circumstances of the patient, or in the action of some vital organ: he may have been chilled by exposure to cold, he may have eaten unwholesome food, he may have been over-excited or over-fatigued; and one of these conditions is then called the "*exciting cause*" of the disease. But, on further consideration, it appears that people often go through weather no less inclement, indulge their appetites as foolishly, have their mental and bodily powers strained as much, without being attacked by the same, or indeed by any disorder. We now have no alternative but to assume that some other cause has also been in operation. This may act in two ways: it may combine with the exciting cause to produce the disease in him who falls ill, or its influence may affect those who remain well, by counteracting the operation of the exciting cause. But in practice we generally assume that all causes of this kind have a positive rather than a negative action; and we are therefore accustomed to group them together under the name of "*predisposing causes.*"

It is, however, important to note that some apparently predisposing causes of diseases do not operate in either of the ways just mentioned. A complaint may affect persons at a particular age, merely because at that age they are more exposed to the action of the exciting cause. Thus scalds and burns are particularly frequent among young children; and scalds of the throat from drinking hot water from a kettle scarcely occur in grown-up persons. On the other hand, young adults are much more liable to break their limbs than are old men. Such differences arise, not from a predisposing influence of one period of life, nor from a counteracting influence of another period, but from infants being ignorant of the effects of heat, and from men at their prime being obliged to work under exposure to accidents. So, again, if general paralysis and locomotor ataxy and many

other diseases of the nervous centres are more frequent in men than in women, the reason may be that more men than women come under the operation of the exciting causes of these complaints, such as overwork, sexual excesses, and the like.

The most obscure of predisposing causes are those which are commonly included under the term "idiosyncrasy." It is well known, for example, that in certain persons nettlerash is produced by strawberries, and that in others the odour of cats excites asthma. In such cases the exciting cause is obvious, and we must regard as predisposing causes the conditions, whatever they may be, which lead to its effect upon one person and not on another. But of these conditions we are altogether ignorant.

In some cases predisposition seems to be unnecessary. The presence of the acarus in the skin is the sole and efficient cause of scabies, and needs nothing more than a normal human skin to produce the lesions and the symptoms which make up the disease scabies. Yet even here it appears that certain abnormal conditions, as typhus, and perhaps other febrile states, counteract the ordinary operation of the irritant. Again, contact with another is the indispensable precedent of every case of variola, yet here again a previous attack renders the organism "refractory" to the normal operation of the contagion. That grown people are little susceptible to measles is probably explained by most of them having already had it.

If we attempt to classify diseases by their causes, we may put first those which are due to the presence of animal or vegetable *parasites*, both such as act mechanically by their presence as irritants, and those minute parasitic fungi which multiply excessively and fill the whole organism that they invade with swarms of microphytes.

Next may be placed, from an ætiological point of view, those disorders which are the direct result of *chemical poisons*, the paralysis caused by lead, the delirium of belladonna poisoning, the tremors of workers in mercury, the fatty degeneration caused by phosphorus. In these cases the effects are very uniform in character, and depend in degree upon the dose of the poison, with little reference to the recipient.

Next we may put *dietetic* diseases, as cirrhosis of the liver and delirium tremens, caused by spirit drinking. Here, although the cause is true and constant, its effect depends upon something in the individual, for some drunkards die rapidly from the effect of intemperance on the brain, others more slowly from its effect on the liver, and others again escape both.

The action of *cold* is still less constant. Exposure to weather produces in one man rheumatic fever, in another bronchitis, in a third pleurisy or pneumonia, so that we cannot predict what disease will be produced by the cause, or whether any at all.

The remaining "causes" of disease are so vague and uncertain in their action that we must suppose that some peculiar combination of conditions is needful to produce the morbid effect, and that of one or more of these conditions we are ignorant.

When we cannot assign any probable cause for a disease we often call it "idiopathic" or "spontaneous;" meaning that so it appears to be, until its true ætiology is ascertained.

A small ætiological group of diseases consists of those which are the result of *defect of formation*, as cyanosis from congenital disease of the heart.

A larger one includes those which depend upon various *senile degenera-*

tions of the tissues and organs. But many of these changes which occur as the result of ordinary wear and tear in most persons above a certain age may also be found in those much younger, in whom excessive strain has prematurely produced the same effects.

On the whole it may be said that the cause of diseases is more confidently assigned by the patient than by the physician.*

When degenerative, developmental, contagious, parasitic, toxic, and traumatic diseases are excluded, there remains by far the greater number of the real origin of which we know nothing.

Even in these cases, however, their natural history—the conditions of age, sex, occupation, geographical distribution under which they most commonly occur—is of much interest. Such knowledge is often a useful help in diagnosis, and may one day lead to more complete conclusions as to ætiology. So that under this head such details are appropriately placed.

It is in reference to the prevention of diseases that a knowledge of their ætiology is of the greatest practical use, but indirectly it often has an important bearing upon their diagnosis and their cure. The process of analysing the clinical symptoms, which constitutes the diagnosis of a case, often stops short of tracing it to a definite anatomical lesion; and we are unable by that path to reach any basis for a sound plan of treatment. Every circumstance in regard to possibly exciting or predisposing causes of the patient's illness may then be of great value in helping us to treat it successfully.

PROGNOSIS.—Before we attempt to modify the progress of a disease by treatment we have to ascertain what is its natural course when undisturbed by our art, so that we may know as certainly as possible whether the remedies which we use are effectual. It is this study that enables us to give what is termed a *prognosis* or forecast of the case to the patient himself or to some near relative, a part of our duty which may be of the highest importance, and in which our credit and reputation are more concerned than even in treatment. In some cases, indeed, the prognosis is of such a character as almost to make any further services to our patient unnecessary. We may be able to declare that the disease will quickly and safely pass off, without tendency to relapse, and without risk of its leaving any ill effects behind it. A case in point is an attack of shingles in a child. Or we may have to say that the malady is one which must inevitably prove rapidly fatal in spite of every effort that can be made to arrest it. Such a prognosis, for instance, must be given in many cases of apoplexy, of aneurysm, and of tetanus, and in almost all of cancer. But, as a rule, our prognosis applies, not so much to the natural and undisturbed course of the disorder, as to that which we believe it will take under the most judicious treatment we can devise.

Prognosis is not only concerned with the favourable or unfavourable event of the disease, but also with its probable duration, the chances of its return, and the possible or probable complications against which it is most important for the physician to be forewarned.†

* As Sir William Gull well put it: "in disease as well as in astronomy, savages explain, science investigates."

† 'Guy's Hospital Reports' (1887), vol. xliv, p. 59.

TREATMENT.—Therapeutics, or the art of curing diseases, depends much more upon the physician's knowledge and skill, and much less upon drugs, than is supposed by the laity. In few cases can a malady be certainly and directly "cured" or arrested by a given method, as soon as the diagnosis has been made; and in still fewer is medicine powerless for the comfort and welfare of the patient.

In some diseases no treatment is needful. All that is important is that they should be recognised, and the patient assured of his safety.

In others the only reasonable treatment is expectant; that is, to refrain from meddling without knowledge, and to watch carefully for opportunities of helping favourable and relieving unfavourable symptoms.

In others, again, treatment is strictly rational, *i. e.* it consists in employing mechanical, chemical, or physiological means for directly meeting an injurious condition. Such is the treatment of laryngeal dyspnœa by opening the trachea, of poisons taken into the stomach by chemical antidotes and by emetics, of high temperature by cold affusion. In the same way we treat iritis by belladonna, dropsy by diuretics, typhlitis by opium, and uterine hæmorrhage by ergot.

There remain a considerable number of cases in which, without our being able to see the physiological action of the means that are used, we are able to modify or arrest the process of a disease by treatment which depends entirely upon experience of its value. As examples, may be mentioned the relief given by external warm applications in most inflammatory states, the benefit of counter-irritation, the use of purging in certain cerebral affections, of colchicum in gout, of tarry applications in psoriasis, of iron in chlorosis, and of mercury in syphilis. These last so-called specific medicines have doubtless a physiological action which, if known, would explain their practical value; and in some cases we can perceive a doubtful connection between their known properties and their therapeutical effects. But at present their action is "special," peculiar, and inexplicable.

Arrangement of the present work.—As above explained, no scientific classification of diseases is possible, inasmuch as they are incommensurable objects. The most practically convenient arrangement is the best. For the reasons already given, the anatomical lesion is, when ascertained, the most definite and satisfactory basis of nomenclature and classification; when this fails, we generally name and group diseases by their most important clinical features, or in accordance with the organ most obviously affected; and these associations are often more important than affinities of pathology and causation. Thus scabies is more usefully studied along with other forms of dermatitis than with ringworm or hydatids, and phthisis goes more naturally with bronchitis and pneumonia than with lupus.

In accordance with general practice, from which it is inconvenient to deviate except for some sufficient reason, we shall take first among diseases those which have been long known as specific fevers affecting the whole body, and running a more or less constant and limited course.

Next, still following the traditional order, will come the diseases of the brain and nervous system, then those of the chest, and afterwards affections of the abdominal viscera.

Lastly, we will for convenience place certain more or less general diseases, the true pathology of which is undetermined, which are neither

strictly local nor yet specific fevers. They are—scurvy and other disorders accompanied by anæmia and hæmorrhage, which may provisionally be regarded as primary *hæmatoses;* rheumatism, gout and other "idiopathic" and multiple affections of the joints (*arthroses*); and the numerous and complicated disorders which affect the skin and its appendages.

In each section we shall more or less closely follow the same order, dealing first with the name and history of the disease; then with its clinical characters, course, and complications; next with its anatomy and pathology, its ætiology and natural history, and its diagnosis from other diseases. Lastly will come its prognosis and prophylactic, palliative, or remedial treatment.

Before, however, we begin the study of the several specific fevers, it will be well to devote a few preliminary chapters to certain pathological processes which are common to them and to many of the local diseases to be afterwards described.

These are contagion, fever, inflammation, pyæmia, and the formation of new growths. Other general pathological processes, as anæmia, dropsy, embolism, and the various forms of degeneration, will be better considered afterwards with the diseases in which they are most important.

INFECTION

Οὐρῆας μὲν πρῶτον ἐπῴχετο καὶ κύνας ἀργούς
Αὐτὰρ ἔπειτ' ἀνοῖσι βέλος ἐχεπευκὲς ἐφιεὶς,
Βάλλ', ἀιεὶ δὲ πυραὶ νεκύων καίοντο θαμειάι.
HOMER.

Transmission of diseases—Contagion and miasm—Theory of infection—Contagium vivum—Microbes—Their origin and life—Their exclusive action in producing specific diseases—Their mode of action—Immunity from contagion—Protective inoculation—Modes of transference of miasmata and contagia—Theory and practice of disinfection.

FROM an early period it has been observed that certain diseases are contagious, or "catching," by direct contact with the sick person, or by means of clothing or buildings which transmit the *materies morbi*. When actual contact took place, the disease was called *contagious;* when it was transmissible by the air, it was given the wider name of *infectious*, and the vehicles of transmission were called *fomites*.

These diseases frequently spread so rapidly that they become *epidemic*, and thus have devastated whole kingdoms and changed the course of history.

They have been, and still are, called *specific* diseases—partly from the special and peculiar course which they run, unmodified by the accidents of diseases produced by injury or cold or local inflammation—partly because each one was believed to be produced by a single definite efficient cause, the supposed *virus* or *contagium* or *materies morbi*, the presence and nature of which is now in many cases demonstrated.

There are, however, other "specific" toxic disorders which must be distinguished from the general contagious diseases. These are:—

(1) The effects of chemical poisons. Mercury, arsenic, lead, phosphorus, morphia, strychnia, when absorbed into the lymph and blood so as to reach the tissues, produce definite and constant effects, specific in their locality and symptoms, and pointing unmistakably to their "specific" origin. But these effects are strictly limited by the dose of the poison, and they are not transmissible from one person to another.

(2) The effects of certain organic poisons the chemical nature of which is imperfectly known. Such is the venom of snakes, a nitrogenous and probably a proteid compound; the albuminoid principle of jequirity seeds, called *Abrin*; and the remarkable ferment discovered by the late Dr Wooldridge, the injection of which produces instant coagulation of the blood in the portal vein.

(3) The effect of certain products of decomposition, causing septic poisoning: these are nitrogenous but alkaline, and probably compound ammonias. They have been called *ptomaïnes* or cadaveric alkaloids, *leucomaines* and *toxines*.

In all these cases the poison is a chemical compound, and acts in proportion to its strength; moreover, the morbid conditions produced are not transmissible.

(4) We must also separate certain diseases which are truly contagious and

which depend on the presence of living organisms, but which are only local in their effects and do not produce any general physiological disorder. Examples are scabies and ringworm.

There remain the specific contagious febrile diseases, in which the contagion consists of a swarm of living organisms, *microbes*, which multiply in the host, set up a disturbance of the whole body and render it for the time a focus of fresh infection.

This group of diseases has been divided as follows:

(1) *Contagious diseases proper.*—Each of these maladies owes its origin to a *virus* or *contagium*, derived from a person already suffering from it, or from one of the lower animals. Among these are the Exanthemata, Typhus, the Plague, Mumps, Whooping-cough, and Glanders.

(2) *Miasmatic diseases.*—These are caused by a contagium which is derived from the soil, or from the water, or from the air of a place, independently of the occurrence of similar illness in another person. Ague is the chief example of this group.

(3) *Miasmatic-contagious diseases.*—This group, which is acknowledged by all German observers, is little recognised in England.* The typical examples are Enteric fever and Cholera, diseases which are undoubtedly spread by human intercourse, and which are usually, if not always, traceable to previous cases, but which are nevertheless believed not to be directly communicable from one sick person to another. Liebermeister's hypothesis is, that patients give off a contagion which is inoperative until it has passed through further stages of development outside the human body, and that these changes may be taken as converting it into a kind of *miasm*.

The distinction is a convenient one for the present, but will no doubt become obsolete with increasing knowledge.

Theory of infection.—Contagia often appear as fluids; this is the case, for example, with the vaccine lymph, with the contents of smallpox vesicles or pustules, with the nasal discharge of glanders, and probably that of measles, and with the faucial secretion of diphtheria. Chauveau in France, and Burdon Sanderson in England, have demonstrated the fact that the activity of these fluids belongs, not to chemical compounds dissolved, but to minute particles suspended in them. They have shown, for example, that when vaccine lymph is allowed to diffuse into distilled water through a porcelain diaphragm, the diffused liquid is incapable of conveying cow-pox by inoculation.

That contagia are "particulate" is therefore clear. The next point is as to the nature of the particles. For a long time the dominant view was that they were inorganic, or at least devoid of life, that their properties were essentially chemical, and that they acted "catalytically" or after the manner of *ferments*. On the ground of this analogy the epithet "zymotic" was coined, and applied to the whole class of infective diseases, and the contagia were called *microzymes*.

But ferments are of two kinds, chemical like pepsin, and perhaps snake's venom, or particulate like yeast; and the particles of yeast are living

* The epithet "miasmatic-contagious" was proposed by Henle in 1840. He, however, only had in his mind the circumstance that smallpox, scarlet fever, typhus and other maladies often seem at first to break out epidemically, as though they might have been caused by a miasma, whereas afterwards the individual cases afford clear evidence of contagion. But Liebermeister and other recent writers have assigned a fresh meaning to the term "miasmatic-contagious," and have limited its application to a special class of maladies.

organisms, so that there can be little doubt that the particulate contagia are living organisms of exceedingly minute size.

Moreover, in two infective diseases—Relapsing fever and Anthrax—the *constant presence* of such organisms was several years ago positively demonstrated. To these may now be added: Tuberculosis, Lupus and Leprosy, Glanders, Erysipelas, and several forms of Septicæmia in the lower animals. The cases of Enterica, Scarlatina, and certain other specific diseases are more doubtful; while in some, as Typhus and Plague, no characteristic microbe has yet been discovered.

The conditions which justify our regarding a given organism as the material cause, the *contagium vivum*, of a given malady may be stated as follows:

(1) The malady must have such distinct and constant features, whether clinical or anatomical, as shall enable it to be identified.

(2) The microphyte must be itself distinguishable from others by its size and shape, its staining properties, but above all by its mode of propagation in "a pure cultivation" (*i. e.* artificially separated from other organisms) and by the form and colour of the colonies it produces.

(3) The organism thus identified must occur in the blood or tissues—not merely on the surface, cutaneous or intestinal, but below the epithelium, in the lymph-spaces or blood-vessels—in every case of the disease in question.

(4) It must not occur in the human body except in cases of the particular disease in question. It may, however, conceivably exist in other animals without giving rise to the same pathological symptoms; or in air or water—as it exists in the test-tubes and plates of "pure cultivations," and in whatever media convey the contagion from one person to another.

(5) When a pure cultivation of the organism is introduced into the blood and tissues of an animal, the phenomena, clinical and anatomical, of the disease in question must be reproduced.

These conditions in all their rigor have been satisfied in the case of Anthrax, and with scarcely less abundant proof in that of Relapsing Fever.

The micrococcus of Erysipelas has also been established as the *contagium vivum* of that disease by good evidence.

In Variola and Vaccinia micrococci are present, but they have not been proved pathogenic by experiment. In Pneumonia several microphytes occur, but not constantly, and which, if any, is the true pathogenic organism is still uncertain. The micrococci of Gonorrhœa and of ulcerative Endocarditis are constantly present, and probably pathogenic, but they are less certainly identified, and the proof is incomplete. The bacilli of Enteric fever, of Diphtheria, and the so-called comma-bacillus of Cholera, are still the subjects of controversy as to identity, constancy, and pathogeny.

The theory that specific infection depends on the transference of living microscopical vegetable organisms is in harmony with the fact that so many other processes which bear a clear analogy to specific infection are constantly associated with the presence of microbes. Lister, in a paper read before the Pathological Society in 1877, established that the lactic acid fermentation, or souring of milk, is essentially dependent upon what he termed the *Bacterium lactis;* and Pasteur had previously shown that the alcoholic and the butyric fermentations have a similar origin. So, again, that putrefaction is set up by certain bacteria (or *saprophytes*) is now certain; the success of

Lister's method of treating wounds is nothing less than a demonstration that they remain aseptic only so long as the organisms which induce decomposition can be kept at a distance.

The organisms concerned in these various processes differ remarkably in size, and therefore in the readiness with which the microscope reveals them. The *Torula cerevisiæ*, or yeast plant, which brings about the alcoholic fermentation, is made up of rounded cells which develop by budding, and have a diameter of $\frac{3}{10,000}$ or $\frac{4}{10,000}$ of an inch. The *Bacterium lactis*, which causes the souring of milk, consists of oval bodies, arranged in pairs or sometimes in chains, multiplying by fission, and measuring at the most $\frac{1}{20,000}$ of an inch, that is, being no larger than the granules which are contained in the cells of the torula. Consequently, as Lister argues, there is nothing improbable in the supposition that yet other organisms may exist which may be as much smaller than the *Bacterium lactis* as it is smaller than the torula. Heydenreich aptly remarks that, were it not for the length of the spiral threads which form the microbe of Relapsing fever, it would in all probability have escaped detection; rounded or oval bodies not exceeding in diameter the breadth of the *spirillum* would be almost invisible with the highest powers of the microscope.

The following are the most important parasitic microphyta. They all agree in not containing chlorophyll:

1. Moulds (*Mucorini*, *Hyphomycetes*, *Schimmelpilze* of Nägeli). These are long-branched filaments, which form numerous spores. To the pathologist they have little interest, but one species is sometimes found lining the interior of dry vomicæ in the lungs (*Aspergillus fumigatus*). Other forms cause certain cutaneous affections, as ringworm.

2. Budding fungi (*Saccharomycetes*, *Blastomycetes*, *Sprosspilze* of Nägeli). These consist of rounded or oval cells, which give off buds, and may form beaded threads. To this group belongs *Oidium albicans*, the parasite of thrush.*

3. Fission-fungi (*Schizomycetes*, *Spaltpilze* of Nägeli). These are small bodies, which multiply by fission, with or without production of spores.

α. *Spherical bacteria* (*spherobacteria* of Cohn, *coccaceæ* of Zopf, *micrococci*) are exceedingly minute. They sometimes cohere in pairs (*diplococci*), sometimes in chains (*streptococci*), and sometimes in heaps (*staphylococci*), and sometimes they are aggregated into masses held together by a jelly-like material (*zooglœa*). *Sarcinæ* are micrococci arranged in square or cubical packets, the result of fission in different planes.

β. *Bacilli* (*desmobacteria* of Cohn) are cylindrical rod-shaped bodies, which sometimes remain united after they have undergone fission, so as to form threads of considerable length.

γ. *Rod-shaped bacteria* (*microbacteria* of Cohn) are smaller than bacilli. They are often slightly constricted in the centre, or dumb-bell shaped.

δ. *Spirilla* and *spirochætæ* are spiral filaments (*vibriones*, *spirobacteria* of Cohn), having a well-marked corkscrew motion.

By Billroth rod-shaped and spherical bacteria were associated together under the name of *coccobacteria*. By other authors the bacteria and bacilli are combined, and, from a morphological point of view, this is well justified. But these names are all descriptive only, and not indicative of botanical

* Another (*Saccharomyces capillitii*) has been recognised on the hairy scalp and elsewhere on the skin. Two other remarkable pathogenic fungi are found in cases of Madura foot in India (*Chionyphe Carteri*) and of Actinomycosis.

relations. A more important distinction is between microphytes, which form endogenous spores, like *Bacillus anthracis*, and those which multiply by fission only.

It can be easily understood that there is often great difficulty in distinguishing micrococci from lifeless granules of organic or inorganic matter. An oscillating motion is of no significance, for it depends on vibrations transmitted from without to the liquid in which the granules float. Brunonian movements also affect dead particles. Bodies which subdivide or which cohere into chains may safely be set down as organisms. Irregularity in size is almost sufficient to show that the granules which differ among themselves are not micrococci; but uniformity in this respect affords no positive proof that organisms are present. The power of resisting liquor potassæ is evidence of a cellulose investment of the protoplasm being present, *i. e.* of the microbe being vegetable.

It is often convenient to be able to refer to them under a common name, without specifying their exact characters; and for that purpose the word *microzyme* was suggested by Béchamp and adopted by Sanderson. *Microphyte* is perhaps a better term. *Microbe* is the one used by Pasteur and the French school.

Microphytes are classified as *aerobic* and *anaerobic* by Pasteur, according as they grow in contact with oxygen, or protected from the air.

An important distinction is that made by Flügge, into *septic* (*saprogenic*) or putrefactive microphytes, *zymogenic* or fermentative, *chromogenic* or pigment-forming, and *pathogenic* or productive of specific diseases.

Zopf carried Nägeli's and Billroth's doctrine of the "pleomorphism" of the schizomycetes to its utmost limit. But more critical biologists admit variation in form of one and the same organism only within certain narrow limits. This was first proved by Lankester in the case of *Bacterium rubescens*, a chromogenic microphyte, since named *Clathrocystis roseopersicina* by Cohn ('Quart. Journ. Mic. Soc.,' 1876).

There is no reason to believe that chromogenic microphytes can be cultivated into zymogenic, or septic into pathogenic. The assertion by Buchner that the common *Bacillus subtilis* of infusion of hay was interchangeable with the Bacillus of anthrax and *vice versâ*, has been refuted by Klein. Pathogenic bacteria, however, may undoubtedly suffer diminution of their malignancy (or may acquire it in intenser degree) by being passed through a series of hosts. This is the explanation of Jenner's vaccination, for there is little question that the cow-pox is variola passed through successive generations of cattle. And on the same principle Pasteur has succeeded in "attenuating" the virus of anthrax, of so-called "chicken cholera," and of hydrophobia, so as to protect against the unmodified disease by inoculation, or, as he calls it, "vaccination," with the attenuated contagium.

Contagious pus.—Mr Hutchinson has repeatedly expressed the opinion that the "contagious inflammations' (as he terms them) differ essentially from the specific fevers, and that they are conveyed from one person to another by leucocytes or living pus-corpuscles, and not by microzymes. The instances he enumerated in the discussion of the Germ Theory held by the Pathological Society in 1875, were Gonorrhœa, Erysipelas, purulent Ophthalmia, and Porrigo. The points on which he relied in support of his views were chiefly two; first, the frequency with which some of these diseases occur apart from any apparent origin in contagion; and secondly,

the fact that the severity of the inflammatory process in each disease varies widely in different cases, and may be modified at will in experimental inoculations, as when gonorrhœal pus is applied for the cure of pannus. This last argument seems, however, to be met by the fact that as much may be said of some maladies which are typically specific; and although erysipelas often appears to be directly caused by exposure to cold, a similar origin can sometimes be traced for diphtheria.

Lister's observations on the lactic acid fermentation teach an important lesson in regard to this question. One would certainly have thought that the souring of milk, which so constantly occurs when that fluid is left standing for a few hours in warm weather, must depend upon causes which are in operation everywhere, and that any microbes concerned in the process would be devoid of specific characters. But Lister found that although the *Bacterium lactis* appears to be universally present in dairies, it is scarce elsewhere: milk which has been boiled may be exposed to the air in ordinary rooms for half an hour at a time, but while other bacteria and filamentary fungi may develop, it will rarely, under such circumstances, become curdled and sour, nor will it contain the *Bacterium lactis*. Is it not probable that though the occurrence of common inflammation favours the development of erysipelas or diphtheria, yet access of specific microphytes is the essential factor in the ætiology?

There can be little doubt that pus is contagious by virtue of the presence of certain micrococci which have been described by Ogston and cultivated by Rosenbach, Garré, and other pathologists under the title *Staphylococcus pyogenes aureus*. This appears to thrive out of as well as in the body, and to be very widely, almost universally, distributed. Without going so far as Hüter in denying that suppuration ever takes place except as the result of access of staphylococcus or allied microphytes, we may say that this is probably true of all contagious and specific pus.

In the pus of Gonorrhœa and of Erysipelas, recent research has shown the constant occurrence of specific microphytes, described as *gonococcus* and *streptococcus* respectively.

Spontaneous origin of contagia.—Some physicians, of whom the late Dr Murchison was one, used to believe that certain typically specific diseases sometimes arise *de novo*, independently of contagion or of any virus derived, directly or indirectly, from previous cases. No doubt all such diseases must have had a commencement at some period of the world's history, just as animals and plants must have had a beginning; but we are only concerned as physicians in the question whether specific diseases are started afresh in the world as it is; and the same answer must be given as to the theory of spontaneous generation of animals and plants. When the origin of an outbreak of a contagious malady has been investigated with sufficient care, it has often been traced in the most conclusive manner to a previous case, perhaps at a great distance, from which it may have been derived in some indirect and unexpected way. Moreover it is worthy of notice that particular instances, of which no satisfactory explanation can be given, occur in the case of smallpox—of which a spontaneous origin was always admitted to be most unlikely—no less than in that of diphtheria, or typhus, or enteric fever, which were once supposed to be frequently so generated. Sir Thomas Watson cites Dr. Gregory as having stated that, of the cases admitted into the Smallpox Hospital in his day, not one in twenty could be referred to any known source of infection.

We may therefore absolutely reject the doctrine that any of the specific diseases ever result from such causes as overcrowding or starvation; or even from the inhalation of sewer gas which contains no specific virus.

It is, however, another question whether the microphytes of an infective malady, after escaping from patients, may not sometimes go on multiplying for long periods of time, and spread to distant localities, before they again penetrate into the human body. In the miasmatic-contagious diseases it seems probable that such a process of development always occurs. In some also of the strictly contagious diseases there is reason to believe that it is a stage in their development. Moreover, some of these maladies are derived from the lower animals, as favus from cats, glanders from horses, and anthrax from cattle.

Course of infection.—When the virus of an infective malady enters the human body, an interval occurs during which the health of the patient remains apparently undisturbed; this is called the period of *incubation*. In some diseases, as in smallpox, it is almost always of the same length. This fact is of great importance, because it enables us to say, when a person has been exposed to infection, that after the lapse of a certain number of days, if not already attacked, he is safe, and may mix with other people without risk to them. It is, in other words, the foundation upon which rests the practice of *Quarantine*. The duration of the incubation of each specific disease has accordingly been studied with care. The only cases in which it can be positively determined are those in which there has been but a single exposure to contagion; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as proofs of a short incubation; and others again, in which the exposure ceased many days before he fell sick, are valid as proofs of a long incubation. It seems now to be established beyond all doubt, that whereas the incubation of each specific disease is fixed within certain limits for the majority of cases, there are yet some cases in which the limits are overstepped. Thus scarlet fever, which commonly has an incubation of nearly a week, sometimes breaks out within twenty-four hours of the first exposure to its infection. It is undoubtedly hard to reconcile this fact with what is otherwise a reasonable explanation of the interval of incubation, namely, that it is occupied by the multiplication of the virus within the body. According to Nägeli, schizomycetes commonly double their numbers in from twenty to twenty-five minutes at the temperature of the blood. Lister found that the *Bacterium lactis* took about an hour in completing the process of growth and subdivision. In diseases, such as syphilis, in which a local lesion precedes the development of constitutional symptoms, the virus may be supposed at first to multiply itself locally (in syphilis, within the hard chancre) and not to enter the blood in any considerable quantity until a later period. There is evidence that in the rare instances of the syphilitic poison being communicated by vaccination, the resulting chancre has yielded an infective material within ten days afterwards. On the other hand, an unfortunate case once occurred at the Charité Hospital of Berlin, in which variola was conveyed to a patient by the operation of skin-grafting, the graft having been taken from a person who happened to be in the incubation-stage of the disease. This seems to show that from a very early period the whole of the skin contains the virus, which can have reached it only through the blood; and if so, the same thing must occur likewise in the exceptional

instances in which smallpox is inoculated into the skin at a particular spot. In such cases, however, there is developed a "primary" or "mother-vesicle" which precedes the general eruption by some days. It therefore seems to be very unlikely that in any disease the multiplication of the virus during the incubation remains altogether limited to a local lesion, although it may be more active there than elsewhere.

Such speculations touch a question which is still discussed, namely, whether in specific diseases generally the various characteristic local lesions or *foci* are to be regarded as sources of the infection-blood or as consequences. Thus in diphtheria, do microzymes first settle upon the mucous surface of the fauces, or upon whatever part happens to be the seat of the diphtheritic process, and afterwards penetrate its tissues and enter the blood? Some have even thought that in enteric fever the blood is infected from the intestinal lesions; and Liebermeister endeavoured to prove that in yellow fever there is primary hepatitis. But in typhus and in relapsing fever we have diseases to which such a view cannot apply, for there are no characteristic local lesions; and, in the exanthemata, fever and other general infective symptoms precede the cutaneous eruptions by a considerable interval of time. Moreover, in diphtheria itself there is a well-marked tendency for the throat to be affected even when the virus has originally been deposited elsewhere and may therefore be supposed to reach the fauces through the blood.

Physiological action of the microphytes.—With regard to the mode of production of the pyrexia and of the other symptoms of specific diseases by their contagia we know at present scarcely anything. The microbes, in multiplying so enormously as they do, must of course abstract nutrient materials from the blood and from the tissues; and as fungi they must also absorb much oxygen from the blood and lymph. The idea of a "struggle for existence" between these organisms and the tissues of the host was carried out in detail by Nägeli. He argued that when a person is exposed to contagion, his taking the disease or resisting it depends probably in part upon the number of microbes which enter his body, in part upon the condition of his blood and of his tissues at the time. Everyone who has experimentally cultivated the lower organisms knows that when more kinds of organisms than one are present, a slight change in the composition of the fluid in which they are placed may completely alter the result, enabling a microbe which previously was weaker to outgrow that which had been the stronger. And so Nägeli supposed that slight alterations in the state of the blood may greatly favour or oppose a contagion in the competition on which it enters. Moreover, some remarkable observations of Metschnikoff, first made on a minute crustacean, but afterwards repeated on frogs, seem to show that bacteria introduced into the blood either kill the leucocytes, or are themselves absorbed and digested by these guardians of the invaded organisms ('Vichow's Archiv,' Bd. xcvii).*

The analogy of the various fermentations suggests (as Lister has remarked) that, besides appropriating the materials for their own growth, microzymes may also cause further disturbance by catalytically decomposing other substances of which they make no use.

Again, it is certain that, while living, these minute organisms must be

* See also Mr Bland Sutton's 'Introduction to General Pathology' (1886).

constantly giving out or excreting waste products, and these, though excessively small, would, when accumulated by an enormous number of microbes, become a large dose of probably poisonous materials for the host—which may be called "toxines," analogous to the ptomaines produced by putrefactive bacteria. Lastly, it must be remembered that the mere physical results of immense swarms of these microbes may possibly lead to blocking of lymph-channels, or starvation of ganglion-cells, or suffocation of living protoplasm.

Protection.—Can we explain by the "germ theory" of specific infectious diseases the remarkable fact that most of them protect the organism which has once survived their attack from future invasion? There are apparent exceptions to the rule, and the degree and duration of the protection varies for each malady; but most of the specific contagious diseases occur only once in the life of the same individual; and perhaps none of them is entirely devoid of a protecting influence. We must suppose that in the course of each disease the blood or the tissues undergo such a "sterilization" that they no longer afford the conditions requisite for the development of its peculiar microphytes. There are obvious objections to the notion that human beings in general are born provided with a number of different materials which serve no known purpose but that of affording nutriment to the various contagia, in case of their invasion. Nor is it easy to suppose that the leucocytes, when once they have got the better of the invading swarm of bacteria, are so habituated to the attack that they gain an easy victory over any subsequent irruption of the same species, while yet they are as unable as at first to deal with any other species; and that they transmit to their descendants the same power of destruction, with the same limitation, down to a more or less distant generation.

Some remarkable experiments by the late Dr Wooldridge appear to throw light upon this curious problem. He found that by cultivation of a certain bacillus in a neutral fluid the latter became so "exhausted," or at least so altered, that it would no longer support a cultivation.

In some diseases, as in relapsing fever and in diphtheria, the duration of the protective action is perhaps no greater than is just sufficient to prevent the patient from reinfecting himself. In others it lasts during the whole of life. Indeed, an interesting question is whether it may not, in some cases, be transmitted from parent to child. As is well known, the exanthemata often rage with extraordinary virulence when they are introduced into communities that have been free from them for a great length of time. Other explanations of this fact are possible; but the reason may be that the victims come of a stock which has not for many generations been exposed to the contagion. It would be very interesting to investigate to what extent differences in the severity of a specific disease, attacking various individuals under the same conditions, in countries where it is always more or less prevalent, may depend upon whether one or both of the parents of the several patients had had that disease in childhood, or at least before the patients themselves were born. It might even happen, in the case of a malady which commonly affects adults, that successive generations should in turn succumb to it, in consequence of the continued absence of such an inherited protection.

Prevention of infective diseases.—These considerations seem to render

doubtful the advisability of attempting, even if it were practicable, to eradicate infectious diseases from this or any other country, and then to keep them off by a system of quarantine. Sir Thomas Watson gave to such a scheme the weight of his authority. But we cannot "stamp out" scarlatina and cholera as we can anthrax or pleuro-pneumonia in cattle—by destroying each patient as soon as he is attacked; and sooner or later each disease would be sure to find an entrance, and it would probably commit unheard-of ravages among a population long free from it. It is even doubtful whether we ought to teach the public to look upon the maladies in question as in a special sense "preventable." They are certainly not so much so as those which result from over-indulgence in food and drink, from exposure to cold, or from syphilis.

A more promising method of combating infection seems to be that of which we have an illustration in the practice of vaccination. And there is reason to hope that in the future this method may be far more widely extended than hitherto. Pasteur has demonstrated the fact that the virus of anthrax can be so diminished in intensity that it is no longer fatal to cattle, and that after inoculation with this modified or attenuated virus they are no longer susceptible of the disease. The same success attended him in dealing with the contagious disease of poultry (called by a misnomer *choléra des poules*); and recently he has applied a similar method to the prevention, and practically to the treatment, of hydrophobia. We may hope for future advance in the same method, which has made smallpox a preventable though unhappily not always a prevented disease.

There is, lastly, another method by which plague and typhus and ague have been almost banished from England—improvement of the circumstances of life, by which either the organism is better able to resist the invasion of the virus, or the conditions have been rendered unfavourable for the multiplication and even the survival of the virus itself.

Method of transference of contagia.—We have still to consider how the microzymes of contagion or of miasm gain access to the human body.

When suspended in the air, they probably penetrate into the capillaries of the lungs through the stomata of the pulmonary alveoli; they make their way through the mucous membrane of the mouth or the fauces by the mucous crypts; and they enter the blood or the lymph through wounds or abrasions.* After being swallowed they penetrate the mucous membrane of the alimentary canal.

In describing the invasion of the several specific diseases, we shall find evidence that their contagia differ much in the readiness with which they pass the natural barriers of the skin and mucous membrane.

Contagia have traditionally been divided into those which are "fixed" and those which are "volatile." As we have seen, it is certain that none of them are gases or vapours, and that they all consist of solid particles of exceedingly small size. But is it possible that the particles in question are capable of escaping with the water which evaporates from liquids or from moist solid surfaces, and of diffusing themselves in the air?

Nägeli seems to have conclusively demonstrated that this is impossible. He performed a series of experiments with V-shaped tubes connected together. In one bend he placed a liquid suitable for the growth of microzymes; another bend he filled with sand saturated with a putrefying

* It has been supposed that examples of such a mode of infection are sometimes afforded

liquid. Even when air was drawn through the apparatus he found that no microzymes passed from one tube to the other. And on theoretical grounds he shows that no other result could have been anticipated.

We have been accustomed to think that the breath of persons suffering under an infective disease is apt to be highly charged with the virus, and that the offensive odours exhaled from his moist skin, or from his excreta, may be highly dangerous. According to Nägeli all such notions are erroneous; he even declares that the presence of fœtor is generally a sign that the substances which give rise to it are still moist, and are therefore incapable of setting free any microzymes which they may contain. But it is obvious that some parts of an infected mass may remain covered with fluid, while others have already undergone desiccation.

Of course it is not disputed that liquids in which the microzymes of a specific disease are floating may convey it to healthy persons. Indeed there are some maladies, such as hydrophobia and syphilis, which seem scarcely to be communicable, except by a process of inoculation, the poison being introduced into a bite or a wound, or a crack in the skin. And diphtheria is often spread by the direct transference of mucous secretion from one individual to another. It is also to be remembered that the punctures made by insects may sometimes be the means of conveying the virus; cattle, for instance, are liable to be infected with anthrax by gadflies.

Moreover, there is the clearest evidence (although Nägeli denies it) that certain specific diseases are transmitted by means of liquids which are swallowed. So far as drinking-water is concerned, these facts will be stated in the chapters on enteric fever and cholera. Still more interest attaches to the remarkable facts which have recently been made out in this country as to the conveyance of the poisons of enterica and of scarlatina by means of milk, and also of tubercle by means of food. It is probable that, even in water, the specific mycrozymes multiply in the interval between its contamination and its reaching the human stomach. But there can be little doubt that milk affords far more favourable conditions for their growth; for Lister proved in his experiments with this fluid that (unlike even Pasteur's solution) it was capable of serving as a pabulum for almost any kind of microscopic organism. Once only did he meet with a bacterium that could not live in it.

With these important reservations, however, Nägeli is probably right in maintaining that infection generally occurs in a very different manner, namely, by means of microbes which have undergone desiccation, and which rise into the air and remain suspended in it in the form of a fine dust: the particles, perhaps, even more minute than those which become visible in the track of a sunbeam. How little influence gravitation has in causing the subsidence of such particles is well illustrated by the facts that volcanic dust from Vesuvius and from Krakatoa is carried in the atmosphere to distant parts of Europe, and that the trade-winds convey dust across the Atlantic Ocean from America.

The conditions under which microzymes thus become suspended in the air must be discussed separately for miasmata and for contagia.

(1) In the case of a *miasm*, at least any miasm which consists of microbes

by the so-called "surgical scarlet fever," which so often breaks out in children after operations; but, since Dr Goodhart has shown, in the 'Guy's Hospital Reports' for 1879, that its occurrence is not prevented by the strictest "antiseptic" treatment, there appears to be more probability of its arising in the ordinary way.

which live in earth, the first condition of its development is the presence of water in the interior or upon the surface of the ground, and the presence in this water of substances fit to serve as pabulum for the microbes. Further, it is necessary that the level of the water should vary from time to time, so that particles of the soil itself, or the stems and the leaves of plants growing upon it, should undergo desiccation, and should receive deposits of microbes, also in a dry state. So far as the miasm of ague is concerned, there is no necessity for supposing that it is derived from far below the surface of the ground, or for invoking the assistance of any force beyond that of the wind, for the purpose of carrying up the dried microbes into the air. But if any value is to be attached to the observations which Pettenkofer and others have made at Munich with regard to enteric fever and to cholera, microbes concerned in the propagation of these "miasmatic-contagious" diseases must be assumed to come in some cases from the soil at a depth of several feet. The conditions which may be supposed to render this possible are very carefully argued out by Nägeli. In the first place, he thinks that in the ground-water which saturates a porous soil below a certain level, the growth of microphytes is likely to be especially active towards the surface of this water. Hence the slightest fall in the level of the water must cause an abundant settlement of microbes upon stones, sand, and fragments of clay and humus, which come into contact with air as soon as the water recedes from them. In this subterranean atmosphere he conceives that currents are produced by various causes: changes of temperature, changes of pressure, winds, and lastly, the suction action of warm air into the interior of houses having deep foundations. Such currents, he thinks, may easily carry away the dry microbes, especially if there is no precipitate of colloid matter from the water, to make them adhere closely to the substances on which they are deposited. A further condition, at least so far as concerns their escape into the air above, is that the superficial layers of the soil through which they must pass should not be damp, and should not be covered with a uniform carpet of vegetation.

(2) The microbes of a *contagium* doubtless leave the body mainly in the fluid excretions, the saliva, the urine, the fæces, and the sweat. They may also be attached to the cuticle which is constantly being shed from the surface; in the case of scarlet fever and in that of smallpox this mode of diffusion of the poison is believed to be of great importance. In either case their escape is due to no process of "elimination," and is of no advantage whatever to the patient. Indeed, the microbes which find their way out of the body are probably altogether insignificant in number, when compared with those which remain in the blood or in the tissues, and undergo destruction there. The microbes which adhere to fragments of cuticle are already dry; those which are contained in fæces, in urine, or sweat, must undergo desiccation before they can reach the air. Nothing is more favourable to this than the soaking of linen or other fabrics with any of the infected secretions. The microbes remain upon the surface of the cloth when it dries, and are afterwards shaken off by the slightest movement. It is a most natural circumstance, therefore, that washerwomen and their families should be very liable to attack by contagious diseases; and instances in which infection has been traced to the hanging out of clothes to dry are readily explicable if one considers how imperfectly the process of washing is often performed.

Persistence of contagia.—One cannot state with any approach to certainty, how long a patient remains capable of infecting others after his recovery from a contagious disease. Perhaps his secretions cease to contain microphytes as soon as the pyrexia is at an end. This at least appears to be the natural inference from the observations which have been made of a rapid disappearance of *spirillum* from the blood in relapsing fever. But for a considerable time afterwards fresh portions of infected cuticle may be continually shed.

Contagia sometimes adhere with great tenacity to the walls of an apartment which has been tenanted by a patient suffering under an infective disease, and to bedding or clothes which have been used by him. Nägeli mentions that at Munich several masons fell ill with smallpox after scraping the ceiling of a room in which smallpox cases had been treated six or seven years before, and which had then been whitewashed. Sir Thomas Watson relates the following instance: A house in which several persons had been attacked by scarlet fever was left empty for a year. When the family returned, a drawer in one of the bedrooms resisted for some time attempts to pull it open. A strip of flannel had got between the drawer and its frame, and had made the drawer stick. This piece of flannel the housemaid put playfully round her neck. An old nurse who was present, recognising it as having been used as an application to the throat of one of the subjects of scarlet fever, snatched it away and burnt it. The girl, however, soon sickened with the disease. Woollen substances seem to afford the most favourable conditions that can be imagined for the preservation of microzymes in a state of activity, short of their being enclosed in sealed glass tubes. By the hygrometric properties of such substances, contagia adherent to them may probably be prevented from undergoing too complete a desiccation, while they are at the same time protected from currents of air.

Destruction of contagia.—Steps must always be taken to prevent a person affected with a contagious disease from being a source of danger to others. But what can really be done in this direction is not always clearly apprehended.

During an illness the utmost care should be taken to hinder the contagion from being carried away from the sick-room by means of attendants, or in the patient's excreta, or upon the linen of his bed or body, or in any other way. In all probability its escape into the open air through windows or chimneys is of no consequence.

At this time, it is impracticable effectually to disinfect the patient himself or his surroundings, and therefore that the attempt should not be made.

The patient, after recovery, and the nurses also, should be most carefully disinfected; minute pains should be taken to prevent clothes, books, and other articles from conveying contagion; and the sick-room with all its contents should be thoroughly purified before healthy persons are allowed access to it.

The measures which should practically be adopted to ensure these objects seem to be the following:

1. The patient himself must be isolated in a separate room or suite of rooms, which, if possible, should be at the top of the house. An intervening passage, of which the windows can be opened, is, of course, an advantage. Ventilation should be carefully attended to, and it is well that there should be a fire, so as to maintain a draught up the chimney. The doors should be

kept closed, and outside them there should be hung an old sheet saturated with a liquid solution of carbolic acid or chloride of lime, and never allowed to become dry. The addition of glycerine to this liquid is probably advisable. It seems to be useless to expose vessels containing carbolic acid in the sick-room itself, or to scatter chloride of lime upon the floor.

Those who nurse or wait upon the patient should not be allowed to enter other parts of the house, and, if possible, such persons should be chosen for this duty as have already had the disease. The medical attendant should take care by ablutions, by change of clothing, and by exposing himself freely to the open air after each visit, to avoid carrying contagion abroad. One is often asked whether the father of a sick child should be allowed to continue his work in an office or chambers, so as to come into contact with others. Probably if he avoids coming near the patient and takes a walk when he leaves the house in the morning, no appreciable risk is run, so long as he himself remains well.

All curtains, carpets, and padded chairs, all articles of wearing apparel in cupboards or in drawers, all unnecessary articles of furniture of whatever kind, should be removed from the sick-room before the patient is placed in it, because their presence will make unnecessary difficulties afterwards. Only such books or toys should be allowed as are not too valuable to be burnt when they are done with. Any food or drink which the patient or the nurse may leave should be thrown away. Cups, plates, and spoons should be put in boiling water before they are allowed to go back to the kitchen; but, as far as possible, it is well to keep the same articles from day to day in use in the sick-room, and to wash them there.

2. All the excreta of the patient should be rendered innocuous by the addition of some disinfectant.

The experiments of the late Dr Baxter, recorded in the sixth volume of the new series of Mr Simon's 'Reports to the Privy Council,' have shown that few at any rate of the substances commonly used really possess the power of destroying contagia. He employed *carbolic acid*, *sulphur dioxide*, *potassic permanganate*, and *chlorine*. Each of these substances was added in definite proportions to vaccine lymph, and a series of vaccinations were performed with mixed liquids upon children, one arm of the child being inoculated with the lymph supposed to be disinfected, while the other arm was at the same time vaccinated with lymph that had been diluted with water to a corresponding degree. The results which he obtained were: that with chlorine and with potassic permanganate there is no security for effectual disinfection short of the presence of free chlorine or of undecomposed permanganate in the liquid, after all chemical action has had time to subside. In the case of sulphur dioxide the criterion of success is that the liquid should be permanently and strongly acid; in the case of carbolic acid that it should contain at least 2 per cent. by weight of the pure acid. It is further necessary that the disinfectant should be thoroughly incorporated with the liquid; there must be no solid matters capable of shielding the contagious particles. Other effectual disinfectants are thymol, and other aromatic compounds, fluosilicate of soda and—in some respects the most valuable of all—corrosive sublimate.

A point on which Baxter lay stress, as Nägeli had also, is that the addition of disinfectants in too small quantities may do harm instead of good. Probably all the substances that can destroy contagia are also antiseptic agents; that is, they can prevent putrefaction. And it appears

that they are capable of doing this even when present in too small a quantity to disinfect. At first sight this seems contradictory. But it is not really so; for Baxter showed, that when an infusion to which (for example) 2 or 3 per cent. of carbolic acid has been added fails to putrefy, the bacteria in it nevertheless remain alive, and that they begin to multiply if a little of the infusion is mixed with another liquid that can afford them pabulum. Now, it is this power of affecting other liquids, and not its capacity for undergoing putrefactive changes itself, which is really comparable with pathological infectiveness. And it is a well-ascertained fact that (except, perhaps, in the case of glanders) the occurrence of putrefaction in a liquid deprives it at once of whatever contagious property it may possess. Thus, if a disinfectant is added in too small quantity, it may easily happen that its action is precisely the opposite of what is intended: by preventing putrefaction, it keeps the contagious microbes alive.

3. For the purpose of wiping off discharges from the nose or the mouth of the patient, pieces of rag should be used, and they should be burnt immediately afterwards. The sheets and body-linen should be placed in a tub of water as soon as they are changed, and they should of course be washed apart from those of other persons. It is usual to soak them in a weak solution of carbolic acid or in diluted Condy's fluid; but this is probably of little use. The really important point is that they should be submitted to the action of water at a high temperature. The safest plan is to boil them in a copper. But there is reason to believe that a degree of heat far below 212° may be sufficient. Thus, Davaine found that the virus of anthrax diluted with water was destroyed in five minutes by a temperature of 131° F., and Baxter that even dry vaccine was rendered inert in thirty minutes by a temperature of 185° or upwards. There is no doubt that in the dry state contagia resist heat much better than when they are suspended in water.

The linen worn by the nurse should be treated in the same way as that of the patient. The dress should always be made of a material that will bear washing.

4. At the end of the illness, when it is believed that the patient is no longer giving off contagion from his body, he must be carefully washed with ordinary soap. Special pains should be taken to cleanse the hair thoroughly. He should then be dressed in clothes none of which have been in the sick-room, and should be moved into another apartment. Similar precautions should be taken whenever a nurse or an attendant goes away, at whatever period of the case.

5. The room which has been occupied by the patient during the disease must be carefully disinfected before healthy individuals are allowed to occupy it.

In the first place, moveable articles that cannot be washed should, if possible, be exposed to dry heat. The temperature required for the destruction of contagia in the dry state appears to be considerably higher than that which suffices when they are suspended in a fluid. It varies with the length of exposure, the time needed being longer in proportion as the temperature is lower. Some hospitals for infective diseases have hot-air chambers, erected for the purpose, in which there is no difficulty in raising the temperature, even in the centre of a flock-bed, to 220°, 240°, or 300° F. There seems to be no doubt that this must be completely effectual in killing any kind of virus.

The next step is to destroy any microbes that may be floating in the

atmosphere, or adhering to the sides of the apartment, or to the furniture. With this object the fumes of sulphur or chlorine gas may be used. All the openings into the room, chimney, windows, and doors, must be made as air-tight as possible. A good plan is to set fire to a few ounces of bisulphide of carbon in an iron pan supported over a pail of water. Or from a quarter to half a pound of brimstone broken into pieces may be placed in the pan and live coals used to ignite it. The room is then closed and left so for several hours.

Finally, the ceiling should be scraped and whitewashed, the walls should be re-papered, the floor should be thoroughly scrubbed, and all the paint in the room, as well as the furniture should be very carefully washed.*

* Since the first edition of this book appeared many excellent treatises on the subject of microphytes and contagion generally have appeared in English. Of these may be particularly mentioned Dr Klein's 'Micro-organisms in Relation to Disease' (2nd ed., 1889), Dr Crookshank's 'Introduction to Practical Bacteriology' (1886), Mr Watson Cheyne's original papers and his translation of German essays on 'Bacteria in Relation to Disease' (published by the New Sydenham Society, 1886), the chapters on contagion and on bacteria in Professor Hamilton's text-book of pathology (vol. i, 1889), and in the admirable 'Student's Manual of General Pathology' by Dr Payne (1888).

FEVER

Febris: calor præter naturam.—GALEN.

Pyrexia—Thermometry—Clinical course of pyrexia—Hyperpyrexia—Theory of fever—Possibly beneficial effect of pyrexia—Idiopathic and symptomatic fever—Raised temperature without fever—The physiological stages of fever—Concomitant phenomena of pyrexia: nutrition: pulse: respiration: muscles—Subnormal temperature.

AMONG the varied effects of diseases there is, perhaps, none which is more commonly met with—and certainly there is none which is more important—than PYREXIA or FEVER. It is not surprising, therefore, that the ancient writers recognised this condition.

But it is strange that in modern times scarcely any attention was paid to it until recently. The specific fevers were studied; but of the febrile state, common to them and to inflammatory disorders generally, little notice was taken. An increased action of the heart was considered as its chief characteristic.

It would be difficult to overstate the value of an advance which was made early in the second half of the present century, by the introduction into medical practice of the clinical thermometer, as a direct and simple method of determining the presence or absence of pyrexia. This was not, indeed, a novelty. About a hundred years previously the instrument had been used by Boerhaave and by his pupils Van Swieten and De Haen; and De Haen, who was a physician at Vienna, had discovered the striking facts that in ague, during the cold stage, the temperature of the blood is increased, and that the temperature of the body may sometimes rise after death. Hunter made many thermometric observations, and Dr John Davy published extensive tables of temperatures in animals and in man, under varied conditions of age and climate, in health and in disease. But when in 1850–51, Traube and Bärensprung independently called attention to clinical thermometry, the practice was new. From that date Wunderlich, of Leipzig, devoted infinite pains to the study of temperature in all diseases. In this country the practice was soon adopted by Parkes, Ringer, Aitken, and a host of others; and we may safely assert that it will never again be abandoned.

Methods.—The use of the thermometer in clinical practice requires considerable care, if the results are to be relied on as being even tolerably accurate. The instrument is most commonly placed in the axilla, although there are other and often more suitable places, of which we shall speak presently. Now, the temperature which we want to ascertain is that not of any part of the surface, but of the deeper structures of the body, an inch or more below the level of the skin. To determine the surface temperature of any region is quite another, and a most difficult matter. Instruments called surface-thermometers have been constructed, with the receptacle for the mercury of such a shape that one side of it can lie flat on the skin while

the other side is protected with a non-conducting material, so as to diminish as much as possible any loss of heat from it. But to cover up any part of the surface of the body is, of itself, to raise the temperature above that at which it previously stood.

When a thermometer-bulb is placed deeply in the axilla, the fold of the groin, or elsewhere, the mercury goes on rising until it indicates a temperature corresponding very closely with that of the body, at the same distance from the skin as that at which the instrument is placed, and it is uncertain what length of time such an observation may take for its completion. A great deal depends upon whether the parts were closely in contact before the thermometer was introduced. Thus, if the patient is lying on one side in bed, one should always choose the more dependent of the two armpits, since it will give a fixed temperature far more quickly than the other. When a thermometer has once risen to its full height in the axilla, a second one placed in the same spot immediately afterwards will rise to the same point in from three to five minutes. But it is a very different matter in a thin person, or if the arm has been separated from the chest, so that the axilla has contained air, and perhaps a fold of underclothing. It must then itself rise through several degrees before it can bring the thermometer to a stationary point; and the length of time required for this to take place will vary indefinitely, according as the circulation of blood in the peripheral parts of the body is active or otherwise. When the skin is hot and turgid, as in scarlet fever, a comparatively short period will suffice; in the cold stage of ague, and still more during the collapse of cholera, it is doubtful whether an axillary temperature can ever be relied on. It is to be observed that this uncertainty is independent of any defect of sensitiveness in the instrument which happens to be employed.

There is reason to believe that the scientific value of thermometric observations made in this country is impaired by the comparatively short space of time devoted to them. It is commonly thought that from three to five minutes suffice for this purpose; and it must of course be admitted that a temperature above the normal, however quickly obtained, is a positive fact, which from a practical point of view can never be worthless. Dr Bäumler, however, has shown ('Brit. Med. Journ.,' 1864) by direct experiments that, even when all ordinary precautions are taken, it may easily happen that too low a reading by ·3 to ·8 of a degree Fahrenheit is arrived at, if the thermometer is withdrawn from the axilla at the end of five minutes. He found that from eleven to twenty-four minutes are required to give an absolutely trustworthy result. Liebermeister goes still further: for even of a period of from fifteen to thirty minutes, he does not say more than that it suffices for the majority of cases. The rule which he lays down is that the instrument should be observed to remain stationary for several minutes before it is removed. In practice, however, this is impossible, and in ordinary cases we may with due precautions be satisfied that after five minutes only a small fraction of the total increase of temperature fails to be recognised.

It is essential in placing the bulb in the axilla that no clothes should be allowed to remain in contact with it, and that the skin should grasp it firmly. If there be any perspiration, the hollow must first be wiped dry. The arm should then be folded across the chest, and the hand may be made to take hold of the opposite arm, while the opposite hand supports the elbow of the side on which the thermometer is.

When, from emaciation or any other cause, the parts do not meet

closely round the instrument, some other region should be selected. In the case of infants and young children, the *groin* is better adapted than the armpit, for the fat is more abundant at that age, and children who resent the arm being held will often allow the thigh to be kept close to the abdomen without moving.

The *mouth* is a suitable place, if one can depend upon the patient's keeping it constantly closed and breathing entirely through the nose. The bulb may be placed either beneath the side of the tongue or at the back of the cheek. Dr Bäumler found that from nine to eleven minutes sufficed to raise it to a fixed point there.

Of all localities for thermometric observations, the *rectum* is theoretically the best, and though it cannot be often used in practice, yet it should be chosen when there is doubt as to the accuracy of an axillary or oral observation; and it is particularly suited for children and for old people. An important advantage is the saving of time, for from three to six minutes are enough to give a result of scientific value. Consequently it is advisable to make use of the rectum whenever much depends upon the exact temperature at the moment, or when an ice-bath may perhaps be required. And it is to be noted that a rectal temperature can be taken with a registering thermometer while the body is immersed in cold water.

By far the most rapid method of taking the temperature of the body accurately is for the patient to hold the bulb of the thermometer in the *stream of urine* when emptying his bladder. The glass and mercury are rapidly heated, for the cooled fluid passes at once away. The method is most applicable for taking the temperature in the evening before going to bed, and also for ascertaining the presence or absence of pyrexia when a patient is going about or visiting his physician. (See a short paper by Dr Oertmann in 'Pflüger's Archiv,' Bd. xvi.)

When, after the withdrawal of the instrument, the index has been read, it is common to dot down the result on a sheet of paper ruled for the purpose. Observations are repeated at regular intervals twice a day, every two hours, or even oftener, and each successive reading is recorded in a similar manner. Lines are then often traced from dot to dot so as to form what is termed a "chart." It is, however, important to remember that the result is artificial. However short may be the intervals at which the thermometer is applied, there is no reason for supposing that the patient's temperature moves straight upwards or straight downwards from one point to another. And when the intervals are long, as when the instrument is employed only twice in the twenty-four hours, there is not even a probability that the dots marked on the chart each day represent respectively the true maximum and the true minimum. We shall, indeed, presently see that in disease as in health there are, as a rule, certain daily fluctuations, the temperature being generally highest at a particular hour in the evening and lowest in the morning. But even when the observations are made just at those times, there is no security that the rule is observed in that special case on any one day; and it is always highly probable that between each pair of observations there may have been two or three or even several ascents and descents of the temperature which altogether escape notice. Some therefore prefer to make no chart at all, but to place the figures in two vertical lines, one for the morning, and the other for the evening.

The course of pyrexia is naturally divisible into certain periods or stages,

which, however, vary greatly in length in different diseases. First comes the "pyrogenic" or "initial" stage; during its continuance the temperature rises more or less steadily, the rise being interrupted, if it extends one, two, or more days, by the daily fluctuations already alluded to. When it is of short duration or when the rise is at first rapid, there is commonly a shivering fit or *rigor*—the first and important indication of a severe disturbance of the central nervous system.

The second stage is the *fastigium;* in it the temperature reaches its *acme* or highest point; but, not infrequently, since this stage may last for several days or even two or three weeks, it is characterised (independently of the daily fluctuations) by a series of irregular slight ascents and descents, so that the highest point or one very near it may be touched again and again at longer or shorter intervals.

The third stage is that of *defervescence*, during which the temperature falls again to normal. Sometimes, however, it is separated from the second stage by an intervening period, called by Wunderlich the "amphibolic" stage, in which irregular exacerbations and remissions are observed. Or, again, there may at the end of the second stage be a marked rise of temperature termed by Wunderlich the *perturbatio critica*. The third period, that of defervescence, is sometimes protracted, sometimes short and sudden. In the former case the pyrexia has been said to end by *lysis*, in the latter by *crisis*. The rule is to speak of a critical termination only when the fall to a normal temperature is completed within thirty-six hours; but sometimes the third stage begins with a slight and gradual descent, which after two or three days ends in a crisis. When defervescence is rapidly accomplished, it is generally accompanied by profuse sweating and sometimes by an abundant flow of some other secretion.*

After defervescence the temperature often remains for some days slightly subnormal. It is also less stable than in health, being easily disturbed by slight causes, so that, for example, a rise of one or two degrees is apt to follow the first solid meal.

It is often convenient to be able to express in general terms the degree of pyrexia without giving the actual temperature. And for this purpose Wunderlich's classification may be adopted; it is as follows:

1. *Subfebrile*, temperature in axilla 99·5°—100·4° Fahr.; or 37·5°—38° C.

2. *Slightly febrile*, temp. 100·4°—101·3°; or 38°—38·5° C.

3. *Moderately febrile*, temp. 101·3°—102·2° in morning; 101·3°—103·1° in evening; or 38·5°—39° C. in morning; 38·5°—39·5° C. in evening.

4. *Decidedly febrile*, temp. about 103·1° in morning, about 104·9° in evening; or 39·5° C. in morning, 40·5° C. in evening.

* That Wunderlich and others thus have adopted the term "crisis" is perhaps to be regretted. For it is scarcely possible for anyone who is familiar with the medical writings of the ancients to shake off the remembrance of earlier mystical notions which formed an important part of their teaching. When we come to describe the specific forms of fever, we shall find that each of them has a more or less definite duration, and that one can often confidently look forward to a future day, a week or two distant, as that in which defervescence is likely to take place. But this is very far from what the Greek writers or even some moderns have meant by critical days, their idea being that all fevers, without reference to differences between them, ought to come to an end on certain days rather than on others, so that when one critical day was passed, the disease might be expected to run on until the next critical day occurred. The whole of the doctrine in question is without foundation.—C. H. F.

5. *Hyperpyretic*, temp. approaching 107·6° or even higher (42° C.).

A less elaborate but possibly as useful a nomenclature is :—Feverish, 99·5°—101° Fahr. Febrile, 101°—104°. High fever, 104°—106°. Hyperpyrexia above this.

We must, however, bear in mind that in the case of children fever is set up very easily ; an evening temperature of 105° may be due to comparatively trifling causes ; and on the following morning the thermometer may not rise far above the normal point. So, again, sensitive women sometimes show a "highly febrile" condition, when the result proves that no apparently adequate cause for it has been present. On the other hand, in old people the temperature is apt to be below what one would have expected from the gravity of the case. So that high temperature in children is often of no ill omen, while even slight pyrexia is serious in an elderly patient. At what point the term *hyperpyrexia* should begin to be used is somewhat uncertain ; but it is generally understood to mean such a temperature as is sufficient of itself to endanger life if long continued. Cases in which the thermometer rises to 109° or 110° (43·5° C.) are very exceptional, and most observers think that they are invariably accompanied by severe and alarming symptoms. Just before the death of a patient from tetanus Wunderlich obtained a temperature of 112·55° (44·75° C.) ; and this perhaps remains the highest recorded point that has certainly been reached.

Paradoxical temperatures.—A few instances have, however, been recorded in this country which, if they can be relied on, seem to show that far higher temperatures have occurred, not only without being followed by death, but sometimes without being attended with serious symptoms.

The first case of this kind was observed by Mr J. W. Teale ('Clin. Soc. Trans.,' 1875) in the person of a young lady, who by a severe accident had several ribs broken, and afterwards suffered from great tenderness over the dorsal vertebræ. Two months later her temperature was one day taken at 110° ; and afterwards the index of the thermometer was on four occasions buried in the bulb at the top of the instrument, at a point above 122°. Sometimes these extraordinary temperatures were taken in the axillæ, sometimes between the thighs, or even in the rectum. She was at first in an exceedingly weak state, but she gradually improved and regained fair health.

Other examples of "paradoxical temperatures," as they have been called, have been met with by Dr Donkin ('Brit. Med. Journ.,' 1879). His first case was that of a nurse who was recovering from enteric fever, when the thermometer was found one night to register 110°. Afterwards very high temperatures were repeatedly taken, on a single occasion one of 111·6°, yet no symptoms could be discovered accompanying this reading beyond a feeling described by the patient as one of "flushing" or "rushes of heat." But perhaps the most singular circumstance of all was the evanescent character of this pyrexia, if it deserves that name ; once the thermometer rose to 107·2° in the right axilla, whereas five minutes later it stood at 98·6°. In the mouth a temperature of 106° was once observed. It does not appear that the instrument was ever held *in situ* while such extraordinary results were being obtained, but the patient's hands were watched, and the idea of imposture was present. Seven other cases are cited by Dr Donkin, all but one of them being in females.

In 1879 a remarkable instance of this kind occurred in Guy's Hospital under Dr Moxon. The patient, a girl of 22, had been in the ward for phthisis

during ten months, when on the evening of July 25th her temperature was taken at 107·4° and about an hour afterwards at 110·8°. She appeared to be suffering somewhat from dyspnœa. On the following morning the thermometer stood at 99·8°. During the next few months the most extravagant variations of temperature were recorded. On one occasion Dr Mahomed obtained simultaneously a reading of 102° in one axilla, and of about 114° in the other axilla, one of 107° in the mouth. On changing over the instruments the highest temperature was attained in the axilla where it had before been lowest, that of the mouth being now 104°. Another day a small registering thermometer gave 102·6° in one axilla, while another one in the other axilla gave 109·4°; but directly afterwards, when two large instruments without indices were used, and when the patient's arms were held all the time, the temperature stood at 103° on each side. Dr Mahomed noted that the skin always felt moist and of the ordinary temperature, even when a very high reading of the thermometer had just before been obtained. He never got a high temperature with a non-registering thermometer, when he himself held the instrument in the axilla, keeping his hand pressed against the patient's arm. It is certainly difficult to avoid the conclusion that, in this case, some deception was practised, although its nature was never discovered. The girl died of disease of the lungs on March 22nd, 1880. Can a patient without being observed, squeeze or rub the bulb of a thermometer, so as to drive the mercury up?

In these cases of abnormally high temperatures the pulse and respiration did not rise in any like proportion, there was no delirium or febrile condition of the urine, and the patient did not die.

Theory of fever.—In studying the nature of pyrexia, we may, in the first place, safely assume that the sources of febrile heat do not differ in kind from those which normally keep the human body at a temperature above that of the external air: that is to say, that they are chemical and depend on oxydation of the food which is consumed. Is this production of heat increased in pyrexia?

In 1863, the late Prof. Traube propounded the theory that the amount of heat generated during fever is the same as in health, so that pyrexia would consist in a diminution in the amount of heat given off from the body. His theory has since been shown to be incorrect, but it formed the starting-point of numerous observations and experiments, by which our knowledge has been greatly increased.

In pyrexia, when the temperature has risen to a certain point, it is often stationary there for some hours; and for several days it may oscillate upwards and downwards, always remaining above normal; exactly as in health it oscillates above and below 98·4°. So that in fever, as in health, the regulation of temperature still goes on, although the point at which the thermometer stands is different. Liebermeister, in 1864, took advantage of an attack of tonsillitis in his own person to demonstrate this fact very clearly. His temperature having risen to between 102° and 103°, he exposed his body to cold air, and washed himself with cold water; and he found that this caused a slight rise of the axillary temperature from contraction of the superficial vessels and diminution of loss of heat from the skin, exactly as would have occurred under normal circumstances. The same observer, in 1868, by noting the changes in the temperature of the water of a cold bath, in which he placed a man suffering from acute pneu-

monia, was able to calculate that the immersion caused a greatly increased production of heat, far more heat being given off than would have corresponded with the mere lowering of the temperature of the patient.* So also, as Cohnheim remarks, one may throw a fever patient into the most profuse perspiration by the subcutaneous injection of pilocarpine, when the loss of heat from the cutaneous surface must necessarily be enormously increased, and yet no fall in his temperature follows.

Dr Burdon Sanderson in his lectures on fever in 1876 gave abundant evidence that pyrexia does not depend on diminished dissipation of heat (which may sometimes be increased), but on disorder of the thermotaxic mechanism.

The heat of fever, like that of warm-blooded animals in health, depends ultimately on oxidation (and other subordinate chemical processes) which takes place chiefly in the muscles and next in the liver and secreting glands, but to some extent in all living tissues. The dissipation of this heat takes place only to a small extent by conduction and radiation, but chiefly by evaporation, and this partly from the lungs, but chiefly from the surface of the body. The regulation of thermolysis, or loss of heat, is accomplished partly by the nervous mechanism of respiration, but chiefly by the vaso-motor and vaso-dilator nerves diminishing the amount of blood which circulates in the liver and hot internal organs on the one hand, and increasing the flow through the comparatively cool integuments on the other. In fever it is now ascertained that more, not less, heat is often given off than in health; but the essential point is that more heat is also produced than in a healthy person, taking so little food as a febrile patient does. The regulation of both thermogenic and thermolytic processes is thrown out of gear. The thermogenesis is increased beyond the power of the thermolysis to cope with. The nervous mechanism of the latter process is known: that of the former is more obscure, but there is reason to believe that both catabolic and anabolic nerves exist, to increase and to check the thermogenesis of the muscles. This part of the theory of fever was ably discussed by Dr Donald MacAlister in the Gulstonian lectures for 1887.

Physiologists, experimental pathologists, and clinical observers are agreed as to the existence of thermotaxic and thermolytic centres; and some progress has been made in determining their exact seat. The motor area of the cortex near the fissure of Rolando, the corpus striatum, the grey matter of the cord, and particularly the mesencephalon, are the parts which experiment and pathological observation indicate.

Dr H. C. Wood, of Philadelphia, by experimental researches on the subject made important additions to the previous evidence on this point.

Dr Hale White published in the 'Guy's Hosp. Reports' for 1884 (vol. xliv, p. 49) an interesting essay illustrating the theory of one or more "heat centres" in the brain and cord by a series of clinical cases, some of increased, others of subnormal temperature.†

A striking illustration of the fact that the physiological process of heat-regulation is still in action during the course of pyrexia, is afforded by the

* The accuracy of this calculation has since been disputed by Winternitz. "Der Einfluss v. Wärmeentziehungen auf die Wärmeproduction," 'Med. Jahrb. Wien,' 1872; and 'Virch. Arch.,' Bd. lvi, S. 181.

† See also Riegel's original paper, "Über den Einfluss des Centralnerven-systems auf die thierische Wärme" ('Pflüger's Archiv,' 1872), and Dr. Isaac Ott's recent review in the 'Proceedings of the American Neurological Association' for 1887.

persistence of the regular daily fluctuations of temperature. Wunderlich, indeed, formerly thought that the range of these fluctuations was wider than in health, even when the fever was such as would be commonly called continuous; but Jurgensen and others seem to have shown that they are identical with the normal fluctuations in every respect except that they take place at a higher thermometric level. In pyrexia, as in health, the rule is for the temperature to rise more or less constantly during the day, and to fall during the night. The minimum occurs at about 6 or 7 a.m., the maximum at about 6 p.m. The range of fluctuation appears to be generally as much as 1° C., or even a little more, so that it approaches 2° Fahr.

According to Liebermeister, these daily curves of temperature are probably due to the influence of muscular exertion and of food upon the body, notwithstanding certain obvious objections to such an interpretation of them. One of the objections is that healthy men work and eat to a much later hour in the day than 6 o'clock, p.m., after which the temperature begins to fall; this Liebermeister meets by what is termed the "principle of compensation," according to which every rise in temperature tends to be followed after an interval by a fall below the normal point; he supposes, in fact, that the power of food and of exertion to raise the bodily heat is exhausted during the early part of the day, after which the inevitable recoil takes place in spite of them. The other objection is that the daily fluctuations do not disappear when a person remains in bed all day, and takes no food. Liebermeister accounts for this by appealing to the influence of habit. Evidently the persistence of the fluctuations during pyrexia, when the patient is perhaps absolutely helpless and has fluid nourishment at regular intervals through the twenty-four hours, requires and admits of the same explanation.*

Clinical conditions of pyrexia.—The chief conditions under which pyrexia occurs, fall naturally into two groups: sometimes it is "symptomatic" or secondary to a local inflammation, sometimes it is "idiopathic" and "essential," and depends on the entrance of a contagion into the blood. It has been experimentally shown that the division of the nerves of the limb of an animal is without effect in preventing the development of fever as the result of local inflammation. The inference seems to be inevitable that inflammatory fever is caused by the entrance of a morbid agent into the blood from the inflamed tissues, and this entirely accords with the observations of Billroth and Otto Weber, who, so far back as 1864, showed that pyrexia could be produced by injecting into the blood either fresh pus or decomposing substances of various kinds.

Secondary pyrexia, however, does not always depend on inflammation.

* "The view that pyrexia is only a modification of a physiological process accords well with a doctrine which is gaining ground, namely, that this morbid condition fulfils a useful purpose in disease. The conception of fever as having a salutary influence is, indeed, as old as Hippocrates, and it can be traced all through the Middle Ages, and down to our own time; but our immediate predecessors recoiled from explanations teleological. It is only since the contagious principles of some at least of the specific diseases have been shown to be living organisms, that it has been possible to understand how pyrexia may bring about its own cure, by destroying the very agents which set it up."—C. H. F.

We must, however, remember that some microbes flourish best at a temperature above that of the blood in health, and only some of them are destroyed by a temperature equal to "high fever." Moreover, as Dr Andrew observes, although the hyperpyrexia of rheumatism appears to remove, at least for a time, other symptoms of the fever, yet a high temperature is of no good omen in practical experience when it accompanies enteric fever, scarlatina, or pneumonia.

If there be centres for heat-regulation in the cord, nothing is more likely than that cerebral hæmorrhage or the status epilepticus should cause, what we find clinically, symptomatic pyrexia without inflammation. On the other hand, there is sometimes an absence of pyrexia as an accompaniment of intense inflammations (especially of the meninges and of the peritoneum) which can hardly be accounted for except on the supposition that some counter-influence is exerted upon these thermotaxic centres, either directly or through nervous channels.

The irregular fever of idiopathic anæmia, of leuchæmia, and of Hodgkin's disease has not yet received a satisfactory explanation. That which accompanies the various forms of acute tuberculous affections probably depends upon rapid multiplication of the characteristic bacilli, or in some cases on extensive concomitant inflammation.

There are certain rare cases of pyrexia which agree in having no local inflammation as a cause, and in not being specific and contagious, which run an irregular course and are not accompanied by disturbance of the pulse, appetite, and other functions commensurate with the temperature shown by the thermometer. These cases are more common in women than in men, and in young adults than in others; and are sometimes connected with neurotic symptoms, anxiety and depression of mind, or actual hysteria. They appear to be of favourable prognosis even when the temperature reaches 104° Fahr. or higher, and may conveniently be styled *febris nervosa*.

According to Liebermeister's definition of fever, the mere fact that the temperature of the body is raised is not of itself a proof that fever is present. In tetanus the temperature is sometimes exceedingly high; but Dr Parkes, in his Croonian Lectures for 1871, suggested that the heat of the body in that disease might perhaps have a different origin from that of the specific and symptomatic fevers. A similar opinion was held by Cohnheim, who would also exclude from pyrexia the phenomena of sun-stroke. In each case it is probable that what occurs is not a shifting upwards of the point to which heat-regulation is adjusted, but a generation of heat in excess of that with which the ordinary mechanism of heat-regulation is able to deal. The same view may perhaps be taken of hyperpyrexia in general, the evidence being that the temperature neither remains stationary at a very high point, nor continues to oscillate backwards and forwards, but either goes on rising until death occurs, or falls again to a moderate level.

We may then, provisionally, arrange the conditions under which we meet with raised temperature in practice somewhat as follows:

1. Irritative inflammatory fever, probably due to poisoning of the regulating nervous centres by "ptomaines," or other soluble chemical principles derived from an inflamed part. The pyrexia is moderate when the skin or mucous membranes are affected; moderate or sometimes absent in idiopathic or "simple" inflammation of the serous membranes, and in even acute inflammations of certain viscera, as the liver and the kidneys; but always present and usually high in inflammation of the lungs and in acute tonsillitis; absent in chronic inflammations; often much increased by pain, as in cases of acute synovitis, of iritis, of syphilitic periostitis, of orchitis and inflammation of the ovary.

2. Suppurative or septic inflammatory fever. This is much higher and more constant than when the inflammation of which it is the symptom is non-purulent. It is most marked when pus is closely confined and subsides

on its liberation; but is rarely absent with purulent inflammation, whether of the connective tissues or bones, the brain, liver, and kidneys, or the serous and synovial membranes. It may be regarded as partly due to the same causes as the "irritative" form, partly to septic absorption, and that probably of microbes, as well as of toxines.

3. Pyæmic fever, where there is superadded to the pyrexia of suppurative inflammation that due to intense septicæmia with embolism.

4. Specific primary or idiopathic fever, produced by the swarms of special microphytes (or their secretions) affecting the heat-regulating centres. With these cases we must range by analogy fevers like typhus and ague, in which no microphytes have been certainly discovered, and also perhaps rheumatic fever.

5. The irregular fever of the most severe forms of anæmia—Addison's idiopathic or "pernicious" variety, Virchow's leuchæmic or splenic, and Hodgkin's lymphatic anæmic.

6. Those febrile conditions which appear to be primarily nervous in pathology, including *tetanus*, *heat-stroke*, and any genuine cases of *paradoxical temperature*.

7. Lastly, the cases of continued pyrexia above described which may provisionally be grouped together as *febris nervosa*.

Course of pyrexia.—In the *initial stage* of fever, there is no question that the loss of heat from the cutaneous surface is greatly diminished. As there is no reason for supposing that the evaporation from the lungs undergoes a proportionate increase, we are probably justified in assuming that the total loss of heat is much less than under ordinary circumstances. We have seen that, as De Haen long ago pointed out, the temperature of the deeper parts of the body during a rigor is actually higher than normal, and in fact rises rapidly. Now, since the introduction of the thermometer into clinical practice, this has commonly been taken as proof of the false conclusion that the painful feeling of coldness of the limbs, and of the skin generally, is merely a "subjective sensation produced by the state of the peripheral nerves." The truth is that the low temperature of the surface is just as much a physical fact as the high temperature of the blood in internal organs. One has only to feel the shrunken hand of a man in the cold stage of ague to be satisfied that he is under no illusion when he complains of being cold, and a surface thermometer affords precisely the same indication by the slowness with which it rises above the temperature of the surrounding air, and the comparatively low point which it reaches, however much time may be given to it. Thus Schülein, among certain observations of surface temperatures (to be presently cited), gives a case of tertian ague; and in his chart it is most striking to notice, how, during each attack, at the precise moment when the temperature in the axilla was rising from 98° to 104° or 105° F., that between the toes fell still more sharply, from 95° to 86°, or even below 77°. The cause of the chilliness experienced by such a patient is that the peripheral arteries are contracted and do not allow of the passage of a sufficient quantity of blood to warm the superficial tissues, and to compensate for the loss which is always going on by radiation and conduction, to whatever extent the exhalation of fluid from the skin may be diminished. A necessary consequence is a great fall in the temperature of the skin, and one must not forget that this in turn involves a lowering of the amount of heat-loss, which affords one of the means by which the rapid rise of tempera-

ture during rigor is brought about. It has, however, been calculated by Zimmermann and by Liebermeister that there is, besides, a very great increase of heat-production during this stage.

During the *fastigium* the temperature of the body may remain stationary, or at any rate undergoes comparatively gentle oscillations, so that for its maintenance no such antagonism between heat-production and heat-loss is required as for the rapid rise in the initial period. However, in many instances there is still a marked distinction between the temperature of the skin and that of the deeper structures. These cases afford special proof of the importance of the clinical thermometer, inasmuch as the axillary or the rectal temperature is found to be above normal while the patient's skin feels cool to one's hand and he has no sensation of feverishness. But the comparatively low temperature of the surface is a reality; and since the heat-loss by conduction and radiation is certainly less than when the skin is hot, it is fair to conclude that for the same degree of internal temperature the production of heat must also be less. On the other hand, there are certain diseases in which, when one places one's hand upon the patient's skin, one feels it to be pungent and burning in a very remarkable degree. Addison used to teach that in acute pneumonia the skin possessed a heat which was not observed in any other disease except perhaps scarlet fever. When the thermometer was introduced into practice his dictum seemed to have no physical foundation. An attempt was made to explain away the difficulty by saying that the surface was peculiarly dry in acute pneumonia; but this obviously was beside the question, since at any temperature above that of one's own hand a moist skin must undoubtedly feel hotter than a dry one, if it were possible to prevent the temperature from being lowered by the evaporation which inevitably occurs. The true solution of the difficulty is that in acute pneumonia the temperature of the surface is maintained at a point which is very nearly as high as that of the deeper parts, instead of being far below it, as in most other diseases, even when there is no moisture upon the skin. This was established by Schülein, as the result of a series of careful observations published in 'Virchow's Archiv' for 1876, upon the relation between surface temperature and internal temperature in various diseases. His method was to insert a thermometer with a very small bulb between the first two toes, fastening them together by means of an elastic ring. And he found that whereas in other diseases (including enteric fever, acute rheumatism, and phthisis) the instrument in this position always indicated a much lower temperature than one placed in the axilla, there were three diseases in which the difference was very slight indeed, namely, acute pneumonia, measles, and scarlet fever. In these almost every movement of the axillary temperature upwards or downwards was accompanied by precisely the same movement of the temperature between the toes; whereas no such relation could be traced between the two sets of observations in any other febrile complaint. It must, however, be remarked that in many diseases surface temperatures are from time to time taken which are absolutely as high as those in acute pneumonia; and it is probable that a still more striking confirmation of Addison's doctrine would be obtained if we were only to note the rapidity with which a surface-thermometer rises, when applied to the skin in different complaints. For in that way we should be estimating the amount of heat which the surface is giving off by conduction; and it is this that one's hand appreciates when laid upon a patient's skin. It is almost certain that at least one other disease would have to be added to

those enumerated by Addison, namely, acute rheumatism when complicated by hyperpyrexia.

It is evident that in order to maintain such a uniform high temperature of the superficial as well as of the deeper parts, in spite of the great loss of heat which must necessarily occur, the generation of heat must be far greater in all these complaints than in others in which only the interior of the body is raised to a similar degree of heat. So, also, when the skin perspires freely during the *fastigium*, the temperature within remaining high, there can be no doubt that the production of heat must be proportionately increased.

During the stage of *defervescence* the profuse sweating which so often accompanies it, when crisis takes place, plays an important part in bringing about the rapid fall of internal temperature. But it is not to be regarded as the cause of the defervescence; the fact being that the state of pyrexia has come to an end, and that the heat-regulation is, therefore, no longer set for an abnormally high point.

Concomitants of pyrexia: tissue-change and wasting.—The sources of febrile heat are, as above stated, identical with those which maintain the normal temperature of the body. Science has now advanced beyond the point at which Virchow stood when, in 1854, he declared that the elevation of temperature in pyrexia "must arise from an increased tissue-change." Liebermeister and other observers have indeed shown that the amount of urea excreted in the urine during fever surpasses by at least 70 per cent. that which is voided by a healthy person living on the same diet. Careful observations by Ringer in a case of ague established the fact of the same excess of excretion of urea for intermittent pyrexia. But it is now well known that the production of heat may be largely dependent upon the increased oxidation of various substances, such as sugar, which have never formed part of the substance of the body. Both Leyden and Liebermeister have proved that the excretion of carbonic acid gas also is excessive in fever, the increase amounting probably to at least 50 per cent. It is essential to bear in mind, however, that neither the quantity of urea, nor that of carbonic acid, is nearly so great as that which may be discharged without any rise of temperature by a person who is taking active exercise, or even by one who has eaten a large meal of animal food with much fat. Thus we are brought back again to the same point as before, namely, that pyrexia cannot be due to any special rate of heat-production, but must depend upon an alteration in the point for which heat-regulation is set.

There is, however, no more striking feature of pyrexia than the wasting of the body which accompanies it; and this often seems to go on quite as rapidly as one could have expected, even if Virchow's dictum had been true in the strictest sense. Formerly there was a tendency to pay too little attention to fever as being, in itself, a cause of loss of weight, and to ascribe this result rather to a drain of pus, or of any other discharges which might occur in a particular case. It may therefore be worth while to cite a case of Liebermeister's in which a patient who had an abscess discharging about three ounces of pus daily, lost from fifteen to twenty pounds in weight during two months, while his temperature was at 101° or 102°, but regained it afterwards when he had no fever, notwithstanding that the suppuration still went on as before. No doubt an inability to digest food plays an important part in bringing about the wasting; it is well known that the secretion of milk ceases during pyrexia, and one may fairly assume that there is a similar

interruption in the formation of saliva (to which, in part, the dryness of the mouth is due), and also of the gastric and intestinal juices. That it is not merely a question of loss of appetite appears probable from an observation of Niemeyer's, who, comparing together a healthy man and one suffering from fever during a period of two days in which they lived on exactly the same food, found that the latter lost much more weight than the former. Liebermeister remarks that the wasting is sometimes concealed by the presence of an excessive amount of water in the tissues: he has observed that from this cause the weight of the body may be raised by as much as ten pounds, although there may be only a very slight œdema of the ankles to indicate the presence of such an abnormal condition. He believes that persons who are in good health and well nourished invariably lose flesh when they become the subjects of pyrexia; but he admits that those who are already wasted may undergo no further emaciation, and may even gain weight in spite of the supervention of a febrile illness. According to a calculation made by Leyden, the daily loss of substance in fever amounts on an average to 7 parts per 1000 of the whole body-weight. Cohnheim has pointed out that, if this be correct, it should take only about eight weeks of a tolerably severe fever to kill a fairly nourished man by the mere destruction of his tissues; at least, if one may appeal to the observations of Chossat, which showed that to the higher animals a loss of 40 per cent. of their weight is directly fatal.

The blood doubtless wastes simultaneously with the solid structures, and Parkes and others have supposed that the excess of pigment in the urine depends upon an undue disintegration of red discs. Hæmometric observations, however, by Baxter and Willcocks, lead to the conclusion that, with a single exception, febrile diseases cause no diminution in the proportion, either of discs or of hæmoglobin, in a given amount of blood. The exception is ague, in which, according to Kelsch, there is a great decrease of corpuscles. In other febrile states the whole volume of the blood is lowered, while the relation of its fluid and solid constituents remains normal.

Increased rapidity of pulse.—We have seen that it is only of late years that elevation of temperature has been recognised as the characteristic and essential feature of pyrexia. Previously, acceleration of the pulse, rapidity of breathing, diminution of the secretions, and a variety of other phenomena, were supposed to be not less important indications of it. Now, it is a matter of some consequence to determine whether these several symptoms are directly dependent upon the increased heat of the body, or whether they must be regarded as independent effects of the various causes of fever. In the opinion of the best modern writers, the rapid pulse which generally accompanies pyrexia is a result of the heightened temperature; Cohnheim adduces in support of this view the well-known fact that the frog's heart beats more quickly in proportion to the warmth of the surrounding air. Liebermeister insists on the effect which hot and cold baths respectively have, when they raise or lower the temperature of the body, of simultaneously accelerating or slackening the pulse. He even lays down as the rule, based upon a considerable number of observations (which, however, vary widely among themselves), that for each degree centigrade above the normal temperature there should be a rise of the pulse by eight beats above the standard rate of eighty. It is admitted, of course, that various other

circumstances may affect the pulse-rate of febrile patients, as of healthy persons, or of those suffering from other diseases. Cohnheim insists on the effect which stimulation of the vagi may have in slowing it, and cites the fact that in basilar meningitis the pulse is often infrequent in spite of high fever; but neither he nor Liebermeister seem to have looked out for those negative instances by which their theory could best be brought to the test, namely, the exceptional cases in which a severe inflammatory process, or some other condition that usually gives rise to pyrexia, happens to run its course without elevation of temperature. Such instances are sometimes met with in peritonitis, in diphtheria, and in meningitis; and then the pulse is often, though not always, quickened. Does not this cast some doubt on the pulse-rate being dependent on the pyrexia?

It is important to observe that the rapidity with which the blood flows in pyrexia, and the arterial *blood-tension*, are by no means proportioned to the acceleration of the heart's action. At the commencement of fever, when the pulse feels full and hard, it is probable that the circulation is often really more active than in health. Much stress was formerly laid upon the "*sthenic*" character of such cases. In the 'Med. Times and Gaz.' for 1873, Dr Mahomed has given sphygmographic tracings of the pulse in various forms of fever, showing that this term is not without meaning; for in certain cases the vessels are full, the arterial tension is high, and the pulse bears considerable pressure. It is therefore a mistake to say that a febrile pulse is necessarily dicrotic. On the other hand, when pyrexia has lasted for any length of time, it almost always assumes an "*asthenic*" type, and in many instances this is the case from the very first. It would then seem that the circulation is more sluggish than natural, one proof of this being that the blood tends to stagnate in the more dependent parts of the body, so that what are termed hypostatic congestions arise in the lower lobes of the lungs, and beneath the integument of the back and buttocks, and in the dependent part of the legs as the patient lies in bed. The pulse still remains quick, but it is now soft, feeble, and often small. The sphygmograph shows a highly characteristic condition of arterial relaxation, with low pressure; dicrotism becomes a prominent feature; and in many cases the pulse is even hyperdicrotic. In fact at this stage of fever the heart, although its beats follow one another so quickly, empties itself incompletely and discharges less rather than more blood into the arteries. Often, indeed, thrombi form in those parts of the heart's chambers which are farthest from the orifices.

Until recently, the impairment of the cardiac beats was held to be sufficiently explained by the fact that its muscle, like all the other tissues, becomes badly nourished as pyrexia goes on. Of late, however, some observers have thought that the cause of the enfeeblement of the heart's action in pyrexia is not merely its wasted condition, but the occurrence of definite morbid changes in its texture. We shall find that in cases of enteric fever an affection, known as "parenchymatous degeneration" or "cloudy swelling," is found in the liver, in the kidneys, in the heart, in the voluntary muscles, and elsewhere. This, however, is by no means peculiar to any one febrile disease, and Liebermeister and others have maintained that it is the direct result of elevation of the body heat, reaching a certain degree of intensity, and protracted over a somewhat long period of time. This view is supported by the experiments of Dr Wickham Legg, who found ('Path. Trans.,' vol. xxiv) that the liver, the heart, and kidneys were

granular in animals killed by the action of heat, even when its duration was only for a few hours. On the other hand, Cohnheim maintains that, although guinea-pigs kept for several days in a high temperature exhibit a fatty change in their tissues, there is nothing really resembling "cloudy swelling." He insists on the absence of such an appearance in acute pleuro-pneumonia, and its occasional presence when there has been no fever (as in carbonic oxide poisoning) as proofs that Liebermeister's opinion is erroneous. He even declares that the statements as to the frequency of degenerative changes in febrile diseases are overdrawn, and that in a large majority of the bodies of those who have had intense pyrexia, fatty gland-cells and muscle-fibres are not at all more common than they would have been if death had taken place in a different way. The question is one of great importance, and bears upon the supposed causes of cardiac dilatation and of permanent failure of the muscular substance of the heart.

Other febrile symptoms.—The increased frequency of the *breathing* in pyrexia is also attributed to the action of heated blood upon the respiratory centre; and this view is supported by a very interesting experiment, performed by Goldstein in Fick's laboratory, of warming the carotid blood alone; he found that the respiration was thereby accelerated. Cohnheim adduces in illustration of this theory, the fact that in acute pneumonia the breathing ceases to be rapid after the crisis of the fever, notwithstanding that the affected part of the lung still remains hepatized. Apart from pyrexia there is, however, no doubt of the close dependence of the regulating centre of respiration on that of the heart.

Cohnheim is indisposed to admit that the *cerebral symptoms* which attend so many cases of fever are dependent solely upon the increased temperature of the blood, and of the substance of the brain. He points out that in relapsing fever the thermometer rises to a great height with but little disturbance of the sensorium, and that in febrile diseases generally no relation can be traced between the intensity of the fever and the severity of the delirium or stupor. He does not even allow that the question is settled by the marked effect of a cold bath in restoring the mental faculties of a patient suffering from fever; for, he observes, it may be a consequence rather of the improvement of circulation which results from the bath.

Obscure in some points as is the connection of these disturbances of other functions with pyrexia, there is no doubt of their practical importance at the bedside; and it may be well briefly to enumerate them before leaving the subject.

In addition then to the raised temperature which is the constant and governing phenomenon of fever, we observe to a greater or less extent, according to the severity and duration of the pyrexial state: (1) Acceleration of the pulse and respiration; (2) thirst; (3) disturbance of secretion, and probably as results, (4) loss of appetite, a dry and furred state of the tongue and constipation; (5) scanty and high coloured urine rich in urea, often albuminous, and depositing lithates; (6) pains, particularly headache, not specially frontal, vertical, or occipital, but central and deep seated; and also muscular pains in the loins, back, and limbs; (7) delirium; (8) emaciation.

The *treatment* of pyrexia by baths and antipyretic remedies will be most usefully discussed in the sections on enteric fever and acute rheumatism, which offer the most frequent and urgent occasions for their use.

Subnormal temperature.—It is an interesting question whether the tempe-

rature of the body ever undergoes a change in the direction opposite to pyrexia, the heat-regulation centre being set at a point below the normal instead of above it. We shall hereafter find many instances in which a thermometer placed in the axilla remains unduly low, but probably in these cases the internal organs generally maintain a temperature nearly as high as ever, at least until death is obviously impending. Cohnheim says that this is the case with those who suffer from inanition as the result of stricture of the œsophagus, of starvation from other causes, or of extreme anæmia. Even were it not so, we should still have to show that an alteration in the heat-regulation had occurred, and that the failure to maintain the normal temperature was not a result of an insufficient production of heat. Such a lowering of temperature by "*force majeure*," as Liebermeister terms it, is well seen in persons who are picked up insensible in the streets during cold weather. Cohnheim alludes to cases of this kind, in which the thermometer in the rectum has not risen above 86°, 79°, or even 75° F. The pupils are then dilated and sluggish, while the pulse and the respiration are greatly reduced in frequency; but unless things have gone too far for recovery to take place, such patients under suitable treatment regain their normal temperature within a few hours, and sometimes pass into a condition of reactive pyrexia.

The two conditions, in which (apart from inanition and from the reaction after pyrexia) the temperature is most frequently found subnormal, are diabetes and the stage of collapse in cholera. It is also habitually subnormal in the curious affection described by Sir William Gull as a cretinoid condition in adults, and since named myxœdema. The lowest temperatures, however, have been observed in certain cases of injury to the cord: in one recorded by Mr Hutchinson the temperature in the rectum was not above 80·6° F.

INFLAMMATION

Notæ vero inflammationis sunt quatuor: rubor et tumor, cum calore et dolore.
CELSUS.

Historical review: Virchow: William Addison, Waller and Cohnheim: Goodsir—Direct observation of inflammation in animals—Redness, swelling, pain, heat, pyrexia—Exudation: membranous, catarrhal, purulent, diphtheritic, gangrenous inflammation—Repair—Causes of inflammation: irritants and predisposition—Chronic inflammation—Granuloma.

No one, at whatever period of the world's history, could have applied himself to the study of disease, without seeing that the hot, painful, reddened state of the eye which follows the entrance of a particle of dust within the lids, is typical of a morbid change that is of frequent occurrence in every part of the body accessible to observation. Hence it is not at all surprising that before the end of the second century we find, in the writings of Galen, inflammation (φλεγμονή) already characterised by its four cardinal symptoms: heat, redness, swelling, and pain. To use the words of the late Prof. Hughes Bennett, inflammation has been in all ages "the pivot upon which medical philosophy has revolved." But, for that very reason, there is little use in our now attempting to recapitulate the different views and theories with regard to it which have been proposed. It is scarcely too much to say that only the most recent advances of pathology and histology have rendered possible a real understanding of inflammation.

Very soon after the promulgation of Schwann's doctrine, in 1839, that nucleated cells constitute a principal element of animal structures, pathologists discovered the presence of these bodies in inflamed parts; but at first it was supposed that they developed themselves in the interstices of the tissues by molecular aggregation, in accordance with the revived opinion that this was a common mode of origin for cells in general. Virchow, however, in his masterly lectures on 'Cellular Pathology,' published in 1858, laid down as a dogma, *omnis cellula e cellulâ*. Upon this foundation he erected a complete theoretical system, assuming that the cells of the connective tissues throughout the body undergo multiplication or "proliferation," so as to generate the "exudation-cells" or "pus-cells" of inflammation.

Even before Virchow's views were made known, certain observations had already been recorded in this country, which, although they at first attracted little notice, nevertheless involved conceptions as to the real nature of cells that have since proved to be more accurate than his. But the first shock to the system which Virchow had elaborated was given by one of his pupils, Recklinghausen. He showed, in 1863, that pus-cells, instead of being rounded bladders, each with its cell wall and its nucleus, are during life solid masses of gelatinous substance, which are constantly changing their form,—now throwing out delicate processes, and now drawing them in again,—and that they actually possess the power of moving from place to place. Four years

later, in 1867, Cohnheim, who also was a pupil of Virchow, discovered that in the mesentery of the frog, when it becomes inflamed under exposure to the air, the white corpuscles of the blood can, with the aid of the microscope, be distinctly seen to make their way through the walls of the smaller vessels, so as to become pus-cells. This very fact, however, had been demonstrated as far back as 1846 by the late Dr Augustus Waller, then in practice at Kensington. His investigations were made on the tongue of the frog. Still earlier, in 1843, Dr William Addison, of Great Malvern, had insisted on the transformation of the colourless corpuscles of the blood into pus-cells, and described the former not only as "lining the vessels" in the web of the frog when inflamed, but also as "lying among the fibres forming their walls and exterior to their boundary." The resuscitation and general acceptance of views which for so long a time were neglected should make us cautious in rejecting any statements that are based upon direct observation, however much they may clash with our preconceived opinions.

In those countries in which Cohnheim's experiment is not forbidden by law, it is performed in the following manner:—A male frog, which has been rendered quiet by curare, has an incision made into the abdominal cavity, large enough to allow a loop of intestine to be drawn out, with its mesentery. It is then placed upon its back on a broad glass plate, which has been fitted with a thin glass disc for the convenient reception of the mesentery, and with a semicircle of cork to which the intestine is to be pinned. Under the microscope, the circulation can now be seen to go on naturally. But the contact of air appears to act as an irritant, and inflammation soon sets in. In from fifteen to twenty minutes the arteries are observed slowly to dilate, until at the end of an hour or two their diameter is nearly twice as great as before. A little later a similar change begins in the veins. At first the blood-stream perhaps flows more quickly than before, but very soon its rapidity begins to diminish, so that the oval red blood-discs can be distinctly seen in the arteries, at least during diastole. A striking change is now noticeable in the veins. Leucocytes begin to fall out of the middle of the current, and loiter against the sides of the vessel, rolling over and over, and sticking from time to time, when their soft substance yields to the pressure of the fluid upon them so that they become pear-shaped. Before long the channel of the veins becomes lined by a thick layer of these bodies, which lie at rest, while the central current of red discs goes on as before. In the capillaries, too, the leucocytes are arrested here and there; but between them there are seen many red discs also adhering to the walls. The most wonderful thing of all is that outside the contour of these vessels there gradually appear a number of minute rounded bodies, each of which is connected with a leucocyte within. Thus the leucocytes may be said to have assumed a dumb-bell form. By an imperceptible process, the extra-vascular part keeps growing bigger, and the intra-vascular part smaller, until at length the latter altogether disappears; whereupon the leucocyte resumes a more or less globular shape, and now lies close to the vein, but outside it. The result is, that large numbers of leucocytes are soon collected not only in the immediate neighbourhood of the vessels, but in all the interstices of the surrounding tissues. At the same time a quantity of fluid exudes, which reaches the free surface of the mesentery and coagulates there, forming a membranous layer, itself thickly set with leucocytes.*

* The important fact of the white corpuscles adhering to the sides of the vessels in the web of the frog's foot had been observed and figured by Dr C. J. B. Williams, in his 'Prin-

The separation of the leucocytes from the red discs flowing through the mid-channel of the veins, and their collecting against the walls of these vessels, are direct physical results of the slowing of the blood-current. The process of *emigration* must be referred to the power of spontaneous locomotion possessed by the leucocytes themselves. It seems, however, to depend to a certain extent upon their being under pressure from the blood, for Cohnheim has observed that it instantly ceases when the flow of blood through the main artery of the part is arrested. But, on the other hand, it is not due solely to blood-pressure, for this is actually less than under normal conditions where no emigration seems to go on, at least in the case of tissues which are not in an embryonic state.

Professor Julius Arnold and Mr Purves have maintained that the points at which leucocytes emerge from the vessels are always situated in the meeting lines between the endothelial cells. But Cohnheim argues that at any rate actual orifices or stomata can hardly be present, or they would give exit also to an unaltered liquor sanguinis, whereas the fluid which exudes during inflammation is of different chemical composition, and contains less solid matter in solution.

Before the time of Virchow, a favourite topic of speculation had been whether parts into which no vessels penetrate could be said to undergo inflammation. One of the merits of his system was that it placed the question on its right footing by showing that the distinction between vascular and non-vascular tissues was after all only one of degree, and that a minute islet of liver-substance ought to be regarded as being outside the blood-current, no less than the cornea or the cartilage of a joint. Nor did Cohnheim's investigations modify this conclusion, but rather carried it a step further; for he actually proved that leucocytes could penetrate into the interior of the cornea from the blood. It had been shown by Von Recklinghausen that when finely divided vermilion was injected into a lymph-cavity in the frog, the leucocytes would take this substance into their interior, or "feed" upon it; and Cohnheim found that after injecting pigment into one of the aortæ many of the leucocytes which appeared in the cornea under inflammation were coloured, so that they had evidently been derived from the circulatory fluid. Still, however, it could be maintained that others of them were formed by proliferation. This view was, and is still, vigorously upheld by Stricker, who described the fixed corpuscles of the corneal tissue as breaking up into amœboid masses, containing bodies which he believed to be the germs of future pus-cells. Other observers afterwards pointed out that by more refined histological methods the stellate corpuscles could be seen in a cornea which was already full of leucocytes; and although it seems to be a fact that their nuclei do undergo division, this is probably an indication (as Dr Thin has suggested) of commencing disintegration rather than of germination. The observations which Cohnheim, writing in 1877, deemed most worthy of notice, as running counter to his own, were some which had been made by Böttcher. He cauterised the minutest possible spot in the centre of the frog's cornea, and showed that pus-cells were to be found in the neighbour-

ciples of Medicine' (1843). Mr Wharton Jones had detected the same phenomena and accurately described the dilatation of the vessels and stasis of the blood ('Guy's Hospital Reports,' New Series, vol. vii, 1850), and Mr (now Sir Joseph) Lister had confirmed and extended these observations by his own (also carried out on the web of the frog's foot) in the 'Philosophical Transactions' for 1858. One of the important points established by Lister was that the dilatation of the arteries depends on paralysis of the vaso-motor nerves.

hood of the injured part, whereas there was no marginal opacity such as would have indicated that they had come from the conjunctival vessels. Cohnheim's rejoinder was that they might have been derived from the fluid secretion which covers the free surface of the eyeball. This, which at first seemed to me a very far-fetched solution of the difficulty, appears to have been since established by the experiment of placing a dead cornea, in which a puncture has been made, into a healthy conjunctival sac for a few hours; "it remains transparent," says Dr Sanderson in his Lumleian Lectures for 1882, "everywhere excepting in the neighbourhood of the injury," so that a breach of surface is evidently all that is necessary to enable leucocytes from the conjunctival fluid to make their way into corneal tissue. It has also been shown that a limited part of the cornea may be destroyed without any opacity of the adjacent tissue resulting, if the caustic employed be chloride of zinc, which does not disintegrate the protective epithelial covering.

It seems impossible to obtain an adequate conception of inflammation, without taking into account its relation to the physiological process of repair. When a part is at all severely injured, the removal of damaged tissue-elements is an essential preliminary to the work of reconstruction. May not the emigration of leucocytes in inflammation serve this especial purpose of clearing away such portions of an injured structure as are no longer fit to remain? We have seen that leucocytes are capable of taking up granules of vermilion; and it is well known that they also "feed" upon milk globules when placed in their way. May we not assume that they can also remove damaged cells or fibres? This very doctrine was promulgated, forty-five years ago, by the late Prof. Goodsir. In his 'Anatomical and Pathological Observations' (1845) speaking of "a rapidly extending ulcerated surface," he described "a peculiarly endowed cellular layer, which takes up progressively the place of the subjacent textures;" and a little further on he attributes the separation of a dead portion of bone to the "cells of the newly-formed cellular mass, contained in the Haversian canals . . . . taking it up as nourishment, and substituting themselves in its stead."* The same explanation is obviously applicable to the detachment of a slough in the softer tissues, and perhaps even to the formation of an abscess-cavity, and to the thinning of the roof of an abscess which is about to "point." We may also have recourse to it when we find that, in certain parts of a solid organ, a dense infiltration of leucocytes is attended with disappearance of the normal structure, although there is no obvious gap or breach of continuity; this, for example, may often be observed round the Malpighian capsules of the kidney, when it is in a state of inflammation.

Such an hypothesis seems more satisfactory than the view of Cohnheim, who appears to place the inflammatory process on a merely physical basis; ascribing it to a molecular change in the walls of the blood-vessels, wrought directly by the irritant which sets up the inflammation. In support of this view he cites the experiments of Winiwarter, who found that the vessels of an inflamed part allowed a solution of gelatin to transude through them under a lower pressure than under normal conditions. But probably the change in question is only a part of the general softening of all the tissues, which has long been known to constitute one of the phenomena of inflamma-

* See pp. 404, 406 of the second volume of Goodsir's Memoirs, republished by his successor Prof. Turner, of Edinburgh, in 1868. Also Prof. Redfern's papers on ulceration in articular cartilage, 'Edin. Monthly Journal,' Sept., 1851.

tion. An acutely inflamed bone is, as Paget remarked, so soft that a knife will easily penetrate it; the ligaments of an inflamed joint yield, allowing the bones to be displaced and distorted; and we are accustomed, in the dead-house, to regard a diminished resistance of the texture of a lung as one of the best rough tests of it having been the seat of acute pneumonia.

Before we quit the study of inflammation in cold-blooded animals, some further phenomena must be mentioned which belong to its more severe forms. When croton-oil is smeared over the tongue of a frog, the blood in the superficial capillaries passes into a condition known as *stasis*. It consists in complete arrest of the circulation, with coagulation of the blood; and it corresponds with the condition which in larger vessels is called *thrombosis;* in fact, the two are often associated. A still better way of observing severe inflammation is to apply caustic to a circumscribed spot on the tongue of the frog. This produces what may be described as a series of concentric zones. In the middle all the tissues are killed; or, in other words, an *eschar* is formed. Next comes an area of absolute stasis, in which, according to Cohnheim, the vessels alone are destroyed. Further outwards, the blood-stream is almost stagnant, and the capillaries are choked with red discs, which escape from them in large numbers. Beyond this again the emigration of leucocytes is the principal change. Last comes a zone in which the vessels are merely dilated, while the circulation though them is somewhat retarded. However, it is not clear to me that stasis has been proved always to depend upon the occurrence of alterations in the vascular walls, exceeding in intensity those in the other tissues. At any rate, no such conclusion can fairly be drawn from the well-known experiments made by Ryneck, of Gratz, in 1870. He showed that stasis could be set up by irritation in the web of a frog, after defibrinated mammalian blood, or even milk, had been substituted for the natural circulating fluid of the animal; but that, on the other hand, when a solution of chromic acid or sulphate of copper had once been passed through the blood-vessels, even for a few moments, no such effect could be produced. The same is true of a frog in which the blood is replaced by normal salt solution. But these observations, although they disprove the now obsolete doctrine that inflammation is dependent upon a change in the blood, have little bearing on the questions which are now under discussion.

In warm-blooded animals the attempt to watch the inflammatory process directly with the microscope was at first found to be attended with considerable difficulties; but these were gradually overcome. Mr Wharton Jones had observed in the bat's wing that the vessels when inflamed dilated, that the blood-stream slackened, and that the corpuscles crowded together to the sides of the vessel ('Med.-Chir. Trans.,' 1853). Mr (now Sir James) Paget, in his 'Lectures on Surgical Pathology' published in the same year, also used the bat's wing, and observed the very short period of initial contraction, the subsequent dilatation, and other phenomena as afterwards described, excepting the emigration of leucocytes. In 1870 Dr Sanderson and Professor Stricker, of Vienna, contrived an admirable method of studying the circulation in the mesentery of the guinea-pig, which was demonstrated at the meeting of the British Association in that year at Liverpool, and this method was afterwards applied to the observation of inflammation. Lastly, Professsor Thoma published in 'Virchow's Archiv' for 1878 a series of microscopical observations on inflammation in the mesentery or the omentum of dogs, cats, rabbits,

and guinea-pigs. The only difference between his results and those described above seems to be that he could not satisfy himself that the calibre of the arteries became wider. Emigration of corpuscles and exudation of coagulable fluid occur in precisely the same way as in frogs.

The phenomena of inflammation.—We may now proceed to trace out in detail such explanations of the four cardinal symptoms of inflammation as accord best with our present views.

1. *Rubor.*—The redness of inflammation depends partly upon the dilatation of the veins and other blood-vessels of the affected area,* partly upon the accumulation of blood-discs in its capillaries. If the separate vessels are visible, the redness is spoken of as "injection;" if they cannot be seen, it is said to be "diffused." The tint is somewhat purple, which seems to be due not only to the preponderance of dilated veins, but also to an augmented deoxidation of the blood in the capillaries, in consequence of the retardation of the current through them. When stasis occurs, this also causes redness; and at all but the earliest stages hæmorrhages also play an important part in the production of rubor. They often give to it a punctiform character, and they may be further distinguished by not fading beneath the pressure of the finger. On the other hand, the presence of any considerable quantity of exudation tends to conceal the red colour, even where it does not actually compress and empty the vessels: thus, at advanced periods of the morbid process, the affected tissues often become grey or even yellow. When non-vascular structures become inflamed, the redness of course shows itself in the vascular parts around, from which they derive their nutriment; for instance, in the conjunctiva at the margin of the cornea, and in the synovial membrane about articular cartilages. After death, so much of an inflammatory redness disappears as was due to fluid blood in the interior of the vessels.

2. *Tumor.*—This may, to some extent, be due to vascular turgescence, but the chief cause of it is exudation. Lassar, Cohnheim's assistant, showed some years ago that the stream which flows away through the lymphatics of an inflamed part is greatly augmented. For example, he irritated the paw of a dog, sometimes by immersing it for a short time in hot water, and sometimes by rubbing in croton-oil; and he found that the lymphatics in the limb above became greatly dilated, and that when he introduced a cannula into one of them the fluid which escaped through it was increased to several times its normal quantity. Evidently, therefore, the swelling corresponds, not with the whole amount of exudation, but with the excess of it, as compared with what is taken up again. Cohnheim even holds that inflammation may run its course, and be attended with exudation and with the emigration of leucocytes, and yet lead to no swelling, provided that re-absorption goes on at a proportionately rapid rate. He suggests, for instance, that this may be the case with the eruptions of scarlet fever, and other exanthemata, in some of which the subsequent occurrence of desquamation of the cuticle shows that there was more than a mere hyperæmia. In the case of erysipelas we shall find a striking example of the same thing.

In distributing itself through the inflamed structures, exudation follows the lines of least resistance. In connective tissue, and in the stroma of a parenchymatous organ, it fills the natural interstices. Wherever there is a

* According to Prof. Thoma, in mammals the veins only dilate, not the arteries also, as in frogs ('Virchow's Archiv,' 1878).

gap or interspace it accumulates. It covers the free surfaces of serous membranes. Mucous membranes throw it off, to mix with their natural secretions. In the skin, it penetrates through the deeper soft layers of the cuticle, and it often raises the horny layer to form small collections of fluid, which are termed vesicles and pustules.

3. *Dolor.*—The pain of inflammation is usually, and no doubt correctly, attributed to the compression of the sensory nerves by distended vessels and afterwards by exudation. Thus it often has a throbbing character, being momentarily intensified by each pulsation of the heart. It is generally accompanied by "tenderness;" or (in other words) it is increased by pressure from without. Doubtless a general relation can be traced between the amount of pain induced by inflammation in different parts and the abundance of their sensory nerves; but, on the other hand, it is well known that some tissues, such as ligaments and bones, may become exceedingly painful when inflamed, although they are not naturally very sensitive. The determining factor in such cases appears to be the degree of resistance to distension and swelling which is offered by the structure inflamed.

4. *Calor.*—That those parts which are most accessible to observation become hotter than natural when inflamed, is matter of common knowledge; anyone may satisfy himself of it by placing one hand over an acutely swollen joint or a recently formed abscess, and the other upon the corresponding spot on the opposite side of the body. But contradictory statements have been made as to the relation between the temperature of an inflamed part and that of the blood and of the deep internal organs.

In 1860 Mr Simon made a series of observations with a small thermo-electric apparatus, so shaped that it could be thrust like a pin into the soft tissues, or even into the interior of large vessels; and he found not only that the arterial blood flowing towards a severely injured limb in a dog was always less warm than the venous blood flowing away from it, but that the inflamed tissues themselves were warmer than either kind of blood. He did not attempt to measure the amounts of difference of temperature which he believed that he detected; and if (as is possible) they were very small, there may be the less difficulty in supposing that there was some source of error in his experiments. However, it appears to be certain that he was wrong in concluding that inflammation is attended with a special local production of heat. For Jacobson, of Königsberg, who afterwards investigated this question with more accurate apparatus, found ('Virchow's Arch.,' 1870) that the temperature of the skin and of the muscles down to the bone never, even under conditions of the most intense inflammation, reaches that which exists in the rectum, the vagina, or the peritoneal cavity, at the same time. The difference was generally as much as 2° or 3·5° Fahr. As for the relation between the temperature of one inflamed part and that of the opposite side of the body, the difference was always less in proportion as the distance from the surface was greater. Thus, whereas a rabbit's ear when inflamed was warmer than the other ear by 7° or 8° Fahr. the temperature of the deep muscles of a limb under the same circumstances seldom exceeded that of the same structures on the opposite side by more than 1°. It would follow that the internal organs under inflammation continue to be of a temperature approximately the same as that of the blood. Jacobson showed by direct experiment that in pleurisy the affected cavity was either of the same temperature as, or even slightly colder than, the other one. Afterwards,

in December, 1879, M. Peter brought before the Paris Academy of Medicine a series of observations made upon patients affected with chronic peritonitis, and drew from them the conclusion that there was a very considerable local production of heat independently of the general bodily temperature. But what he really showed was nothing more than that the temperature of the abdominal wall may in such cases exceed that of the axilla by as much as 3·5° Fahr. Unfortunately he omitted to place a thermometer in the rectum, and, therefore, it may be fairly contended that in his cases the temperature of the inflamed part may after all have been no higher than that of the blood.

If Jacobson's observations are correct, the real increase of temperature in the skin and other superficial structures when inflamed may yet receive a very simple explanation. For the blood-current is to be regarded as a great warming apparatus, which carries to the tissues all over the body heat that is generated chiefly in the liver and other glands, and in the muscles. If there be no additional local production of heat, the temperature of each part must be the resultant of four varying conditions: (1) the temperature of the arterial blood supplied to it; (2) the proportion between the space occupied by its blood-vessels and that occupied by its extravascular material; (3) the rapidity with which its vessels are traversed by the blood; (4) the greater or less extent to which heat is dissipated from it by conduction, by evaporation, or otherwise.

Of these conditions the most liable to great fluctuations are the second and third. We have already seen that the vessels of an inflamed part are dilated. That the flow of blood through an inflamed part is much augmented has long been suspected. Sir William Lawrence is said to have roughly demonstrated this long ago by bleeding from both arms simultaneously a patient whose hand and forearm on one side were inflamed. But Cohnheim firmly established the same fact by careful experiments. Having set up inflammation in the paw of a dog, he measured the amount of blood which afterwards escaped through a cannula from the principal vein of the limb; and he found that it was sometimes more than twice as much as flowed from a corresponding vein on the opposite side in the same length of time. The only exceptions were, when extensive gangrene set in, and when there was very profuse suppuration. Any such increased velocity in the blood-current of an inflamed part must necessarily raise its temperature.*

The second of the conditions above mentioned explains why sometimes an inflamed part is not apparently hot. When profuse suppuration was going on in a dog's paw there was sometimes no excess of blood flowing through its vessels. No wonder, therefore, that in cases in which the inflammatory process runs its course slowly, one occasionally meets with what is termed "cold abscesses." But it is unlikely that even they can develop from beginning to end without any elevation of temperature.

Varieties of exudation.—Inflammatory exudation presents many differences, according to circumstances which are as yet but imperfectly known.

In some cases, usually when a serous membrane is the seat of inflam-

* No doubt if superficial vessels are involved in the inflammatory process, more heat is at the same time dissipated, and this tends to prevent the surface temperature from approaching that of the internal organs. But a point of considerable importance is that such an increased dissipation of heat actually makes the surface feel hotter to one's hand than it would otherwise feel.

mation, the exudation assumes the form of a solid fibrillated material—"plastic lymph," as it used to be called. This is no doubt the same substance as the fibrin of coagulated blood, and is formed in the same manner. When this, with the leucocytes contained in its meshes, seems, as in a dry pleurisy, to constitute the entire exudation, it must be formed very rapidly, for it appears as a thin uniform layer upon the sloping surface instead of accumulating in the more dependent parts of the serous cavity. But perhaps the whole of the exudation never does solidify; a fluid residue may have been removed by the active absorbing process which we have seen to be at work.

As Cohnheim remarks, we should expect beforehand that inflammatory exudation would always contain fibrinogen, since that substance is one of the constituents of the blood-plasma. Now, since white blood-corpuscles are certainly seldom absent, the question arises why fibrin, instead of being constantly present, is found principally in inflammation of certain structures—the serous and synovial membranes and the alveoli of the lungs.

As a matter of fact, we find great difference in the products as well as in the course of inflammation. The following are the chief varieties:

Catarrhal inflammation.—When mucous membranes are slightly inflamed, as from exposure to cold, they throw off an abundance of more or less altered epithelial cells. In the lungs or in the kidneys such "catarrhal products" are often seen in immense numbers; not only do they accumulate in and fill up the pulmonary alveoli or the renal tubes, but a great many of them are also carried away in the sputum or in the urine. The relation between catarrhal inflammation and inflammation in general raises a question of the greatest importance, especially in reference to tubercle, as we shall see hereafter.

We must at present suppose that the excessive formation of epithelial cells is a result of the greatly augmented supply of pabulum offered to them in the inflammatory exudation. A tissue which in normal circumstances is constantly undergoing renewal may fairly be supposed to be ready to grow still more actively when such a stimulus is supplied to it.

Serous inflammation.—When a joint or serous cavity is affected with inflammation of moderate severity, it pours out a transparent fluid which accumulates in its cavity. There may be a thin layer of fibrin upon the free surfaces of the membrane; or shreds and flocculent masses may be floating in the fluid. Sometimes not a trace of fibrin is to be seen; fibrinogen is present, but for some reason remains uncoagulated. Virchow many years ago pointed out that fluid effusion, after its removal from the pleura by paracentesis, would often throw down a coagulum of fibrin, and that if this were removed, a fresh one might form in the course of the following day, and so on for several days in succession.

Suppuration.—In the more intense inflammations of joints or serous membranes the exudation is purulent, and does not coagulate. Intermediate conditions, however, are frequent, in which the surfaces are coated with fibrin, while the cavity contains liquid pus; and in the pericardium pus unmixed with fibrin (plastic lymph) is seldom, if ever, seen. This, however, is an exception, for in the case of other serous membranes it may happen that every part of the surface retains its natural smoothness and lustre notwithstanding that pus is present in large amount.

The quantity of pus which collects in a large serous cavity, such as the pleura, is often enormous; and since it may be formed very rapidly the

question has sometimes been raised whether the blood could possibly have yielded a sufficient number of leucocytes. But, as Cohnheim remarks, this difficulty in reality applies far less to the emigration theory than to that of pus-formation by proliferation. It has been shown that while inflammation is going on, the blood throughout the body contains an excess of leucocytes; and, indeed, its well-known hyperinotic condition may be said to be nothing more than a rough illustration of the same fact. One can easily suppose that the lymph-glands and the spleen throw into the circulating fluid a largely increased supply of leucocytes to make up for the drain that is going on. At the same time it appears very probable that pus-cells may themselves multiply by fission. Dr. Thin has figured a dumbbell-shaped leucocyte which seemed to be dividing into two, from the inflamed cornea of a rabbit; and similar appearances have been observed in cold-blooded animals by Stricker, Klein, and Ranvier. Possibly, since leucocytes evidently must have nutriment to enable them to multiply, the absence or scarcity of fibrinogen in pus may be accounted for by its having been used up by the multiplying corpuscles.

Croupous exudation.—We have seen that the rarity of lymph upon the surface of mucous membranes is far from being a solitary breach of a general law for inflammatory exudations throughout the body. The exceptional frequency with which a fibrinous layer is found lining the higher air-passages may be due to the same unknown causes which render the alveoli of the lungs also capable of a "croupous" as well as of a "catarrhal" inflammation. In connection with this question certain points have recently been raised which deserve careful consideration. Cohnheim has drawn attention to the fact that in the interstices of the tissues generally, exudation seldom coagulates; and he has shown that this probably depends not upon the absence of fibrin-factors, but rather upon a controlling influence which living structures exert over coagulation in their immediate vicinity. It would follow that whenever we find solid exudation in the connective tissue of any part of the body or within the stroma of an organ, we may be sure that the vitality of the part was endangered, if not lost, before coagulation took place. Now, there is an important class of inflammations in which the exudation of fibrin is thus associated with a local death of the inflamed structures. Wertheim asserted that a destruction of the epithelium of the trachea is a necessary antecedent to the development of a false membrane in the trachea, both in animals under experiment and in the human subject during disease.*

Diphtheritic inflammation.—The processes which combine fibrinous exudation with loss of vitality in the affected tissues are those which morbid anatomists now call "diphtheritic." The term is an unfortunate one, since it suggests an impression that they are connected with the disease Diphtheria, whereas this is by no means generally the case. Still we cannot avoid using it till a better name is found, for the condition is one which demands recognition. It is that in which a free surface, usually a mucous membrane, becomes coated with an adherent layer which in part at least consists of the upper strata of the mucous membrane itself, infiltrated with a fibrinous material. The distinction between a *croupous* and *diphtheritic* false membrane

* So far as I know, the suggestion has not yet been made that the endothelium of a serous cavity must die before a layer of plastic lymph can be formed upon it. Yet surely inflammatory exudation is far less removed from the influence of living structures when it is contained in the pleural or peritoneal cavities than when it is free upon a mucous surface.—C. H. F.

is not merely, as was formerly taught, that the one is easily separable while the other is firmly attached. It is chiefly in the air-passages, where there is a thick basement-membrane, that fibrinous exudations lie loose; upon the fauces and elsewhere they often adhere closely, if their fibrillation is dense and strong. A surface affected with diphtheritic inflammation looks dry, and is of a slate grey or pale yellowish colour unless it has been accidentally stained by a coloured fluid, such as bile. It feels rough and granular, and a still more marked character is its tough elastic hardness. In the dead body, if an incision is made into a diphtheritic membrane, the dry grey appearance is found to penetrate to some depth and to cease somewhat abruptly. Under the microscope is to be seen a granular, more or less distinctly fibrillated substance, containing in its meshes altered epithelial cells, and leucocytes which have lost their nuclei. In the more superficial part of such a false membrane the fibrin is often developed into thick and glistening fibres; in the deeper layers, where it is infiltrating the interstices of a pre-existing tissue, it has no room to show even fibrillation, and it looks granular. As we have already shown, so much of the substance of the part as is involved in the diphtheritic process has already lost its vitality, and the only way in which the disease can end (unless it destroys the life of the patient) is by the separation and shedding of all the dead tissues, with the false membrane of which they form a part. This detachment is effected by *ulceration*. From the first there was an inflammatory action of more or less intensity in the living structures beneath. But now leucocytes collect in large numbers along the boundary line, and probably the connecting material is directly eaten away and absorbed by them. Perhaps the best specimens of the shedding of an extensive diphtheritic membrane are afforded by the urinary bladder. This organ is exceedingly liable to diphtheritic inflammation, which often involves the whole thickness of its mucous coat; and it is no uncommon thing for the *mucosa* to be cast off almost entire. An autopsy was lately made by the writer in the case of a woman from whose bladder during life a piece of considerable size had been removed. All the rest of the vesical mucous membrane lay loose, as a thick ash-grey substance, with markings on one surface corresponding with those of the hypertrophied muscular trabeculæ.

It is obvious that recovery from a diphtheritic inflammation, when possible at all, involves the regeneration of the tissue which has undergone destruction, or at least its replacement by cicatricial tissue. After the separation of a croupous membrane there is nothing to be renewed unless it is the epithelium.

Not only is diphtheritic inflammation characterised by loss of vitality of the affected tissues, but also by their putrefaction; as is shown by their dirty grey colour, and by the fœtid odour which they give off. Indeed, this form of inflammation is but one member of a group, which may properly receive the name of "putrid inflammations," and in many of which large masses of tissue undergo death—*mortification* or *gangrene*.

Gangrenous inflammation.—It is customary to describe several forms of gangrene, and to refer them to different causes; but the more one studies these supposed varieties the more difficult is it to regard the distinction between them as fundamental. Of course, it is true that a part may be directly killed in various ways without the intervention of inflammation; as, for instance, by escharotics or by certain degrees of either heat or cold, according to the length of time during which they are in operation.

But in every case it is probable that a less intense application of the destroying agent would set up a violent inflammation, which itself would end in gangrene. So, again, when the death of a part is due to a failure of its blood supply, no doubt it does not always first undergo inflammation throughout its whole substance. Here, also, it seems impossible to draw a hard and fast line. Surgeons are now familiar with the fact that in many instances senile gangrene of the foot, which used to be attributed merely to obstruction of the arteries, arises from slight injury to a toenail, and begins as an inflammatory process. Even when a limb undergoes mortification as the direct result of embolism of its main artery, there is always at the margin of the gangrenous part, active inflammation, which leads to a further death of the tissues. Indeed, Cohnheim showed that the effects of a temporary ligature applied round the ear of a rabbit depend entirely upon the duration of the constriction. After a certain length of time the blood fails to penetrate into the vessels when the ligature is removed, and the part dies; but in other experiments, which were terminated at earlier periods, the result was only an intense inflammatory swelling of the ear with hæmorrhages into it. Lastly, in those forms of gangrene which seem to be determined by the occurrence of pressure—whether directly, by its lowering the vitality of the tissues, or indirectly, by its cutting off the blood-stream through them—there are always further conditions: a previous enfeeblement of the circulation, and some slight local injury which under normal conditions would have produced no appreciable ill-effects. Thus, bedsores occur chiefly in those cases in which there is also a marked tendency to hypostatic congestion of every dependent part, and the gangrene is preceded by inflammation that can be attributed to such causes as irritation of the skin by excreta, or unavoidable bruising during washing and drying, or friction against folds in the sheets. Thus, if we exclude what may be termed the immediate or instantaneous occurrence of gangrene, all its forms may be said to arise out of an inflammatory process.

We have seen that putrefaction is an essential part of gangrene. But in different cases there are great variations in the rate at which chemical changes in a dead tissue advance, and in the degree of fœtor produced by them. In the "senile" form of gangrene, the affected parts dry up and shrivel into a hard material like that of a mummy; and "dry" gangrene was formerly described as the characteristic effect of obstruction of the arterial blood supply. But it is now known that the desiccation depends upon the escape of fluid from the surface in consequence of detachment of the epidermis. It is only when mortification spreads very slowly indeed through the more massive segments of a limb that they can "mummify." The gangrene which ensues upon embolism of a large artery is more or less moist. But it often happens that deep structures remain soft, while superficial ones dry up into a horny mass, which is then termed an *eschar*. The colour of gangrenous parts is generally greenish or purplish black, but sometimes grey; it is due to chemical changes in pigment which has escaped from the blood-discs and has diffused itself through the tissues. The horrible odours so often emitted are attributed to the formation of sulphuretted hydrogen and volatile fatty acids. Gases frequently collect to such an extent as to render mortified tissues emphysematous, so that they crackle or "crepitate" when they are touched. In other cases they are saturated with a dark, thick, red fluid, which perhaps raises the cuticle into bullæ. That structures which have undergone mortification are com-

pletely devoid of sensibility and of any other vital function, is a matter of course. They also feel cold, since their supply of warmth is cut off with their supply of blood; but it is to be noted that very superficial patches of cutaneous gangrene sometimes receive so much heat by conduction from the subjacent textures, that their temperature scarcely appears lower than that of the skin in their neighbourhood.

Gangrene is often from the first "circumscribed," or limited to a certain part, and even when it spreads from one spot to another, its progress—if indeed the patient's life is saved—is at last always arrested. A "line of demarcation," as it is termed, is then formed. Here active inflammation develops, abundant exudation of leucocytes takes place, and the connection between the living and the dead tissues is gradually eaten away, until the latter become completely detached and are cast off.

Hitherto we have been regarding the different characters of inflammatory exudation as consequences either of special tendencies which we find to be manifested by the several tissues, or of the greater or less intensity of the irritation by which the inflammatory process was set up. There is, however, another side to this question. Many years ago Sir James Paget examined the fluid contained in blisters raised by the application of cantharides to the skin, in thirty patients of St Bartholomew's Hospital. In some cases it formed a firm, elastic, fibrinous coagulum, in others it was purulent. The former condition was observed in persons who were in sound health, the latter in those who were suffering from advanced phthisis or a similar disease. And, with a little practice, he found himself able to form an opinion, from the contents of a blister, as to the degree to which the person was cachectic, and as to the extent to which inflammation in him would tend to be "adhesive" or "suppurative." Again, in those who are suffering from Bright's disease it is well known that the exudation produced by even slight inflammations is apt to be very profuse and watery. Lassar found, by direct experiment, that when hydræmia was produced by the free injection of a saline solution into the veins of a dog, in whose paw inflammation had previously been set up, the lymph which escaped from the lymphatics of the limb became much increased, while the amount of solid matters in it was as greatly lowered. Moreover, patients affected with dropsy from renal disease are especially liable to gangrenous forms of inflammation, and the same thing may be said of those who labour under diabetes.

Events of inflammation.—Up to this point we have been dealing with the advance of the inflammatory process. But, except when it is cut short by the death of the patient, it always sooner or later enters upon a further stage, one of *subsidence* and *repair*.

If the inflammation is but slight, and if its exciting cause does not continue in operation, it may end directly in what is termed *resolution*. The circulation through the vessels of the affected parts then gradually resumes its natural condition; any leucocytes which may have escaped into the tissues pass away through the lymphatic channels; and any solid exudation that may have formed undergoes conversion into a fatty emulsion, which is likewise carried off in the lymph-stream.

On the other hand, if pus-cells have accumulated in large quantities in a serous cavity, or in the sac of an abscess, and are not removed by a natural or artificial opening, they die there and break up into granules. The fluid belonging to them is then absorbed, while the solid matters remain as an

opaque yellowish mass. This process is known as *caseation;* it is one to which we shall frequently have occasion to allude. Such cheesy residues are supposed by many pathologists to be incapable of undergoing further absorption; but this is very doubtful. They often become the seat of deposits of earthy salts, which give to them the consistence of mortar, or may render them as hard as bone. The tissues which surround them also present formative changes; but these may often be regarded as the result of fresh irritation set up by the dead caseating masses, which act as "foreign bodies."

In all other cases, the *formation of new tissues* constitutes a direct and essential part of the subsidence of inflammation. When a slough or an eschar becomes detached, or when an abscess discharges its pus, a hollow space is left, which is filled up by a process of growth. And even where the morbid change does not go beyond the exudation of plastic lymph this is often to some extent developed into a permanent structure. The steps by which this is brought about were studied with remarkable success by Ziegler in 1876. He embedded in the subcutaneous tissues of dogs a series of minute flat chambers, each consisting of a pair of glass plates, with a capillary space between them, cemented together at the corners, but open at the sides. These he removed after an interval, and found not only that leucocytes had penetrated into the space between the plates, but sometimes that tissue elements and even blood-vessels had developed there. The earliest step was the formation of large granular cells with vesicular nuclei. These he termed "epithelioid" cells on account of their appearance. They are generally round, but sometimes oval or irregular in shape. Their size is very variable, but sometimes they are larger than the epithelial cells of the tongue. They evidently arise out of leucocytes, which appear to grow at the expense of other leucocytes. Indeed, some go on developing until they become what are termed *giant-cells*—irregular masses of protoplasm, each containing a large number of nuclei. That these also are formed by the growth of a single cell, which destroys several others, there is no reason to doubt. In support of this view Ziegler lays stress on the fact that they are constantly found surrounded by an empty space. At first, he supposed that all the nuclei of a giant-cell were developed by fission from the nucleus of the leucocyte in which it had its origin; but he has since admitted that the nuclei of the absorbed cells may perhaps persist in it. Another feature of some of Ziegler's preparations is a *reticulum*, forming sharply outlined septa round the individual cells.

The "epithelioid" cells become pear-shaped or fusiform, and give off bundles of delicate fibrils. These fibrils assume parallel directions, and ultimately their extremities unite with those of fibrils derived from other cells. There is, however, one condition which appears to be essential to such a result; namely the development of blood-vessels in the exudation. They seem to arise partly as buds and loops from the sides of already existing capillaries, partly out of rows of cells, placed end to end, so as to open into one another, and form hollow tubes.

When much suppuration occurred round Ziegler's glass chambers, no formative changes were discernible in them. This corresponds with the well-known fact that only fibrinous effusions into a serous cavity are capable of organisation. It is particularly to be observed that in speaking of "plastic lymph" as undergoing development into connective tissue and vessels one means, not the fibrin itself, but the cells that are included in it.

In human pathology it is chiefly by the study of granulations that one has

an opportunity of observing formative changes originate in leucocytes; but Cohnheim speaks of large round or elliptical cells or even cells with many nuclei, as being generally discernible in small numbers in the pus which is discharged from a wound or an abscess of not less than a week's standing.

The connective tissue which is developed out of inflammatory exudation is sometimes of an enormous thickness, as in the pleura. It very slowly reaches its permanent condition. At first it contains no elastic fibres; according to Paget, they may appear as long as twelve months after its first formation. Lymphatic channels in the false membrane were described by Van der Kolk. In the cardiac valves, amorphous calcareous matter may be deposited in large quantity.

Inflammatory adhesions and cicatrices have a strong tendency to contract, and this may entail serious consequences if vital organs are concerned. On the other hand, adhesions sometimes after a long time relax and soften, even if they do not altogether disappear. Paget cites the following observation of Bichat's: a man had made from twelve to fifteen attempts at suicide, at different times, by stabbing himself in the abdomen. In the situation of the more recent wounds it was found that the intestines adhered to the parietes; but the adhesions corresponding with the older ones were reduced to narrow bands, or had even become divided and were hanging free. In cicatrices also a marked "loosening" may at length take place. These facts, and others, like the ultimate subsidence of sclerodermia, deserve to be borne in mind in reference to analogous changes in internal organs, which we are too apt to regard as hopelessly permanent.

The development of *epithelium* during the subsidence of inflammation is a necessary step in the process, not only after ulceration of the skin or of a mucous membrane, but in many other circumstances. That the cuticle which ultimately covers a granulating wound always arises in contact with pre-existing epidermis is well known; Reverdin's practice of skin-grafting is an interesting illustration of it. This fact does not prove that epithelial cells are always developed out of cells of the same kind; it might be explained upon Rindfleisch's hypothesis of "epithelial infection," the existing epithelial cells being supposed to influence the adjacent granulation tissue, so as to lead its cells to grow into epithelium. Most pathologists, however, now teach that it is impossible for leucocytes, which belong to the mesoblast to form epithelial cells, which are constituents of the upper and lower layers of the embryo.

Local varieties of Inflammation.—It will be well to summarise the various forms which inflammation assumes according to the organs which it affects.

1. The skin is very readily liable to inflammation. This may be slight and transient without visible exudation; or the exudation may produce general swelling—*inflammatory œdema;* or the exudation may appear as clear plasma in vesicles, bullæ, or on a raw weeping surface. Or the exudation may become rich in corpuscles and pustules; pus will result, with the formation of scabs when the pus dries up. Or the inflammation may be chronic and lead to fibroid thickening from "hyperplasia" of the deeper parts of the skin. When similar proliferation affects the inflamed epidermis, epithelial scales instead of spindle-cells and fibrous tissues are produced, and the inflammation becomes desquamative. Or the inflammation may be destructive, and cause loss of substance in the form of *ulceration.*

If this is rapidly progressive it is called *phagedæna*. If visible fragments of dead tissue result it is called, not molecular necrosis or ulceration, but *gangrene* or necrosis in mass.

2. The mucous membranes rarely form vesicles or pustules. Serous exudation, with more or less admixture of pus, is combined with mucin and with desquamation of the superficial epithelium. The whole process is called *catarrhal* inflammation, and the exudation mucous or muco-purulent, or purulent. This is the characteristic form of inflammation of mucous membranes, and the term catarrhal is best kept for it, although it has been extended to any watery discharge, such as the exudation of serous inflammation. Under certain conditions some mucous membranes are liable to a plastic or *membranous* form of exudation. This may affect the respiratory surfaces, including the larynx, pharynx, and fauces, nasal fossæ and conjunctiva, and the trachea and bronchi; it scarcely occurs elsewhere, and even here only during childhood or under special circumstances in adult life.

Again, mucous membranes are very liable to *ulceration*, especially the mouth, stomach, colon, rectum, and larynx. Much less frequently they exhibit the peculiar mixture of plastic and gangrenous inflammation, which has been called *diphtheritic* (p. 52), and which is rarely seen except in the fauces and larynx, in the large intestine, and less frequently in the bladder and uterus.

3. The serous membranes exhibit serous inflammation or plastic inflammation, or, more frequently, a mixture of the two. Occasionally, especially in children, the exudation is purulent; but in many cases this is due to infection with septic bacteria. The membranes lining the great pleuro-peritoneal sac—pleura, pericardium, peritoneum, and tunica vaginalis are also liable to chronic plastic inflammation with adhesions and thickening. Other surfaces covered by endothelium and hollowed out of mesoblast also undergo plastic inflammation. These are the lining membrane of the heart and arteries (endocardium and intima), the air-vesicles of the lungs, and the iris; also, under some conditions, the urinary tubules.

4. Connective tissues. Areolar tissue is readily susceptible of suppuration; but this is almost always secondary to pustules or ulceration of the skin, or perforating abscesses from internal cavities, or general pyæmia. It is also liable to acute serous exudation, as in the anasarca of Bright's disease. Fat very rarely inflames—tendons scarcely ever; ligaments only from strains and ruptures, or from synovitis.

5. Cartilage is little liable to inflame, but is capable of ulceration—chronic or acute and infective; it also may undergo interstitial inflammatory changes, which make it opaque and calcareous. The cornea resembles articular cartilage in its inflammations, which are either acute and ulcerative or chronic and interstitial. The fibro-cartilages, both yellow and white, seem almost exempt from inflammation.

6. Bone is frequently attacked by acute suppurative inflammation, which usually leads to death, either molecular ulceration or caries, or else massive sloughing or necrosis. The chronic inflammation of bone is characterised by hardening (osteoporosis), with hypertrophy and obliteration of the cancelli.

7. Muscles are rarely liable to inflammation, and probably never to suppuration. Myocarditis leads to fatty degeneration. Chronic inflammation of the muscles is supposed to be the cause of myalgia, or muscular rheumatism.

8. Peripheral nerves are almost exempt from suppuration. Neuritis, whether parenchymatous or interstitial in origin, ends in thickening of the perineurium and destruction of the myelin.

9. The solid viscera are liable to three chief varieties of inflammation. These are :

a. Infective, suppurative, traumatic or secondary (usually bacterial), acute.

β. Idiopathic, non-suppurative, parenchymatous, softening, acute or subacute, varying with each organ.

γ. Interstitial, fibroid, contracting, hardening, chronic, and insidious.

Under the first head come abscess of the brain (usually from caries or pyæmia), pyæmic pneumonia, abscess of the liver, "surgical" kidney, and pyæmic abscess of the spleen.

Under the second—it is doubtful whether the acute red softening of the brain is inflammatory or hæmorrhagic, and whether acute myelitis is always truly inflammatory ; but some cases of the latter are proved to be so by the meningitis which goes with them. True acute lobar fibrinous pneumonia is the typical instance of this group ; but its inflammatory nature has also been called in question ; at least it is never traumatic, or suppurative, or chronic. Acute yellow atrophy of the liver should probably be classed as a diffuse parenchymatous hepatitis. Glomerular and tubal nephritis, acute, subacute, and chronic, belong to the same group. The spleen does not appear liable to a similar affection, nor the lymph-glands, nor the testes, nor the ovary.

Under the third head falls the grey degeneration or, as it is now called, sclerosis (hardening and shrinking) of the brain and spinal cord, varying in its symptoms according to its locality, but always showing the same histological features. The corresponding process in the lung is chronic interstitial pneumonia, iron-grey induration with contraction, sometimes called cirrhosis. That in the liver is the chronic interstitial hepatitis named cirrhosis by Laennec. That in the kidneys is the chronic intestinal form of Bright's disease. A very similar process is seen in the fibroid testis, which results from chronic syphilitic orchitis, and which, when gummata are also present, closely resembles syphilitic cirrhosis of the liver.

Ætiology.—With regard to the causes of inflammation in general little need be said in the present chapter ; they will be discussed hereafter in detail. It must be pointed out, that the analogy of the pathological processes concerned, compels us to classify as inflammatory a large number of affections in which we cannot directly recognise the operation of any irritant upon the vessels or the tissues. How internal inflammations are caused by exposure to cold we do not know. That cold often excites pleurisy, pneumonia, myelitis, nephritis, and many catarrhal affections, is generally admitted. But probably there is no general principle to which their ætiology can be referred, so that the relation between these several diseases and their cause is different for each one. A point of importance is that there are some organs and tissues which, so far as we know, are not capable of inflammation as the result of cold. This is true of the brain, and, with few exceptions, of the peritoneum. The same remark applies to the pericardium and to the liver, spleen, adrenals, testes, and ovaries. Of the mucous membranes, those of the entire respiratory tract, including the conjunctiva, frontal sinuses, larynx and bronchi, are most prone to inflammation "from cold ;" next the fauces and intestines ; rarely the bladder ; while the mouth, œsophagus, and stomach are exempt.

There can be no doubt, however, that many inflammations depend only in part upon the external cause that excites them ; the larger share of their

ætiology belongs to what may be fairly termed a "predisposing" morbid condition of the tissue itself or of its blood-vessels. It would seem that this has an important bearing upon the way in which some inflammations spread from point to point, and even from tissue to tissue, without decrease in their severity. When an irritant is applied to any part of the body its effects may extend for some little distance beyond, but their intensity rapidly diminishes—unless, indeed, the resulting morbid process belongs to a separate group of affections, those which are "septic" or "infective." But many inflammations not obviously traceable to an external cause diffuse themselves very widely. For instance, we may find pleurisy complicated with pericarditis or peritonitis, pneumonia leading to meningitis or to nephritis, and so on. In one remarkable case, a boy was admitted into Guy's Hospital under Dr Taylor for what at first appeared to be meningitis, but a day or two later the symptoms seemed rather to be those of peritonitis. After death the oldest lesion was found to be a chronic pericarditis, probably of three or four weeks' standing; but in addition there was acute pleurisy, diffused acute peritonitis, and equally universal acute meningitis. In no part of the body was there any trace of tubercles, or even of caseation.

CHRONIC INFLAMMATION AND GRANULOMA.—We have seen how difficult it is to draw a clear line between acute and chronic inflammation on the one hand, and between chronic inflammation and hypertrophy or fibroid degeneration on the other.

There is, however, another form of chronic inflammation which ends not in fibrous thickening or hypertrophy, but in the formation of a new growth of cellular tissue which resembles the granulations of an ulcer. This process appears to be intermediate between inflammation and the production of neoplasm or tumours, and the diseases which come under this head were included by Virchow in his great work on morbid growths. Anatomically they consist of young spheroidal cells (leucocytes), often with "epithelioid" and "giant-cells" among them. There is no fibrous tissue and a rather scanty vascular supply.

The term *infective granuloma*, which is now commonly applied to the pathological group in question, includes Tubercle, Lupus, Syphilis, and the rarer forms of disease known as Glanders and Leprosy. They all agree in their histological characters, and more or less in their clinical course. We now know, moreover, that they owe their peculiarities to the presence of microphytes which act as permanent irritants and keep up a "specific" form of inflammation. This interesting pathological group has therefore relations to inflammation in one aspect, to contagion and microbes, and so to specific fevers, in another, and to tumours and neoplasms in a third.

Its several members will be discussed hereafter. Tuberculosis and syphilis will take their place among specific febrile diseases, inasmuch as apart from the local granuloma there is pyrexia from infection (or as the French pathologists call it, "intoxication") of the whole body by the microbes conveyed in the blood.

Glanders combines local inflammation of a granuloma-type, specific infection, generalisation and fever, and, as a matter of convenience, will be described under the general head of specific fevers along with others of animal origin.

Lupus and leprosy, however widely spread, continue local diseases, and will be noticed among affections of the skin.

PYÆMIA

Putrefaction in wounds may be avoided without excluding the air, by dressing them with some agent capable of destroying the vitality of the atmospheric organisms, provided that it does not act with too great violence upon the human tissues.—LISTER.

Theories of pyæmia—Metastasis—Phlebitis—Septicæmia—Embolism—Infection—Microphytes—Practical application—External or traumatic and internal pyæmia—Channels of infection—Local distribution of secondary abscesses—Idiopathic pyæmia—Slighter forms—Diagnosis: especially from enteric fever and tuberculosis—Prognosis—Treatment.

IT is remarkable that so little attention was formerly paid to the malignant process which often rendered slight wounds and injuries mortal, and which up to the present day was the great danger to be feared after the most skilful surgical operations. The very names of blood-poisoning, septicæmia, and pyæmia are recent, while tetanus, the far rarer sequel of wounds and injuries, was familiar from ancient times.

Probably one reason for this was that the immediate effect of wounds in producing what was called "irritative fever" was confounded with the later appearance of pyæmia, and that this was not distinguished from the still later "hectic" (*i. e.* continued) fever which accompanied prolonged suppuration. Another reason may have been that in the hotter climates with which the physicians of ancient Greece were familiar (and those of Italy in the sixteenth and seventeenth centuries) cases of tetanus are more common than in the north of Europe. Lastly, whatever other miseries followed an ancient or mediæval battle, the wounded were at least free from the dangers which made the crowded wards of a city hospital, until recent times, a hotbed of pyæmia.

Pyæmia (wound-fever, surgical infection) is happily of much diminished practical interest to the modern surgeon; but its pathological interest is as great as ever, and it enters so far into certain forms of internal disease that a treatise on the principles of medicine would be incomplete without some notice of this general morbid process. Its place is naturally after inflammation and infection and before specific contagious diseases.

Theory of pyæmia.—When an unhealthy-looking wound was accompanied by fever, with wasting, sweats, and more or less jaundiced tint of skin, the earlier pathologists supposed that an "ill-habit of body" prevented the wound from healing; and when abscesses were discovered after death in the lungs, liver, and other organs, they were ascribed to *metastasis* or transference of "disease" or "inflammation" from the wound to the internal organs. Even the strongest humoralists seem not to have supposed that the ill-habit of body was due to poisonous humours absorbed from the wound.

The first attempt to ascertain the exact nature of the process of pyæmia was made by Cruveilhier. He believed it to depend upon *suppurative*

phlebitis, which Hunter had long before (1784) distinguished from adhesive phlebitis.* Hence, for many years surgeons dreaded injury or even ligature of veins.

One of the achievements of Virchow was to discover the true meaning of this phlegmonous, suppurative, or infective phlebitis. He showed that what was supposed to be a mass of pus-corpuscles, the product of inflammation in the lining tunic of a vein, is really a discoloured clot composed of leucocytes and fibrin. He showed that fragments of this clot may be detached and carried by the still open veins to the right side of the heart and thence to the lungs; and that in like manner *thrombi* (*coagula*, clots) may be carried from the right side of the heart to the lungs and thence to the systemic capillaries. This remarkable process of *embolism*, by which detached particles of clot are carried from the original *thrombus* by the blood-stream, had been independently discovered by Dr Kirkes, of St Bartholomew's Hospital. It was extended by the great pathologist of Berlin not only to explain the mechanical effects of a plug of fibrin, but also the more dangerous results when the emboli are derived from a septic or putrid thrombus and act not only as mechanical obstructions to the blood-vessel in which they lodged, but also as centres of infection to the surrounding tissues. The mechanical effect of emboli upon the hydraulics of the circulation will come under consideration when embolism of the cerebral arteries is discussed. It is of practical importance in this case, and also when it occurs in the retina, in the kidney, or in the spleen; and it is most important of all when it affects the lesser circulation and obstructs a main branch of the pulmonary artery. When the clot is septic, two pathological processes are combined, the mechanical effects of the embolism and the infective power of the poisonous embolus. This latter condition was distinguished by Virchow as *septicæmia*.

Previously, the effects of pyæmia had been ascribed to actual contamination of the blood by pus. But when pus-cells were ascertained to be only, as Virchow believed, young cells, like white blood-corpuscles, or, as we have seen in the last chapter they are now proved to be, true blood-corpuscles out of place, it was clear that the mere presence of these elements in the blood could no longer explain the process of pyæmia. Moreover, as Virchow had proved, the white cells found in the thrombus were not pus (for they had never left their habitation), but leucocytes aggregated by coagulation and not by inflammation. Accordingly, various terms, such as septicæmia, ichorræmia, and saprræmia (since applied in a special sense) were invented or adopted from earlier pathology to express the element of "blood-poisoning" which complicated that of embolism.

In this as in other departments of pathology, direct experiment upon the lower animals was at once the herald and the guide of speculation,—speculation based on the exact facts of minute anatomy, and controlled by clinical experience. It was found, first, that minute fragments of healthy blood-clot, introduced into the circulation, acted like seeds or any other non-irritant foreign bodies as mechanical obstructions leading to anæmia, to hæmorrhage, and other local results, but not to suppuration or to fever. Secondly, it was found that pure *pus laudabile* when injected into a vein either mingled with the blood, the corpuscles gradually breaking up and no effect following, or if

* The refutation of the hypothesis that suppurative phlebitis spread by continuity from the wound to the heart, was due to Arnott and Dance, who wrote before 1830. See Virchow's 'Gesammelte Abhandlungen,' S. 637.

in large quantity formed an ordinary non-infective thrombus. Thus "pyæmia" was proved not to be what the name signified, pus in the blood. Cruveilhier had before ascertained that globules of mercury when injected acted as irritants and produced inflammation and abscess around the point where they stuck in the capillaries. Thirdly, it was found that when ichorous, putrid, unhealthy pus, carefully filtered so as to get rid of all fragments of solid matter, or even dialysed so as to leave corpuscles and most of the albumen and globulin behind, was injected into a vein, the result was increased temperature, disturbance of the nervous system, diarrhœa and congestion of the lungs; but neither during life nor on dissection after death were "metastatic" abscesses discovered, nor lobular pneumonia nor embolic "wedges."

Thus the result of observation and experiment up to a recent date was that pyæmia or wound-fever was not due to "metastasis" of suppuration from the seat of injury to internal organs; that it was not due to suppurative, still less to ordinary adhesive phlebitis; that it was not due to the entrance of pus into the blood nor to mere embolism, nor to poisoning of the blood with chemical compounds in solution—such alkaline products as have lately been recognised as ptomaines; but that it was made up of two pathological processes, embolism and septicæmia. An infective or septic thrombus gives rise to infective emboli, and these reproduce the parent suppuration, not by a mere deposit of pus, but by acting as an irritant and exciting around them as foci true inflammation which ends in the formation of pus. Every "pyæmic" abscess of the lungs is preceded by lobular pneumonia, every such abscess of the liver or kidneys by corresponding inflammation. The "secondary" abscesses resemble the primary suppuration as the secondary "deposits" of cancer resemble the primary tumour.

But all these observations and experiments were, in the words of Bacon, "light-bearing" rather than "fruit-bearing." Knowledge of the process of pyæmia had come, but power to prevent it lingered. The mortality from pyæmia after operations in well-ventilated, cleanly hospitals was grievous, but in hospitals like the old Hôtel Dieu, of Paris, or the old hospital at Halle, it was terrific. In order to avoid this dreaded scourge, the most elaborate dressings were devised, as complicated, as cumbersome, and as useless as the armour of the fifteenth century; breasts were removed by *cautérisation à flèches*, wedges of chloride of zinc were thrust into the tissues and allowed to eat away the organ, tumours were grubbed out with the fingers, and even limbs were amputated by an *écraseur*. Meantime, thoughtful surgeons noted that pyæmia, so fearful under all the appliances of great cities and the dexterity of the most skilful operators, was far less frequent in country places, in open tents and in cottage hospitals.

At length the explanation came. It was not access of air, nor of oxygen, nor of pus, nor of ptomaines, but it was the invasion of certain lowly-organised plants which made the utmost skill and the most elaborate precautions unavailing, and which had almost led some distinguished surgeons to abjure operations altogether. A brief account of these microphytes was given in the chapter on Contagion. They have been divided by their physiological effects, rather for convenience than as a scientific distinction, into those which are fermentative, those which secrete pigment, those which give rise to specific diseases and those which produce putrefaction. The members of the last group, the septic bacteria and micrococci, are those which cause pyæmia. When they are precluded from gaining access to a wound, no infective thrombi

are formed, no septicæmia occurs, no fever results, and no secondary abscesses are produced.

The recognition and classification of these perilous microscopic organisms was the work of many observers, microscopists and botanists, physiologists and pathologists. The application of this knowledge to the practical end of preventing the mischief they do was due to the scientific training and the philosophical insight, the skilful ingenuity and the untiring efforts towards perfection of methods which enabled our illustrious surgeon, Lister, to effect a revolution in practice.

Pyæmia, then, though like other terms it may be retained when its original significance has been disproved, is a somewhat complex condition. It combines (1) septicæmia or absorption of chemical soluble products, themselves probably the result of the action of septic bacteria; (2) thrombosis and embolism or transference of minute clots from the veins to the lungs and thence to the systemic capillaries; (3) the presence of infective bacteria, surviving their transference in the blood-stream and propagating in the spot to which they are conveyed; (4) local inflammation of a putrid, infective, and virulent character, which leads to (5) necrosis and abscess.

Probably the local determination of a pyæmic focus is often determined by the size of the capillaries of the part; those of the lung are comparatively large, and transmit objects which will block the far more minute vessels of the joints. But beside this, Drs Wilks and Moxon ('Path. Anat.,' p. 626) argue with much force, and from large experience, that local stagnation (as in the back of the lung when a patient is in bed, in the cerebral sinuses, in the large veins of the prostate, and in the recesses of the valves of veins) will cause an accumulation of infective particles, and thus determine the local foci of their earliest and greatest activity.

Internal pyæmia.—In the great majority of cases pyæmia results from an external wound, accidental or operative, and so far it belongs to what is called surgical pathology rather than to internal medicine. But not unfrequently we meet with cases where the origin of the pyæmic process is internal and not traumatic. It is always a breach of surface; but it is not always a mechanical breach in the skin or accessible mucous membranes.

One variety of internal pyæmia, and the most important by its frequency, is when infection takes place from the uterus after delivery. The septic peritonitis and other pyæmic symptoms which follow the formation of septic thrombi in the great uterine sinuses, constitute one form of the dreaded puerperal fever; and here also antiseptic precautions have been introduced. Another form of internal pyæmia is when the origin of the process is in bones, most often in the petrosal bone of the skull; such cases will be described under diseases of the brain. A third depends on suppuration of the urinary tract, but this is most frequently the result of stone in the bladder or of stricture, diseases which admit of mechanical treatment; so that urinary pyæmia, or as it has been called, "erysipelatous," "diphtheritic," or "infective" inflammation of the urethra, bladder and kidney falls for the most part within the arbitrary limits of surgical pathology. Again, when there is suppuration in the alimentary canal (as in dysentery), a local or a general infection of the blood may result.

In most of these cases the primary seat of inflammation is more or less directly in communication with the exterior of the body, and therefore liable to contamination from bacteria on the surface. But in another remarkable

group of cases the original focus of infection is in the interior of the vascular system. These cases of ulcerative, infective, or malignant endocarditis are in the fullest sense internal pyæmia, and here, as was first shown by Professor Heiberg ('Virchow's Archiv,' Bd. lvi), the primary ulcer is characterised by the presence of septic micrococci. This remarkable variety of disease will be described under diseases of the heart in the second volume of this work. How microphytes gain entrance to the interior of the cardiac chambers is most difficult to understand, but not more so than their access to the bones in cases of acute osteo-myelitis.

Mode of infection.—The virulent, putrid or septic, bacterial inflammations which accompany pyæmia appear to spread in three ways:

(1) By continuity: chiefly seen in the case of mucous surfaces, as when a gonorrhœal urethritis extends to the bladder and then along the ureter to the pelvis of the kidney; or when scarlatinal angina passes along the Eustachian tube and infects the middle ear; or when septic metritis travels up the Fallopian tube to the peritoneum.

(2) By lymphatic channels: as when an unhealthy sore on the finger sets up inflammation of the lymphatics of the arm and causes a glandular abscess in the axilla. By a somewhat different route, but probably still by means of the large lymphatic channels which unite the areolæ of connective tissue with each other and with the great serous lymph-sacs of the body, an acute suppurative pleurisy spreads to the pericardium, or infective perimetritis to the peritoneum, or bacterial peritonitis to the pleura.

(3) By veins: in this process alone of the three, septicæmia and conveyance of septic organisms is combined with thrombosis and embolism. It is the commonest and most fatal of all forms of pyæmia. It may infect the veins of the extremities after amputation, the sinuses of the dura mater from caries of the bones of the skull, the portal system of veins from dysentery, and the systemic capillaries from ulcerative endocarditis.

Apart from these forms of local pyæmia, which can be explained by the anatomical relations of the parts involved, certain remarkable facts have been observed which appear to point to the great principle that, when morbid influences affect the whole body in common, each tissue and each organ has its own greater or less proclivity to suffer, and each has, to some extent, an individual power of modifying the general process when it is itself affected. We see this law in operation when the organs and tissues are affected by starvation, by overfeeding, by lead-poisoning, by alcohol, by the diseases which, like poisons, affect not the blood only but blood and bone and flesh —tubercle, syphilis and cancer, lardaceous and fatty degeneration, syphilis, scarlatina, and malaria. Every organ and tissue, every living cell in the body must be acted on by the same exciting agent in each of these diseases, but all react differently.

So with pyæmia. Percival Pott in the last century noticed that abscesses in the liver were particularly apt to follow injuries to the skull. The serous membranes suffer most in puerperal septicæmia. The lungs and liver are more prone to pyæmic suppuration than the brain. The pancreas and mamma and testis almost always escape. Arterial pyæmia affects the spleen and kidneys and brain and liver more often than other organs. Infection starting from a bone is most apt to produce secondary abscesses in the heart and in the kidneys, as Wilks and Moxon discovered.

Idiopathic pyæmia.—Ulcerative endocarditis, portal pyæmia, urinary

pyæmia, and cerebral pyæmia, with the results of infective caries of the vertebræ and pelvis, are the most frequent kinds of internal or, as it may be styled, "medical" pyæmia. Occasionally it results from an empyema or from some internal abscess, from typhlitis, or from sloughing ulceration of the fauces, the rectum, or the skin.

The most puzzling cases to account for are those which we are forced to call Idiopathic Pyæmia. It occasionally happens that on a *post-mortem* examination unmistakable signs of general infective embolism are found, lobular pneumonia with pulmonary abscesses, ecchymoses of the pleura and pericardium, staining of the inner surface of the aorta and multiple abscesses in the liver or kidneys or spleen, or suppuration in the joints; and yet it is impossible to discover the source of the infection. There is no external injury or wound, the urethra is found healthy, there are no anal fissures or inflamed piles, no chronic ulcer of the fauces or pharynx or nasal fossæ, no necrosis of the long bones, or caries of the vertebræ or ilium or internal ear. In one case, the late Dr Moxon, after laboriously searching these and other parts, extracted every tooth from its socket to make sure that there was no alveolar abscess or caries; yet all was in vain. Sometimes we are rewarded by discovering the source of the mischief in unexpected situations,—a pin or other foreign body in the appendix cæci, caries of the ethmoid bone or of the sacro-iliac joint, pyosalpinx as the result of gonorrhœal inflammation of the vagina. But in a few cases we are compelled at the end of the search to confess our failure and to call the case one of idiopathic or spontaneous pyæmia.

Whatever doubts may still be felt as to the possible origin *de novo* of infective diseases, such as enteric fever, scarcely any pathologist will admit that bacteria appear in the blood spontaneously. They must, either as minute micrococci, or possibly as still more minute bacterial spores, gain an entrance by some undiscovered breach of surface.

Slighter forms of pyæmia.—It is probable that under this head should be classed certain cases, common in surgical practice, and not unknown in the medical wards of a hospital, where a patient, suffering from some internal suppuration which has been relieved either naturally or by incision, is from time to time attacked by more or less marked rigors, rise of temperature, quick pulse, loss of appetite and sometimes profuse sweats. We can usually trace this to the flow of pus being interrupted, and when free exit is restored the symptoms disappear without further inconvenience.

Such accidents we see in the course of an empyema which has been opened, of purulent pyelitis, of suppurating hydatid of the liver, and of otorrhœa from caries of the petrosal bone. In many instances an abscess has been opened under strict antiseptic precautions, and there is no reason to suppose the entrance of impure air or secretions afterwards. The symptoms referred to are certainly connected with retention of pus and disappear when free discharge is established. They are quite different from the mere effects of pain when healthy secretions are retained, or when an over-full cyst or an inflamed testis produce general disturbance by pressure which disappears when the tension is relieved. Hence, these symptoms cannot be referred to the mechanical effects of pressure upon nerves. They seem more likely to be caused by the increased pressure leading on hydrostatic principles to entrance of chemical products of inflammation or possibly of septic organisms themselves into the adjacent lymph-spaces or blood-vessels.

Diagnosis.—The symptoms of pyæmia as it occurs after a surgical operation are happily less familiar to the present generation of medical students than before the introduction of antiseptic surgery. Still, however, they occasionally occur and are well recognised. The difficulty arises when no wound or injury is found to account for the symptoms.

These cases of internal or non-traumatic pyæmia present themselves to the physician under the form of fever, and pyrexia is the only constant symptom. It may be accompanied by local pains, by tenderness, or by œdema, which are of the utmost value in leading to the source of mischief. But these are often absent. Headache, delirium or stupor, muscular twitchings, diarrhœa, profuse sweats, a jaundiced tint, are none of them constant symptoms, and all may be present in cases of typhus, of enterica, or tuberculosis. Pain and swelling of the joints may also be present and simulate rheumatic fever. In cases of ulcerative endocarditis the cardiac murmurs with pyrexia sometimes make this last resemblance extremely close, and pericarditis and pleurisy may rather increase than diminish the difficulty of diagnosis, while the fact of previous attacks of rheumatism is far from making the more serious alternative unlikely. This difficulty will be discussed in the chapter on rheumatism in the second volume of this work. The age of the patient, the persistence of local synovitis, the presence of jaundice, and signs of embolism in the brain, spleen, kidneys, or retina, may more or less conclusively decide the diagnosis, but in some cases it is impossible to decide until time has developed the course of the disease. Moreover, rheumatism occurring repeatedly with concomitant lesions of the valves may at last appear in combination with a new and infective form of endocarditis.

The most characteristic single symptom of pyæmia is pyrexia, which is of a remittent type, rarely falling below normal, but rising suddenly and irregularly, to fall again with equal abruptness. It has none of the regular daily variations and steady general rise, culmination and subsidence which we observe in typhus, enterica, and the exanthems. It has not the regular evening rise of hectic fever, which in many respects it resembles, nor the frequent subnormal temperatures, alternating with irregular elevation, characteristic of some forms of acute tuberculosis. Hyperpyrexia is more common than in enteric fever or in phthisis. The resemblance to a regular form of remittent malarial fever is sometimes remarkably close and deceptive.

When a patient is found suffering from fever not due to external inflammation, we first ascertain the absence of internal acute disease, and especially of pneumonia and of phthisis. We may then have to await the appearance of a characteristic exanthem, as the rash of scarlatina, the mulberry eruption of typhus, or the erythematous blush of erysipelas—which is often delayed for a day or two after the temperature has risen. If the first week of fever has passed without these appearances, and if there are no physical signs of pneumonia—which sometimes only appear after two or three days of pyrexia—we then reconsider the possibility of enteric fever and of acute tuberculosis, and lastly the question of internal pyæmia.

Enterica usually begins insidiously, the temperature rises gradually, and the state of the tongue and of the bowels make diagnosis easy even before the rose rash appears. Occasionally it begins more abruptly, the temperature rises high in the first week, the rash is absent or has passed unnoticed before the patient was seen, the bowels are constipated, and diagnosis may then become extremely difficult.

Acute tuberculosis can usually be traced to previous tubercular disease of the lungs or lymph-glands or abdomen of which the signs remain in evidence, or we may find a caseous testis or symptoms of "strumous" pyelitis. Or we may have evidence of meningitis, or we may discover one or more tubercles in the choroid. The lungs are in these cases almost always the seat of disseminated tubercles, and this may be recognised, not so much by physical signs, which are often limited to a slight occasional rhonchus, but by the high ratio of respirations to pulse and temperature, by slight duskiness or marked cyanosis, by the alæ nasi working in aid of the chest, and by dyspnœa unaccounted for by examination of the chest.

When combined with tubercular enteritis in a child it may be almost impossible to distinguish acute tuberculosis from enteric fever, until the subsequent course of the disease makes its clear.

When we suspect pyæmia, our first investigation is directed to the ear for a purulent discharge, to any of the bones or joints which may be tender or œdematous, to the genito-urinary organs, and to the heart. Symptoms of pyæmia, with a cardiac murmur, point almost certainly to ulcerative endocarditis; this is the more probable if previous valvular disease has existed, and particularly if the febrile symptoms have come on during convalescence from pneumonia, as was stated by Dr Osler in his lectures before the College of Physicians, and the signs of embolism in other organs may confirm this diagnosis. In the rare case of this disease affecting the right side of the heart, of which we lately had an example in Philip Ward, the lungs are the first organs to be affected, and the diagnosis is comparatively easy. When infective emboli lodge in the spleen it becomes enlarged, partly by the formation of fibrinous wedges (*infarcta*), partly by the febrile intumescence of the whole organ.

The splenic dulness being increased and the viscus being felt below the ribs on inspiration, does not in itself distinguish the case of pyæmia from one of enteric fever or of ague, but the "typhoid spleen" and the "ague-cake" are not usually sensitive, while the pyæmic spleen is tender to the touch, probably by reason of acute local peritonitis. Hæmaturia, hemiplegia, and, above all, the presence of icterus would confirm the diagnosis.

When otorrhœa, especially fetid otorrhœa, with a perforated tympanic membrane, is present, we may often discover tenderness or œdema over the mastoid process or down the neck. The infection travels by the internal jugular vein to the heart, and then affects the lungs, setting up suppurative lobular pneumonia and acute pleurisy.

On the whole, it may be said that in most cases internal pyæmia has an origin which with care and patience can be discovered during life; that enterica is the most variable of all specific fevers in its course is most frequently unrecognised; and that in doubtful cases we may consider that "common things most commonly occur," or, to state a converse truism, that we rarely meet with rare diseases.

The following recent example of idiopathic pyæmia shows the difficulty of diagnosis, or at least the fact that a wrong diagnosis may be made.

A girl, twenty years of age, who had been confined four months earlier, was admitted into Mary Ward on December 6th, 1886. She was in high fever, with more or less constant delirium and severe headache, and photophobic. She had, we were told, complained only that morning, had gone out without eating her breakfast, and was found moaning and almost insensible in a corner of the room where she had gone to sell bottles to the

hospital patients. The pupils were contracted, there was broncho-pneumonia on both sides, and the temperature rose high, notwithstanding cold affusion and ice-bags, reaching 105·8°. She had, we afterwards learned, complained of headache three or four days before the attack began, but otherwise appeared to be in her usual health. Her mother and a brother had died of consumption. She was in a very dirty condition and her scalp was covered with scabs from *impetigo pedicularis*. When the temperature was 104·2°, the respirations were 44, and the pulse 120. Next day she was duller and lay curled up in bed. There was a faint apex-systolic murmur. On the third day a pleuritic rub was heard and the respirations rose to 54, with temperature 104·8°, and pulse 140. The next day the fever rose to its highest point and was once more reduced by ice; albumen appeared in the urine, and she died after three and a half days' illness. I supposed that she was suffering from acute tubercular meningitis with similar disease of the lungs. The brain, however, was found normal. There was recent plastic pleurisy and a vomica in the apex of the right lung, but no miliary tubercles in the lungs. There was no ulcerative endocarditis, but a little recent lymph on the mitral curtains. One small abscess was found on the surface of the liver. The spleen was swollen but contained no embolic foci. Both kidneys contained numerous small abscesses as in ascending pyelitis, but the pelves were normal, as were the ureter and bladder. The vagina, uterus, and Fallopian tubes were healthy; one ovary contained a serous cyst. The skull and internal ear, the sinuses, the vertebræ and sternum, and all the large joints were searched and all the internal organs, but nothing further was found except one small ulcer in the duodenum. The portal vein was perfectly normal.

Prognosis.—As a rule cases of internal pyæmia are hopeless, and the exceptions are very few. But there are cases, to which Sir James Paget has particularly drawn attention, of chronic pyæmia where the fever is never high, internal organs are spared, and the joints are successively attacked by suppuration. These cases sometimes recover, and it appears certain that some forms of bacterial infection can be dealt with successfully by the living leucocytes and tissues when the number of microphytes admitted is not too large. So that we may say that, even with our present means of treatment, pyæmia is not an absolutely fatal disease.

Treatment.—The indications are first to ascertain, if possible, the source of infection, and give free exit to the septic materials. When an empyema has been laid freely open and drained, a mastoid bone trephined and the pus given exit, a sequestrum of bone cut down upon, or a joint freely incised, infection may not only be avoided, but even a pyæmic process already begun may be prevented from becoming general. A second indication is the free exhibition of stimulants, quinine, and as much concentrated food as can be taken. Quinine in large doses is believed by many surgeons to have a "specific" as well as a "tonic" effect in pyæmia. It is possible that it may check the multiplication of microphytes, and we may one day find in perchloride of mercury, or some more harmless and energetic parasiticide, a more efficient means of dealing with even internal pyæmia than we at present possess.

TUMOURS

Καρκίνωμα ἐστὶν ὄγκος κακοήθης καὶ περίσκληρος, ἀνέλκωτος ἢ ἡλκώμενος· εἴρηται δὲ ἀπὸ τοῦ ζώου καρκίνωμα.—GALEN.

New growths—Innocent and malignant tumours—Virchow's characters of malignancy—Malignancy not an attribute of structure—Histological classification of tumours—Malignant osteoid tumour—Sarcoma: round, spindle, and giant cells; hæmorrhagic and alveolar sarcoma: melanotic sarcoma—Multiple sarcomata as a general disorder—Osteitis deformans—Lymphoma—Carcinoma—Heredity—Theory of new growths.

THE term Tumour was once applied in modern languages (as *tumor* in Latin) to all swellings which were not obviously inflammatory, so that it included hydroceles, retention-cysts of all kinds, hydatids, hypertrophies of the spleen, enlargements of the liver, and even the swelling of a dropsical limb. One of the first lessons in morbid anatomy was that circumscribed solid masses, like external wens, are to be met with in the internal organs as well as on the surface. From a pathological point of view these solid outgrowths were distinguished as *pseudoplasms*, *neoplasms*, or *new growths*. This conception of an independent centre of growth, as distinct from mere hypertrophies of existing tissue on the one hand and from inflammatory swelling on the other, still determines the character of a tumour in the modern sense.

When a tumour grows on mucous membrane, it often projects from the surface, and may hang by a distinct pedicle; it is then (by a curious perversion of an ancient medical term) called a *polypus*. When it forms a rounded mass in a solid organ it is called a *nodule* or a *tuber*.

When the life-history of the cystic entozoa was unknown, nothing was more natural than that tumours should be imagined to be parasitic like galls on an oak. This notion, however, has proved without foundation. Their life is that of the individual in whom they develop, and at present there is no evidence of their depending on microzoa or microphytes.

Benign and malignant growths.—It was early noticed that whereas some tumours remain for years without affecting the health or causing any inconvenience, others rapidly destroy the patient's life. The distinction between "*innocent*" and "*malignant*" growths is therefore a most natural and obvious one. Further observation brought to light the fact that whereas many of the former kind are like the natural tissues of the body—fat, or cartilage, or bone—many of the latter kind are unlike any healthy tissue. The microscope confirmed this distinction by showing that some soft, white malignant tumours, which from their resemblance to the brain or to marrow had been called *encephaloid* and *medullary*, have an intimate structure which bears no resemblance to normal tissues. Thus a fundamental division was made between *homologous* growths, generally innocent, and *heterologous* growths, generally malignant. It was further supposed that, although there were several varieties of malignant growths, differing in character and appearance, yet that all these belonged to a single disease, which was termed Cancer.

The *word* "cancer," or its equivalent *carcinoma*, was of much earlier origin.* Hitherto its tendency to ulcerate and to eat away the natural structures had been regarded as its fundamental character rather than its anatomical structure. Thus, not only had the venereal "chancre" been confounded with it, but even the "cancrum oris" of children, which we now term noma. Henceforth these were finally separated.

Lebert and the early pathological histologists saw no difficulty in supposing that the tissue of a heterologous growth might differ completely from all the normal tissues of the human body. They made it their aim to discover some particular specific element or "cancer-cell," the presence of which might be an infallible criterion, and solve the frequent difficulties which arose in the anatomical diagnosis of tumours.

In 1847, and the years which followed, the genius of Virchow placed this question in an altogether new light. It had already been shown by Johannes Müller that the structure of every growth, however heterologous in appearance, always corresponded in its ultimate elements with some natural tissue; if not with any of the permanent tissues, at least with some one of those which exist during early embryonic life. Virchow carried the same idea into full detail. As he pointed out, the physiological type of the tailed and irregular cells which had been supposed to characterise cancer is to be found in the epithelium of certain mucous membranes; while the round- or spindle-shaped cells which really make up the substance of many malignant tumours correspond with those which are seen during the development of connective tissue, or (as Billroth has since taught) of muscle or of nerve-tissue. In general, he insisted on the principle that the structure of a tumour in man is always human; so that, for instance, such a tumour might contain hairs but never feathers; whereas, in a bird a tumour might have feathers in it but not hairs. For Virchow, therefore, no growth was *heterologous* in the sense hitherto attached to that term. The distinction, as he drew it, was between tumours resembling in structure the tissues in which they were placed, and those differing from these, though resembling others. Thus, he said that the same growth, which in one situation would be homologous, would in another situation be heterologous. And he further pointed out the necessity of recognising that between certain tissues of the body there naturally exist close relations which are altogether wanting

* Cancer is merely the Latin translation of *καρκίνος*, a crab. This word was applied by the Greek physicians to any eating, gnawing sore. Both *καρκίνος* and *καρκίνωμα* occur in this sense in Hippocrates ('Aph.,' 1257, *et passim*) as well as in later writers. Celsus speaks of diseases, "cum quid intra se ipsum corruptum est, ut in *Cancro*," in close proximity to those which result "cum quid extrinsecus læsit ut in vulneribus" (lib. v, cap. xxvi, 1). A little further on (ibid., xxvi, 31) he says that a cancer ensues from too great inflammation, or too great heat or cold, or too tight binding of a sore, or because the patient is old or of an ill habit of body. "Omnis autem cancer non solum id corrumpit quod occupavit, sed etiam serpit." He goes on to say that the Greek physicians divided cancers into species for which there were no Latin equivalents: as erysipelas, gangrene, &c. He describes (ibid., xxxviii, 2) *καρκίνωμα* as chiefly affecting the upper parts of the body, the nose, ears, lips, and the breasts of women.

Galen admits, however, of *καρκίνος χωρὶς ἑλκώσεως* ('De tumoribus præter naturam,' c. 12), and Paulus Ægineta says, "Cancer tumor est inequalis—interdum sine ulceratione, quem Hippocrates latentem (*κρυπτὸν*) nominavit." This author derived the name from the distended veins of a cancerous breast resembling the legs of a crab (lib. vi, cap. 45), and so Galen; others from the difficulty of getting rid of it ('Expos. vocum medicinalium,' 1564).

Our English word *canker* is an older form of cancer and more generally applied. It is used in the Authorised Version of the New Testament to translate the Greek *γαγγραίνα* (2 Tim. ii, 17).

between them and other tissues, and that such relations have important bearings upon pathology. Thus, all the structures belonging to the connective-tissue series being physiologically related to one another, the presence of bone, or of cartilage, or of fat in fibrous tissue, would not be inconsistent with homology, whereas that of epithelium or of muscle would be.

A still greater advance was made by Virchow in regard to the mode of development of tumours. It had before been generally held that cancer was something deposited from the blood. Chemists had made analyses in the hope of finding in it some peculiar principle for which the name "carcinomatin" had been invented in advance. Its cause was believed to be a "dyscrasia" or ill-mixture of the blood. The fact that active tubercle and cancer are but seldom found in the bodies of the same individuals was thought to support this notion, it being supposed, first, that tubercle is deposited from the blood, and secondly, that the blood could hardly present two different dyscrasiæ at the same time. Yet wounds and injuries undergo repair in exactly the same way in those who are affected with cancer as in other persons: which fact alone should have led to doubt of the validity of this hypothesis.

Virchow, in accordance with his dogma, *omnis cellula e cellulâ*, maintained that the substance of a cancer was developed *in situ* by the growth of the cells and other structures of which it was composed. He was mistaken in the view that they arose by a proliferation of the connective-tissue corpuscles. But this was an insignificant detail in comparison with his rejection of the notion that cancerous and other tumours were "deposits from the blood."

One necessary consequence of Virchow's conception of Cancer was that the so-called cancerous cachexia, instead of preceding the formation of malignant growths within the body, must be a result of their actual presence; and this is quite in accordance with clinical experience. No doubt one of the earliest symptoms of an internal tumour is sometimes the fact that the patient becomes cachectic, that his skin acquires a waxy yellow colour, and that his features look pinched and sharp. But in such cases the tumour really exists long before these indications have appeared. It wears down its victim by pain and distress of mind even if it does not attain a large size or undergo ulceration and hæmorrhage. Lastly, it is certain that some innocent growths, when they cause severe pain and much bleeding, are accompanied by well-marked cachexia; for instance, certain uterine polypi, and even hæmorrhoids and non-malignant tumours of the rectum.

Again, Virchow defined the term *malignancy* far more clearly than previous writers. Hitherto the pain caused by a cancer, and its tendency to destroy life, had been enumerated among the signs that it was malignant. He set these points altogether aside, and insisted that the following four characters are the real indications of malignancy.

1. The *local progress* of a growth, its tendency to increase by extending into the tissues around it. A point on which much stress has since been laid, is the fact that malignant tumours show little or no respect to the natural boundary lines marking off one kind of tissue from another. Thus in a case of cancer of the bladder, the tumour having doubtless started in the mucous membrane, ate its way through the whole substance of the organ, through both layers of the peritoneum, through the muscles, aponeuroses, and other structures of the abdominal wall, until it formed an immense ulcerated cavity, reaching from the pubes to the umbilicus.

There is, however, one exception to this power of overcoming the resist-

ance offered by the different tissues, in the fact that malignant growths seldom penetrate the walls of the larger arteries, whereas they frequently extend into the interior of veins, even into the venæ cavæ. Mr de Morgan remarked that cicatricial structures also sometimes form a barrier to the spread of a cancer, so that when recurrence takes place after an operation the disease creeps along one side of the scar for a considerable distance, without passing over to the other side.

It is a point of some interest that cancers are capable of passing across a serous cavity without previous union between the two surfaces. Of this we had a remarkable instance some years ago. The fundus of the uterus being occupied by a malignant growth, the great omentum hung down into the pelvis, so as to lie in contact with it: these parts were not in the slightest degree adherent to one another, but the lower edge of the omentum contained a cancerous mass. It is not improbable that infection in such cases is the result of active amœboid movements on the part of the cells of the primary tumour; Waldeyer ('Virch. Arch.,' lv) has at any rate detected slow changes of form in cells from a cancer of the breast, and in those of a round-cell sarcoma of the axilla, when placed on a warm stage; but he did not see them move from one spot to another.

2. Its *tendency to return in loco* after having been extirpated by the surgeon. This is one of the facts which used to be urged in favour of the constitutional origin of cancer. According to the modern view, it depends upon the circumstance that a malignant tumour always has outgrowths far beyond the area in which the tissues can be seen by the naked eye to be affected. It is true that surgeons who adopt the new theory are very seldom able to prevent the return of the disease by increasing the size of the mass which they excise; but this, there is little doubt, depends upon the apparently healthy surrounding tissues being more or less widely infiltrated with separate malignant cells. A point which is worthy of notice is that in the case of double organs, like the breasts, the removal of the whole of one gland for a malignant growth is not followed by recurrence in the organ on the opposite side, as might certainly have been expected if the constitutional view were correct. Should such an instance occur, one would probably be right in regarding it as an example of the independent development of a primary cancer in each breast; for this sometimes, though rarely, happens when no operation has been performed.

3. Its *spreading to lymph-glands*. As a rule, the glands first attacked are those which immediately receive the lymphatic vessels from the diseased part. Occasionally it seems to pass over certain peripheral glands, and to begin by affecting others, which lie nearer to the thoracic duct. When remote glands become seats of the growth, the infection has probably been carried, not by lymphatics, but by blood-vessels. As a rule, lymph-spaces round the margins of a tumour take up from it something which is carried into the lymph-stream and deposited in the next gland. There is no difficulty in supposing that this something consists of nuclei, or even of cells from the growth; for Virchow has shown that sometimes an entire lymphatic network—beneath the pulmonary pleura, for example, or the peritoneum—may become filled with such materials, as if it had been artificially injected ('Kr. Geschw.,' p. 52, fig. 4). Whether lymphatic channels exist in the interior of tumours is said by Cohnheim to be still open to question, although Van der Kolk long ago, as he believed, discovered them.

4. Its *becoming generalised*, i. e. *spreading to distant organs and tissues by infection, or, as was formerly said, by "metastasis."*

It is not the mere multiplicity of tumours which indicates that they are malignant. Sebaceous cysts and fatty tumours are sometimes present in great number and yet are perfectly innocent. Perhaps there is no form of malignant disease in which so many nodules are found as in some cases of molluscum fibrosum. But all such innocent multiple tumours have their seat in some particular tissue, and remain limited to that. On the other hand, cancerous growths may appear in almost every tissue of the body at the same time.

Again, in most cases of death from cancer it is easy to recognise some one tumour as the starting-point of all the others. A skilled pathologist recognises this "primary growth," either by its anatomical character or from what he knows of the origin and distribution of the particular kind of tumour. The anatomical difference consists not so much in its greater size as in its being of firmer consistence, from slower growth; or an advanced state of degeneration, or extensive ulceration, may show its earlier date. Locality is another important criterion; for primary cancers, like almost all other diseases, do not arise at random through the body, but are common in certain regions, rare in others, and almost unheard of in most. As a matter of fact it is exceedingly rare to find in the same case two malignant tumours, with equal claims to priority.

Another curious circumstance is that organs and tissues which are especially apt to be the seats of primary growths are seldom affected with secondary or "metastatic" growths of the same kind. For instance, a primary cancer is often developed in one of the breasts; but when secondary growths appear they avoid the opposite breast, nor are they met with in the cervix uteri, another locality from which cancer frequently starts. The most common seats of primary cancer are the lower lip, the tongue and pharynx, the œsophagus, stomach, colon and rectum; the mamma, uterus, penis, and testis; and in these regions secondary cancers are almost unknown. On the other hand, the lymph-glands, liver, lungs, bones, and serous membranes, so frequently attacked by secondary cancer, are very rarely the starting-points of the disease. Nor is this a matter of structure, for the whole epithelial surface of the small intestine is all but exempt from cancer; and some glands, as the parotid, are equally so. With sarcomata no such rule holds.*

In many cases the distribution of secondary nodules or tubera is so limited as clearly to indicate by what channel they were derived from the primary growth. For example, a malignant tumour in the stomach or in the intestine may give rise to secondary nodules in no other organ except the liver, showing that something was carried by the blood of the portal vein which

* An old woman died in Guy's Hospital with cerebral symptoms, and I discovered three tumours in the brain or in its membranes. From their appearance I felt convinced that they must be secondary to a primary growth elsewhere. But I searched every part of the body in vain, until at length I happened to notice that the extreme lower end of the rectum had not been taken out with the rest of the intestine. This, when removed, was found to contain a large ulcer with prominent fungating edges, which evidently was the lesion for which I was looking, although it had given rise to no symptoms during life; even the administration of enemata had failed to lead to its discovery. A microscopic examination of the secondary cerebral tumours afterwards showed that these were "columnar epitheliomata." Now, if I had been able to examine them previously, my task would have been very much more easy, for I should have known almost exactly where to look for the primary affection which gave rise to them.—C. H. F.

served to propagate the disease; or the primary growth being in one of the limbs, the secondary growths may occur only in the lungs, so that the infection obviously took place through the systemic veins and the pulmonary artery. Malignant tumours often penetrate into the interior of veins; there is, therefore, no difficulty in understanding how nuclei, or cells, or even pieces of the growth, may get washed away by the blood-current, and carried to distant parts. Cancerous thrombi form soft colourless masses, parts of which are blended inseparably with the coats of the vessel, while other parts, often much elongated and lobulated, project into its channel, either perfectly smooth on the surface, or coated with a layer of fibrin.* Dr Moxon mentions in the 'Guy's Hospital Reports,' vol. xviii (3rd series), that he had twice seen cancer growing in clots which lay within the cavity of the right ventricle at its apex, brought, no doubt, by the blood of the systemic veins. In 1871 Dr Payne showed to the Pathological Society a heart in which both the right auricular appendix and the apex of the left ventricle contained similar cancerous thrombi projecting from between the muscular trabeculæ. In that instance the infecting agent must have traversed the pulmonary capillaries from one side of the heart to the other.

The distribution of secondary growths is often different from what would have been expected on anatomical grounds. Thus, in a case of epithelioma of the œsophagus, the lungs were found healthy, but a secondary nodule existed in one kidney. Frequently the metastatic formation of cancer is limited to some particular tissue throughout the body, without any obvious relation to the seat of the primary growth. Thus, in a woman with cancer of the breast, almost every bone may become cancerous, while all viscera remain entirely free from the disease.

5. To complete the description of the ways in which malignant growths multiply in the body, a fifth point should perhaps be added to those enumerated by Virchow, namely, the possibility of portions of a tumour becoming *transplanted* when set free upon a mucous surface by ulceration or otherwise. In 1868 Dr Moxon showed to the Pathological Society a specimen in which he thought that an epithelioma of the œsophagus extending into the trachea had in this way infected the lungs: secondary nodules were seated in the interior of the lower lobes, and not beneath the pleura, as is usually the case; they occupied the centres of lobules, and small tubes could be traced up to them. He there mentions that Mr Simon had exhibited specimens in which "cancer germs" had appeared to take root in the bladder after descending the ureter from the kidney. In a case observed by Dr Bristowe, a malignant growth of the interior of the skull had apparently infected the cord low down by fragments which had fallen: and in the abdomen we sometimes find a nodule just opposite to the primary tumour and in contact with it. Nevertheless, all supposed facts of this kind should be cautiously weighed before they are accepted; for there can be no doubt that the particles detached from the face of a malignant growth are, as a rule, dead and already in a state of decay. Cohnheim denies that any authentic case has been recorded of cancer of the uterus in a woman producing the same disease in the penis of her husband. No experimental proof has yet been offered of the possibility of inoculating any kind of tumour from one animal to another. There has sometimes been

* Some time ago I saw an instance in which, the primary growth being in the liver, and the hepatic veins extensively involved, the lower border of one lung contained a whitish-yellow wedge-shaped mass, exactly like an infarctus due to embolism from ordinary thrombosis: Dr Pye-Smith, however, under whose charge the case had been during life, found that it was really a secondary nodule of the tumour.—C. H. F.

a slight apparent commencement of growth at the spot inoculated, but it has always before long undergone reabsorption. Not only has the microscope, aided by modern methods of illumination and staining, entirely failed to discover "germs" or "microbes" in cancers or other infective growths, but the same negative result has been reached by numerous attempted "cultivations," of which the most recent and laborious is that carried out by Messrs Ballance and Shattock ('Path. Trans.,' 1887, p. 412).

Malignant behaviour and cancerous structure.—It must be particularly noticed that the above characters which Virchow laid down as distinguishing malignant from innocent new growths, so far from being peculiar to any special kind of tumour, belong also to other processes, such as inflammation, tubercle, and infective lupus. These exhibit "local progression," and they sometimes pay no more respect to the natural boundaries of the tissues than does cancer itself. The question of their "recurrence after removal" is seldom or never raised. But each of them "spreads to lymphatic glands." Suppuration, at any rate, assuming the form of Pyæmia, undergoes "generalisation" or "metastasis" through the blood. Surely, then, it is more probable that malignancy should belong to several kinds of new growths, perhaps in different degrees, than that it should be a fixed property of one particular species, isolating it from all the rest. Now, that is precisely Virchow's doctrine. Other observers had independently taught the impossibility of drawing an absolute line of distinction between innocent and malignant growths. Sir James Paget described, in 1853, under the name of "*recurrent fibroid*," a growth characterised by an inveterate tendency to return *in situ* after removal, without infecting distant structures. Dr Wilks, in his 'Lectures on Pathology,' published in 1859, made a separate group of "semi-malignant" tumours including, besides the "recurrent fibroid" of soft structures, the "osteo-sarcoma" of bone; and he specially recorded instances in which "myeloid" or "enchondromatous" growths, which were then generally regarded as innocent, appeared metastatically in the lungs. Cancer was left as an independent affection, of which the chief varieties were Scirrhus, or hard cancer; Encephaloid, medullary, or soft cancer; Epithelioma, or horny cancer; Melanosis, or black cancer; Osteoid, or bony cancer; and Colloid, or gelatinous cancer. These forms of cancer were redistributed by Virchow, according to their histological characters. He showed that most instances of "encephaloid cancer," "melanosis," and "osteoid cancer," really belonged to the *connective-tissue* series of growths; and he associated scirrhus with epithelioma, since both forms consist of alveoli containing cells of an *epithelial* type.

Virchow's theoretical views have since been substantially adopted by all pathologists, and the words cancer and carcinoma are now only applied in their strictly histological sense.

Classification.—It is impossible to frame a completely satisfactory classification of tumours. But the following arrangement, which is adapted from that of Lücke, in Pitha and Billroth's 'Handbuch d. Chirurgie,' is probably as good as any. Like all others now in use it is itself a modification of Virchow's.*

* So-called cystic tumours are excluded. If merely dilated cavities containing fluid (retention-cysts), they are no more "tumours" in the modern sense than is a hydrocele. If due to an animal parasite (hydatids) they are not neoplasms, but foreign bodies. If degenerations of solid tumours, their place is with the structure of which they are the outcome.

A. Of the type of connective tissue : mesoblastic
  (α) Following normal or fully developed connective tissues.
    1. *Fibroma*, or fibrous tumour.
    2. *Glioma*, resembling the neuroglia.
    3. *Lipoma*, or fatty tumour.
    4. *Chondroma*, or cartilaginous tumour.
    5. *Osteoma*, or bony tumour.
  (β) Following embryonic connective tissues.
    6. *Myxoma*, resembling the tissue of the umbilical cord.
    7. *Sarcoma*, resembling undifferentiated mesoblastic tissue.
B. Of the type of lymph-glands : mesoblastic.
    8. *Lymphoma*, or lymphadenoma.
C. Of epithelial type : epi- and hypo-blastic.
    9. *Papilloma*, or papillary growth.
    10. *Adenoma*, or glandular tumour.
    11. *Carcinoma*, or cancer.
D. Of the type of higher tissues.
    12. *Myoma*, or muscular tumour.
    13. *Neuroma*, or nervous tumour.
E. Of composite type.
    14. *Angioma*, or vascular tumour.
    15. *Lymphangioma*, or lymphatic tumour.
    16. *Teratoma*, or embryonic tumour.

Many of these forms of tumour have surgical rather than medical interest, and do not need more than mention, but others which affect numerous internal organs will be more conveniently treated here than elsewhere.

1. *Fibroma.*—This is made up of fibrous tissue, which may be either arranged in loose meshes, or felted, sometimes very tightly. In the former case its substance is soft and succulent; in the latter it may be exceedingly hard, so as to creak when cut through.

True fibrous tumours are far from common. The most important are the multiple fibromata of nerve-sheaths, often called *neuroma*. They also occur in tendons and periosteum, particularly in children after rheumatism.

The remarkable little pedunculated fibrous growths, which are sometimes found covering the skin in immense number, will be described with cheloid and other cutaneous tumours, as *fibroma molluscum*. Similar growths of the mucous membrane of the intestine, uterus, or bladder are usually pedunculated, and are called fibrous *polypi*. Fibroma of the breast or ovary is a very rare disease. It is not uncommon for several small fibrous growths to be found in the kidneys, which appear as minute soft white masses, and are apt to be taken for secondary tumours, when there is a growth in some other part. More than once a blunder has only been avoided by the microscope.

1*a*. English writers have, of late, been accustomed to separate, under the name of *Myoma*, a common variety of tumour containing much fibrous tissue, on account of the presence in it of more or less numerous bands of smooth muscle. Most German pathologists now replace these growths, which are seen in the uterus and in the prostate, among the fibromata; and Rindfleisch declares that the opinion that they contain muscular fibres is a mistake. Some small "fibroids," as they are called, certainly have rod-shaped nuclei and other characters of muscle.

There are true myomata (*rhabdomyomata*), or tumours of striated muscle

sometimes found in the kidney and elsewhere. They are of purely pathological interest.

2. The modified connective tissue of the central nervous system, called neuroglia by Virchow, gives rise to a characteristic kind of new growth confined to the brain and (as a rare event) to the cord and the retina, and named *Glioma.* It will be described as the most frequent among "simple" tumours of the brain.

3. *Lipoma.*—This is a tumour consisting of adipose tissue like the subcutaneous fat, but circumscribed, and generally enclosed in a well-marked capsule. They are the largest of all new growths, are often multiple, and common on the surface of the body. When they occur in the interior of the body fatty tumours do not produce symptoms so as to come under medical observation, unless they are so large as to press upon important organs, which is very seldom the case. But Dr Frederick Taylor ('Path. Trans.,' vol. xxvii) met with a case in which a lipoma grew in the post-pharyngeal space of a child, and caused death by suffocation. In the abdomen such growths have occasionally reached an immense size, and have been mistaken for ovarian tumours.

4. *Enchondroma.*—Tumours made up of cartilage were so named by Johannes Müller, although *chondroma* seems to be the more correct form. Histologically they differ widely in different cases; the matrix may be either hyaline or fibrous, and the cells may present various characters. They usually begin in bone, occasionally in the testis or parotid, and are sometimes malignant in their course. As primary growths they scarcely ever come under the notice of the physician. Virchow speaks of multiple chondromata of the lungs, situated chiefly near their roots, and probably bearing some relation to the cartilages of the bronchia; but these, he says, are found accidentally in the *post-mortem* room.

5. *Osteoma.*—This is the technical name for growths which consist of osseous tissue, but it is not often used. For when a tumour projects outwards from a bone it is called an *exostosis,* and when it extends inwards (as into a frontal sinus) it is sometimes called an *endostosis*; and most other bony growths contain soft structures as well, which are regarded as the more essential parts. In medical practice an intracranial exostosis might cause the symptoms of a cerebral tumour. Moreover, as rare exceptions, bony tumours have been found in the brain and in the eye.

There is a very remarkable kind of growth, for which the name *Malignant Osteoid Tumour* seems the most appropriate, and which is of much interest to the physician, because it not infrequently comes under his notice rather than that of the surgeon. By Sir James Paget it was called "Osteoid Cancer." Virchow termed it "Osteoid chondroma," an unfortunate appellation, since (as he himself pointed out) the structure of the non-calcified part of the growth is not that of cartilage, but rather of periosteum. Moreover, as might have been anticipated, great confusion has arisen from its being supposed to be identical with an "ossifying enchondroma," which is a tumour really consisting of cartilage, the deeper and older parts of which have undergone conversion into bone. Wilks and Moxon term this growth "Periosteoma," and define it as representing the varied tissues of ossifying bone.

These malignant osteoid tumours are made up of an exceedingly dense, firm, and tough material, which is of a pale greyish colour, and cannot be teased out. Under the microscope it may appear more or less distinctly fibrous. Sir James Paget describes the fibres as "crisp and stiff," or as

moderately broad, "with uneven thorny edges," and arranged in bundles, "looking like faggots." Embedded in this substance are cells, which are rather small, of round or oval shape, without alveoli. When calcification takes place, earthy salts are deposited in hard granular masses, but in some parts more or less perfectly formed bone may be seen. The calcified growth has a peculiar dull white chalk-like or mortar aspect. It cannot be cut, and must be sawn through, but it may be "rubbed or scraped into a fine dry powder." Malignant osteoid tumours are most frequent in young subjects. Of nineteen cases collected by Paget, five occurred in persons between ten and twenty years old, nine in those who were between twenty and thirty, and none after fifty. They are generally attended with severe pain. Their favourite seat is the lower end of the femur. One in Guy's Hospital affected the humerus close to the shoulder. This kind of growth forms a very large, hard mass, surrounding the whole circumference of the bone, slightly if at all nodulated on the surface, and gradually sinking down to the level of the rest of the shaft. Thus it might seem to be outside the bone, but on section the medullary cavity and the cancellous tissue are found to be completely occupied by the opaque, hard substance above described. When a flat bone is attacked by it, the growth is said by Paget always to project from both surfaces. We had a remarkable case in which the two iliac bones were affected symmetrically, each having a large bossy prominence projecting from both the dorsum and the venter, which might have been felt during life. There may indeed be a large number of such tumours growing from different bones in the same patient, and the clinical aspect of the case may be that of a cerebral tumour or of paraplegia from compression of the cord. Or, again, the symptoms may be thoracic, from an immense mass lying at the root of the lungs, or from numerous nodules scattered in their substance and beneath the pleuræ. The secondary growths in lymph-glands and viscera look like the tumours of the bones themselves. They may be calcified in almost their entire extent, so that only a very little dry, tough, white, fibrous-looking substance remains round their edges. It was this which so surprised the older pathologists, who almost imagined that they had before them cases in which osseous tissue itself possessed the property of malignancy.

6. *Myxoma.*—The true relations of this form of tumour were first recognised by Virchow. Older synonyms for it are "collonema," "sarcoma gelatinosum," and "fibro-cellular tumour" (Paget), but in practice it was probably often confounded with colloid cancer. It consists of a semi-translucent material, sometimes so soft as to quiver like a jelly, of a bluish or yellowish tint, and emitting a sticky fluid when squeezed or scraped. This comes from the intercellular stroma, and owes its viscidity to the presence of mucin. The addition of acetic acid to a thin slice of the growth renders it white and opaque from precipitation of the mucin.

The cells are generally scattered at considerable intervals. They may be round, but they are often stellate, with long processes ramifying in the stroma. Virchow pointed out that this structure is identical with that of the gelatinous tissue, which in earlier fœtal life occupies the place of the subcutaneous fat, which at birth is found in the umbilical cord, and which constitutes the vitreous body of the eye. Myxomata are seen chiefly by the surgeon, but the physician may occasionally meet with them in various situations, even in the brain. Sometimes they are found in large numbers upon the nerve-trunks, constituting a part of the tumours which were formerly

incorrectly grouped together under the name of *neuromata*. Myxo-lipoma and myxo-chondroma are sometimes met with. Some polypi of mucous membranes are fibro-myxomatous in structure, and so according to Virchow are "hydatid moles" of the uterus.

7. *Sarcoma.*—Virchow referred to the *connective-tissue* series of growths as certain tumours which present a high degree of malignancy, especially those known as "encephaloid" and "melanotic" cancers. He grouped them with other tumours of innocent character, or which at most exhibit a tendency to return *in loco* after extirpation, and he adopted for them all the name of sarcoma. His views with regard to them have since been universally accepted; in this country the more readily because no confusion could arise. For the word Sarcoma, although it dates back to Galen,* and had been employed by many subsequent writers down to the time of Abernethy, had fortunately fallen into complete disuse except in the compound form of osteo-sarcoma.

A point on which Virchow laid some stress as distinguishing sarcomata from growths of which the cells were epithelial, was the absence in the latter of any intercellular substance or matrix. This still holds good in so far that, where the cells of a tumour are embedded in a structureless or granular material, the conclusion may be drawn that they are not epithelial; but the converse is not universally true, for the elements of certain spindle-cell and other sarcomata are often tightly packed together without anything between them.

The cells of a sarcoma may be of three kinds:

a. Round-cells.—These vary in character in different cases. Sometimes they are undistinguishable from leucocytes. Sometimes they are much larger, and they are then often exceedingly delicate, so that the addition of water to a microscopical specimen may cause the disappearance of all but the nuclei, which then seem to be free.

b. Spindle-cells.—These are the growths which by French writers had been called *fibro-plastic*, and which had in England been recognised by Paget and others as characterising the class of tumours which he termed "Recurrent Fibroid." They present many differences of size and appearance, but they all agree in having an elongated shape, tapering gradually into a point at each end, or having their ends prolonged as delicate sinuous fibres. Their nuclei are always elliptical, and sometimes narrow and oat-shaped. They are commonly "fasciculated," *i. e.* arranged side by side in bundles.

c. Giant-cells.—Under this name are now described certain bodies, which, however, are rather irregular masses of protoplasm than cells, and which contain round or oval nuclei to the number of ten, twenty, thirty, or even more. From similar elements being found in the medulla of bones, especially during fœtal life, as Kölliker and Robin pointed out, they were until recently termed "myeloid cells," or by French writers *myéloplaxes*. Indeed, the growths in which they occur, and which are comparatively infrequent, were by Paget, Wilks, and others described as a special kind of tumour. Virchow, however, insisted on the fact that such growths always contain spindle-cells as well, and that therefore their true place is among the sarcomata.

But different kinds of cells by no means constitute the whole of the formed elements of a sarcoma. It also contains blood-vessels, which are sometimes so large and receive so abundant a supply of arterial blood, that

* Σάρκωμα ἐστὶ σαρκὸς ἐν τοῖς μυκτῆρσι παρὰ φύσιν αὔξησις, ἐστὶ δὲ σάρκωμά τι καὶ ὁ πολύπους.—'Galeni de Deform. medendis.'

it may pulsate, so as to be mistaken for an aneurysm. The walls of the smaller branches of these vessels are usually themselves of embryonic character, consisting of spindle-cells applied to one another in such a way as to enclose a blood-channel between them. It is, therefore, not surprising that they are apt to allow blood to escape into the substance of the growth, where it may coagulate, so that on section the mass looks like a mere clot. Such tumours increase in size with extraordinary rapidity; and they were formerly known as *fungus hæmatodes*, a name now almost forgotten. In other instances, there are so few cells between the vessels that there may be the greatest difficulty in recognising the presence of any new growth.*

Again, instead of being embedded in a merely granular material, the cells of a sarcoma may be supported by formed stroma, consisting of connective tissue. This, however, is hardly to be regarded as equivalent to the ordinary intercellular substance, for the fibres have no doubt been developed out of a corresponding number of the cells, and have taken their place. The growth is in fact a mixed one, a *fibro-sarcoma*. Sometimes, as Billroth pointed out, there is a regular alveolar structure, like that which characterises carcinoma; the cells are, however, smaller and the stroma less developed. Of late years, since it has been thought that cancers proper can arise only where there is epithelium, "alveolar sarcomata" have acquired importance, from enabling pathologists to account for apparent exceptions to this rule.

Still greater complexity is caused by the combination, in the same tumour, of a variety of structures belonging to the connective-tissue series, such as cartilage, mucous tissue, bone or fat. Perhaps the occurrence of certain rare cases in which a growth, ordinarily innocent, sets up secondary tumours in distant organs may be explained by the presence in it of more or less numerous embryonic cells, the diffusion of which by the blood-stream causes the infection. According to this view all such tumours are "mixed," and should be designated accordingly. In a case at Guy's Hospital fibromata of the uterus led to the formation in the lungs of similar growths in which the appearances regarded by many pathologists as indicative of unstriped muscular fibres were as conspicuous as in the uterine tumours themselves. And in another case, that of an infant fifteen months old at the time of death, there were in the liver, secondary to a mixed sarcoma of the neck, a number of dense flat button-like nodules, which consisted almost entirely of well-developed fibrous tissue, although at the margins some spindle-cell elements were with a little difficulty discovered.

Another circumstance which greatly modifies the appearance of sarcomatous growths is the occurrence in them of degenerative changes, of which caseation is the chief.

Can we, without having recourse to the microscope, distinguish the

* In 1877, I made an autopsy in the case of a lad, aged fifteen, who had been under Mr Cooper Forster's care for a fracture of the femur, which seemed clearly to have been caused by external violence. Repair did not take place, an incision led merely to the escape of blood; and amputation was followed by the death of the patient. I found the injured bones and muscles soaked in blood, and at first I had no suspicion of there being any tumour elements. But presently I noticed that the blood-stained appearance of the muscles was limited definitely by convex margins; and the microscope showed masses of delicate spindle-cell tissue in them. In the lungs there were five or six scattered secondary nodules, the size of marbles, and reddish in colour, projecting above the level of the pleural surface. These, however, felt quite soft; and when cut into, each of them collapsed, leaving a cavity surrounded only by a very narrow margin of indefinite-looking tissue. For further details of this case see the 'Guy's Hospital Reports' for 1880, p. 17.—C. H. F.

great toe. He complained of pains in his right arm, in both shoulders, in the loins, and (especially during defæcation) in the lower part of the spine. He also suffered severely from headache. The urine was at one period albuminous, and the case was therefore regarded as one of Bright's disease of gouty origin. Subsequently the urine became normal, and some of those who saw the patient at that time suspected that he was exaggerating his symptoms. However, he grew more and more wasted and bloodless, and his pains became fixed in the iliac fossæ, especially on the right side. The most careful search was made for tumours, which it seemed could hardly escape detection in so emaciated a subject. A few weeks before death hæmaturia occurred, and this of course led to the suspicion of primary malignant disease of the kidney. At last he became unconscious, with rigid flexion of the right arm, and in this state he died. The autopsy showed that sarcomatous growths had occurred to an enormous extent, but that there was nowhere any tumour discoverable by manipulation. In each iliac fossa a layer of sarcomatous tissue was spread out beneath the periosteum, and there was a large quantity diffused over the surface and in the interior of many other bones. One tumour, which was removed, and sawn vertically through, had in its cancellous tissue many rounded masses as large as walnuts. On the under surface of the dura mater there were a number of button-like nodules indenting the brain. One kidney had two small tumours affecting the mucous lining of its pelvis. The growth consisted of large cells of the most irregular form embedded in a fibrous matrix.

Another case is that of a boy, aged sixteen, who came under the writer's observation when clinical clerk to Dr Owen Rees in 1854. He said that he had been strong and robust until eleven weeks before his admission, when he began to suffer from pain, at first in the loins, and afterwards in the shoulders and limbs. There was great tenderness of the whole surface of the body. He had also experienced a sensation of tingling in the area of distribution of the ulnar nerve to each hand. He was a very delicate-looking lad, with a clear complexion, a pink flush on each cheek, a moist skin, a white tongue, and a pulse of 144. The first diagnosis was of subacute rheumatism; subsequently of chronic inflammation of the spinal membranes. He lay for about two months, during which time he became emaciated to the most extreme degree. Towards the last, masses of enlarged glands could be felt through the walls of his contracted abdomen. He continued to suffer excruciating pain on the slightest movement, and there was exquisite tenderness of the skin. One day he became insensible, and had a convulsive attack, after which he lived only three hours. A large mass of medullary sarcoma was found in front of the spine, eroding the vertebræ, and involving the nerves as they emerged from the spinal canal.

Sometimes the growth of multiple sarcomata leads to a still more rapidly fatal illness. In vol. xxv of the 'Guy's Hospital Reports' are recorded certain cases which were attended with purpura and other hæmorrhages from mucous surfaces. One of the most curious is that of a man, aged twenty-five, who was admitted in 1879 for what was supposed to be an attack of rheumatism. He said that on Whit Monday, June 2nd, he had got wet through; and from that time up to his admission, two months later, he had complained of pains in the left shoulder, in the chest, and in the hips. He had been confined to bed for nine days with profuse sweating; the temperature varied from 102° to 102·3°. The skin had an unpleasant sour smell,

and a systolic apex murmur was detected on one occasion when he sat up. He went on well for twelve days, when a purpuric rash came out on the chest and the abdomen, hæmaturia occurred, and his eyelids and his scrotum became greatly swollen. Five days later, on August 14th, he died. At the autopsy the scattered spots of purpura still remained visible. Some of them were flat, but others were slightly raised and indurated, and a few had a central pale elevation with a narrow ring of purple discolouration around it. These proved to consist of small round or irregular cells infiltrating the little lobules of subcutaneous fat. In the kidneys there were several white or pinkish sarcomatous nodules. In the cæcum and adjacent part of the ileum there was what appeared to be the primary growth, a homogeneous-looking yellow mass, of considerable thickness, involving all the intestinal coats.

Another case occurred in a patient, aged thirty-eight, under Dr Moxon in 1877. Up to five weeks before admission he had always, he said, been a strong man. He then "caught cold from being exposed to draughts." After this he suffered from pains in the back, legs, arms, and chest, chiefly in the joints, and flying from one joint to another. Five days before he came into hospital he was attacked with severe pain in the back, hæmaturia set in, and purpuric spots came out on the neck, the groins, and the legs. Afterwards he had epistaxis, his gums became sore, and bled. His temperature had been taken before admission, and was found to range from 100° upwards. On admission he was much blanched and extremely weak, with no perceptible pulse. The nose began to bleed almost immediately, and on the following morning he died. The kidneys were found to contain a large number of white tumours, and there was in the right vesicula seminalis a firm growth, to which they were apparently secondary.

A third case was that of a man, aged twenty-eight, admitted under the care of Dr Wilks in 1872. He said that for some weeks he had suffered from headache and neuralgia, that his teeth had been loose and painful, and that his gums had been spongy and had bled. For ten days he had been suffering with severe pains in the elbows, the knees, and the shoulders, but these seemed to be diminishing in intensity. He was an anæmic man, with a brown tongue, foul breath, and teeth and gums caked with dried blood. His pulse was 128, his temperature 100·2°, his respirations 28. His gums and his nose continued to bleed, and eight days after his admission purpuric spots appeared on the abdomen. On the following day he died. A whitish firm growth was found in the anterior mediastinum, probably affecting the thymus; and there was a large quantity of a similar material in the subserous tissue of the peritoneum and in the mesentery.

A fourth case, somewhat like the others, occurred in February, 1876. The patient, a bank clerk, aged twenty-five, said that he had got very cold about a fortnight before Christmas, 1875; for three days he was chilled through, and he was never well afterwards. His temperature was 101°. His mouth and his tongue were stained with blood, his gums were slightly spongy, and he had purpuric spots on the legs. It appeared that he was not in the habit of eating any vegetables, and the disease was supposed to be scorbutus. But he rapidly grew extremely anæmic and feeble, hæmorrhage from the bowels set in, the lymph-glands in various regions of the body became enormously large, and at the end of a few months he died. There was no autopsy.

Different views may be taken as to the relation between the symptoms which presented themselves in these various cases, and the underlying

other hand, few German pathologists employ it at all. So that its true position in the nomenclature of disease is that of being a French equivalent for the German term lymphoma. Several English writers apply it in a more limited sense, to correspond with what will be hereafter described as "Hodgkin's disease." In that morbid condition there are, indeed, growths which spring up in immense numbers, and sometimes with extreme rapidity, not only in the lymphatic glands and the spleen, but in almost every organ and tissue; and they may possess at least the first of Virchow's characters of malignancy, spreading from one part to another without regard to the natural boundary lines between them. It is impossible to reserve for them the term lymphadenoma, or to describe them under any one pathological designation. For their histological characters vary widely in different instances, so that we must regard "Hodgkin's disease" as a clinical name for a group of cases in which the same organs are affected, and of which the symptoms and course are similar, but not as anatomically denoting any one special kind of tumour.

Another term, "lymphosarcoma," also needs explanation. Like lymphadenoma, it has been employed in somewhat different senses by different writers. Some seem to apply it to any sarcomatous growth having its starting-point in lymphatic glands; they forget that the seat of a sarcoma is in itself a matter of indifference, and certainly does not require to be marked by any special name. On the other hand, Virchow is disposed to designate as lymphosarcomata all lymphomatous tumours of any size, of which the elements show no tendency to caseate, and which undergo progressive and sometimes very rapid enlargement; he would include under this head most instances of Hodgkin's disease, and all the lymphomatous tumours of the mediastinum. But it is certain that many of the cases in question, even where a large number of organs are involved, exhibit no histological characters other than those of a pure lymphoma. The only logical course seems to be to reserve the name of lymphosarcoma for such growths as appear to be transitional between a lymphoma and a sarcoma. Lymphoma will then denote a tumour composed of lymphatic, adenoid, or cytogenic tissue.

New growths of epithelial type.—As already remarked, we owe to Virchow the important doctrine that the "cancer-cell," which was believed by histologists to be heterologous, *i. e.* unlike any of the elements of the healthy body, is, after all, identical with the cells of epithelium. He explicitly pointed out this fact in a paper in the first volume of his 'Archiv,' published in 1847; and at the same time he indicated the region in which the most perfect representatives of cancer-cells are to be found, namely, upon the mucous surface of the ureters and of the bladder, especially in infants. He also made a striking comparison between the alveolar structure of a cancer and that of the lung in a case of lobular pneumonia, where the pulmonary vesicles are filled with corpuscular exudation which is just turning into pus. It is a remarkable fact that, at a much later date, pathologists, still ignorant of his teaching, actually made the diagnosis of cancer in the urinary passages or in the lungs, on microscopical evidence alone, in cases where no disease was present.

It is much to be regretted that Virchow did not carry out his usual method of nomenclature, and stamp with his authority the practice of applying the term "epithelioma" to all tumours of epithelial type. Unfortunately,

it was already in use, as a designation for a very limited group of cancers, characterised, however, not by the presence of ordinary columnar or spheroidal epithelium, but by that of horny epidermic cells. Hereafter, perhaps, the term "Epithelioma" will universally be employed, in the wider sense which seems naturally to belong to it.

Epithelial growths, however, are not all of them included under cancers. There are certain innocent forms which bear the names *papilloma* and *adenoma*. These bear the same relation to one another which an intestinal villus does to a Lieberkühn's tubule. The former consists of a solid protrusion outwards of mucous membrane or of skin, upon which epithelial cells are arranged; the latter is a hollow inversion of mucous membrane or of skin, with an epithelial lining.

9. *Papilloma.*—Of this we have examples in common cutaneous warts, and in the horny growths which are sometimes seen upon the face, neck, or chest. Another common variety occurs as warts on the genital organs, and a fourth as condylomata and mucous patches; these will be mentioned among diseases of the skin. Yet another form is sometimes met with in the larynx, especially at an early period of life. Lastly, there is an affection to which the vesical mucous membrane is liable, and which is known as "villous tumour" of the bladder, or sometimes (on account of the alarming and dangerous symptoms to which it gives rise) as "villous cancer." It consists in the formation of one or more tufts of long, soft, delicate-branched filaments, each containing a wide vascular loop, and covered with a series of layers of large epithelial cells. This kind of new growth does not spread to lymph-glands, nor produce metastatic nodules in distant parts. Billroth still continues to place it among the carcinomata, on the ground that masses of epithelial cells lie in meshes at the basis of the villi; and in some cases the resemblance to the structure of a cancer is certainly very close.

10. *Adenoma.*—Of this form of tumour (which resembles true gland tissue, not that of a lymph-gland) an instance may be found in molluscum contagiosum. Other examples also are met with, corresponding some with the sebaceous, some with the sudoriparous glands. Upon mucous membranes the most common variety is one which constitutes a large number of so-called "polypi," growing from the surface of the intestine, of the cervix uteri, or of the nasal fossa. In the female breast adenoma or adenocele, the "chronic mammary tumour" of Sir Astley Cooper, very frequently occurs.

11. *Carcinoma.*—The structure of the two kinds of new growth last described is by no means always identical with that of the part in which they arise; thus a nodule of molluscum differs considerably from the healthy skin in its histology, and a villous tumour of the bladder from the vesical mucous membrane. So that it is impossible to maintain in its integrity the dictum of Waldeyer, which is cited by Lücke with approval, that the carcinomata can be absolutely distinguished from other epithelial growths by their being "atypical." Probably the fact is that, as in so many other departments of pathology, no positive line of demarcation exists. The carcinomata, indeed, may be said to combine in many cases the distinctive features of a papilloma with those of an adenoma, in so far that they consist partly of outgrowths covered with epithelial cells, partly of inversions lined with them. Thus a cancer of the skin often begins in a growth resembling a wart; many cancers of the stomach, of the intestine, and of the gall-bladder present abundant villous processes; and in the os uteri, nothing is more

common than for a cancer to begin as what is termed a "cauliflower growth." The glandular type is shown in the characteristic alveolar and epithelial structure of cancer.

a. Ordinary carcinoma (*glandular cancer, malignant adenoma*) presents a great many varieties of structure according to its exact seat. But it is always made up of oval or rounded alveoli, which contain cells like those of epithelium. The walls of the alveoli usually consist of fibrous tissue, and they are more or less abundantly supplied with blood-vessels. In very soft, rapidly growing cancers, however, the interalveolar stroma may be composed of spindle-cells; or, in other words, its tissue may be sarcomatous. Otherwise the softness or hardness of a carcinoma depends chiefly upon the proportion between the amount of stroma and the size of the alveoli. In many instances the substance of the growth is as easy to break down as that of a healthy spleen, or even of a spleen from a case of fever; and then one finds that the alveoli are large and that their walls are made up mainly of blood-vessels, and have but little fibrous tissue in them. Sometimes it is so hard as to be spoken of as "stony;" the microscope then shows that it consists almost entirely of interlacing dense white bands, the alveoli being very small, and perhaps so few in number that their presence may easily be overlooked. It is to such growths that the old name of "scirrhus" is still applied. They are commonly very slow in their course; and they contract and shrink so much that their general appearance is often that of a cicatricial relic, rather than of a tumour; and if they occupy the wall of a canal (such as the intestine or the common bile-duct) they greatly narrow it.

As to the extent to which the alveolar walls and stroma of a simple carcinoma are a new formation, there is still some uncertainty; and perhaps different cases differ in this respect. The analogy of keratoid carcinoma, which will be described further on, suggests that they may be in great part made up of pre-existing tissue-elements, modified in their arrangement by the pressure of the masses of epithelial cells which are in contact with them on all sides. On the other hand, it is certain that in growing cancers the alveolar walls generally contain numerous leucocytes, from which a new fibrous tissue may well be supposed to be developed. As Waldeyer remarks, when a carcinoma spreads in the interior of a thrombus within the channel of a vein, its alveoli can be nothing else than entirely new formations. In cases of this kind, however, the alveoli are sometimes ill marked.

In thin sections of a carcinoma the alveoli look like closed spaces, but in reality they freely communicate with one another in different planes. Thus, when one gently squeezes the cut surface of such a growth, or when it is scraped with a knife, the cell-masses, with more or less of an albuminous fluid, escape readily, forming what has long been known as the "milky" or "cream-like" *cancer-juice*, or appearing as a soft, curdy, solid substance. The cell-masses themselves—the "*cancer-bodies*," as Waldeyer proposed to call them—may sometimes be withdrawn in continuity from two or more alveoli, so as to have a branching or reticulated appearance. The cells of which they are made up present every variety of shape, "polymorphism" being indeed one of their characters. Each has commonly a large, sharply defined, round or oval nucleus, and sometimes two or even more. The protoplasm is abundant, sometimes clear but often granular.

b. Keratoid carcinoma.—This is the form of growth which is commonly known in England by the inappropriate name of "Epithelioma," originally

given to it by Hannover, of Copenhagen, in 1852. Many French writers and the late Dr Hughes Bennett, following Lebert, called it "cancroid." Its characteristic is the fact that some of its cells undergo a chemical change identical with that which constitutes *cornifaction* in the superficial layers of the epidermis. The horny cells are situated near the centre of the "cancer-bodies" in which they occur. They become fixed together, and the pressure to which they are subjected moulds them into globular masses, more or less laminated in structure, but often having a transparent centre, which looks like a single large cell. These bodies were called by Lebert "epidermic globes." In this country they are known as "birds' nests." It must be clearly understood that in themselves they afford no proof whatever of the presence of a malignant new growth. On the contrary, they may be found under various circumstances, as, for example (according to Thiersch), "in lupus, in the hyperæmic borders of cicatrices, in sebaceous tumours, and in the epithelial covering of the gums." Their real importance lies in the fact that when they are seen in a new growth they show it to be not merely a carcinoma, but one which has its origin either in the skin or in some mucous membrane having a squamous, laminated epithelium. This form of cancer is exceedingly frequent about the orifices of the body, especially the lips, the anus, and the vulva. The mucous membranes upon which it may occur are those of the mouth, tongue, pharynx, larynx, and œsophagus, the lower part of the rectum, the urethra and bladder, the vagina and os uteri. In many of these situations, however, it is common to meet with growths which, although the presence of horny globes justifies the title of keratoid carcinomata, yet contain the globes in small numbers, and in their general appearance and structure approximate very closely to the ordinary form of cancer. Such transitional varieties are met with in the bladder, and they are very common in the cervix uteri. In the œsophagus they appear to increase in frequency from the pharynx downwards, until at the cardiac orifice of the stomach a point is reached at which the liability to cornifaction in the cancer-bodies ceases altogether. Another proof of the close relation between the two forms of cancer is sometimes afforded by an examination of the structure of the secondary nodules in distant parts from a case in which the primary growth is keratoid. In some instances a nodule in the kidney or in the lung will present horny laminated globes (birds'-nest bodies) as well formed as those in the original seat of disease in the tongue or œsophagus. In a recent case of keratoid cancer of the lip in which the cervical glands contained typical globes, some large secondary masses in the liver, which were softening centrally into cavities, had more the structure of a simple carcinoma, the indications of cornifaction of the cells being slight and imperfect. The keratoid form of cancer is, however, much more apt to infect the lymph-glands which correspond with its primary seat than to be carried to distant parts by the blood-stream.

In the more characteristic cases of keratoid carcinoma, the material which can be scraped or squeezed from the cut surface of the growth is firm and granular looking, like the secretion of a sebaceous gland. It often comes out in the form of long worm-like masses.

Histologically, keratoid carcinoma differs from common cancer in some other respects as well as in containing the "globes" or "birds' nests." Not infrequently some of the cells have prickle-edges. Another peculiarity is that the "cancer-bodies" are much more plainly seen to form continuous bud-like branching processes which grow more or less vertically downwards

from the surface, penetrate the deeper structures and push them aside. Indeed, in its typical forms keratoid carcinoma may be said to present no definite alveoli. The substance that intervenes between the different cancer-bodies was all of it pre-existent, and it has undergone little change except that it is commonly infiltrated with leucocytes.

It seems now to have been clearly proved that the so-called "*Rodent ulcer*," which occurs chiefly on the face of persons advanced in life, and which is characterised clinically by its very slow course, is only a variety of keratoid carcinoma. In all probability the same may also be said of the affection termed "*Cylindroma*" by Billroth, in which certain peculiar hyaline bodies are found.

c. Columnar carcinoma.—In 1852 Bidder, of Dorpat, described in 'Müller's Archiv' a case in which the cells of a soft cancer of the pylorus were identical with those of "columnar" or "cylinder epithelium." Many other instances of the same kind have since been recorded under the title "cylinder" or "columnar epithelioma." The area of distribution of this form of tumour, as a primary growth, includes the stomach and intestines, from the cardia downwards to within a short distance of the anus, the biliary passages, and gall-bladder, and (according to Cornil and Ranvier) the nasal fossæ, the upper part of the uterine cavity, and perhaps the ovaries. Secondary nodules may occur almost anywhere: we have found them in the liver and the brain, and other observers record them in the lungs and in bones. The appearance of a primary columnar carcinoma is commonly that of an irregular shallow ulcer with a soft, raised, slightly projecting border. It may yield an abundant juice, full of the columnar cells, which are often still coherent sideways. A thin section generally shows that they are regularly arranged around the borders of long-branching channels or alveoli. But, as Cornil and Ranvier admit, some of them may be polymorphous. Indeed there are clear transitions between this and other forms of cancer, not only among different primary growths, but also in comparing primary and secondary growths from the same case, when the latter sometimes approximate to the common type of carcinoma.

Histogenesis of carcinoma.—That epithelium alone can produce epithelium is almost established by what is known of the healing of wounds and ulcers from their margins, and by the success of Reverdin's practice of transplanting cuticle. So also primary tumours of epithelial type arise only in those structures which contain epithelial elements. This fact is perhaps not quite without exceptions. German writers still cite a case of Virchow's, recorded in 1850, in which the tibia is said to have been affected by a primary "cancroid," the skin being healthy, and Otto Weber is quoted as having in 1859 related a case in which there was a similar lesion of the lower jaw. One may, however, hesitate as to the value of observations made at a time when their theoretical significance could hardly have been appreciated. Far more important is the fact that Waldeyer and other recent investigators have failed to meet with a single instance of the same kind. It would, indeed, be easy to account for the occasional occurrence of exceptions to the rule. Apart from the difficulty of discovering in all cases the growth which is really primary, it might be argued that it is possible for a lesion starting from the skin to extend downwards and to spread into the deeper structures, so as to appear to have begun in them. Paget, for instance, speaks of having "seen two examples of primary epithelial cancer in lympathic glands;" one was in a sweep, whose

groin contained a large mass, notwithstanding that the penis and the scrotum appeared to be unaffected. But it may be doubted whether the microscope might not have thrown fresh light upon the matter. Thiersch records a case in which a nodule seemed for a time to be subcutaneous, but really had its origin in the sweat-glands. Some carcinomata seem definitely to arise from the cells belonging to the glands of the skin or mucous membrane, rather than from those which cover the surface. Further, it is conceivable, as was suggested by Remak in 1854, that a fragment of germinal epithelium, embedded in other tissues from fœtal life, may afterwards develop into a cancerous tumour, instead of forming an innocent dermoid cyst. According to Thiersch it is no uncommon thing for cancer to arise in the wall of such a cyst after it has existed for many years; and Waldeyer cites an instance in which a similar change occurred in a congenital sacral tumour.

Even recently, however, some pathologists have maintained that although epithelial cells are formed only in contact with pre-existing epithelium they yet arise out of cells which are not themselves epithelial. Klebs speaks of an "epithelial infection," by which, for example, in a healing ulcer the rete mucosum of the skin at its margin is supposed to convert the cells of the adjacent granulations into epithelial cells. But, as Waldeyer remarks, there are striking instances in which a cancer fails to impress its own characters even upon epithelium with which it happens to come into relation. Thus, although the secreting cells of the liver are directly concerned in the development of some of the primary carcinomata of that organ, they are pushed aside by secondary nodules, and take no part in their formation.

In 1867 Köster propounded the view that the cells of cancerous growths in the skin are formed from the endothelia of lymph-channels. This, however, has met with no support; Waldeyer says that he has repeatedly seen cancer-bodies lying in immediate contact with perfectly unaltered endothelium, and although he admits that they often penetrate into lymph-channels and occupy them, he maintains that it is far from being always the case. On the whole, if we are not to cling to the notion that the epithelial cells are solely devloped by germination or fission from pre-existing epithelium, it would seem to be more easy to accept the view that they arise out of ordinary leucocytes. The tissues in immediate contact with a primary cancer are very commonly infiltrated with leucocytes; and, if they do not contribute to the new growth, their presence requires some other explanation. It is more likely that they are the results of a reactive or inflammatory process, of which a more intense degree is seen as ulceration of the growth. Waldeyer observed that the cancer-cells themselves are altogether passive when ulceration occurs; they break down and escape with the discharge. But there can be little doubt that the putrid and offensive character of the ichor which exudes from an open cancer is due to the extension inwards of a septic contagium along the epithelial columns and through the alveoli, which, as we have seen, communicate with one another.

Caseous decay.—Carcinomata are liable to certain retrograde changes beginning within their substance. Like sarcomata and almost every other kind of hard growth, they are apt to undergo "caseation." Whether this is altogether spontaneous—an indication that the cancer-bodies have reached their natural term of life—or whether it is due to interruption of blood-supply, has not, apparently, been ascertained. It often affects all but the narrowest possible growing margin of a tumour, especially of a

secondary nodule. And not uncommonly, where a caseating tumour is seated in the interior of a solid organ, such as the liver, its centre softens down so as to produce a cavity filled with a straw-coloured albuminous liquid.

Colloid carcinoma.—The most remarkable transformation of cancers, and one which never occurs in sarcomata, is that known as "*colloid.*" Chemically it resembles the substance of the thyroid body when enlarged. Colloid material differs from mucus in not being precipitated or rendered opaque by acetic acid, and also in containing sulphur. Cancers which undergo this change sometimes occur in the breast, but scarcely anywhere else except within the abdomen. At the pylorus one can sometimes trace very clearly the gradual conversion of a simple carcinoma into colloid. Until the nature of the colloid degeneration was understood by pathologists, there was no greater puzzle. The abdomen was sometimes found filled with enormous gelatinous masses, in which the microscope showed no definite structure, but which yet penetrated organs and destroyed them in such a way as clearly to show that it possessed malignant characters. In 1847, Virchow imagined it might be a persistent condition of the gelatinous exudation out of which, at that time, all cancers were supposed to develop. Long afterwards it was described as a separate variety of carcinoma; but of late the doctrine has met with general acceptance that it is not a primary species of tumour, but the result of a peculiar degenerative process. There can be no doubt, however, that much new colloid material is in a growth which is undergoing this change, for its alveoli have so enormously increased in size that they are often visible to the naked eye. Indeed, formerly, when the alveolar structure of all carcinomata was not known, the terms "alveolar," "cystic," or "reticulated" cancer were often applied to what we now call colloid.

The epithelial cells become swollen out into glistening, structureless globes; and it is only towards the centre of some of the alveoli that a few unaltered cells may still remain visible. Immense masses of cancer, having undergone this degeneration, sometimes fill the abdominal cavity, lying in great part loose and unattached to the serous membrane. In such cases it may be only after careful search that one discovers any portion of the growth in an unaltered state. The primary growth is usually found in the stomach or intestine. Secondary tumours, even when apparently recent, reproduce the colloid structure, and this fact renders it doubtful whether this structure is really a mere degeneration.

Ætiology.—There is no doubt that malignant growths are hereditary more frequently than others. The experience of Sir James Paget is very striking; in his hospital days he found that the proportion of cases in which a family tendency could be traced was one in six; subsequently in private practice, he made it one in four, and still more recently it has become one in three. It is true that these results might perhaps lose some of their significance if we were to inquire as to the proportion of healthy persons, chosen at random, who would be found to have had one or more relations affected with cancer, supposing their family history to be scrutinised with sufficient care. Some of the cases recorded by Paget and by others are far too striking to be set down as mere coincidences. Thus a lady, who died with cancer of the stomach, had seven children and about thirty grandchildren who grew up. Some of them were still living; but a daughter had already died with cancer of the stomach, two granddaughters with

cancer of the uterus, a granddaughter with cancer of the breast, a grandson with cancer in the bladder, a grandson with cancer in the rectum, and a grandson with cancer of the axillary lymph-glands.

A point of considerable importance is that where the disease is transmitted by inheritance it often appears in the descendants at a much earlier age than in the first patient. Thus, a young lady of twenty-four died of "epithelial cancer" of the pharynx; her mother had been attacked by cancer when between forty and fifty years old, her grandfather between sixty and seventy, a great-aunt at about forty, her great-grandmother at eighty or older. A schoolfellow of the writer died of cancer of the rectum before he was forty years of age; his father and his grandfather were believed to have also suffered from cancer, but at advanced periods of life.

In the 'St Bartholomew's Hospital Reports' for 1866, Mr Morrant Baker analysed 103 of Paget's cases, and carefully investigated the question whether the appearance of cancer in successive members of the same family could be attributed to the mere propagation of a *local* defect, or (in other words) of a tendency of some one organ to a special morbid change. But he found that it was hardly more common for the disease to occur at the same spot in two members of the same family, than for its seat to be different. Hence it would appear that we cannot explain the inheritance of cancer as a case of inheritance of the structure of particular organs.

A scarcely less important question is whether heredity is limited to cancer or applies to all neoplasms. On this point Cohnheim states that sometimes a mother suffers from adenoma of the breast, and her daughter afterwards from cancer of the same organ; and among Paget's observations there are several in which it seems clear that carcinoma occurred in one member of a family and sarcoma in another. So again, Sir William Jenner mentions the case of a man who had cancer of the tongue, and whose child, twenty-two years before, had died at the age of two or three years with disseminated malignant growths, which one may presume to have been sarcomata. In truth, there are grounds for stretching to the widest possible extent our conception of the inheritance of a tendency to develop tumours. In the course of the discussion of the Pathological Society in 1874, Mr Hutchinson remarked that persons who had common warts in large numbers generally knew of relatives affected with cancer. And Dr Goodhart shared with the writer the impression that, in the deadhouse at Guy's Hospital, it was very common to find all kinds of innocent tumours in the bodies of those who had died of malignant growths. Moreover, Paget has insisted on the frequency with which liability to sebaceous cysts, or to certain multiple osseous tumours, is transmitted by inheritance.

Thus, it appears that the hereditary propagation of cancer is only part of the widely-spread occurrence of new growths of various kinds in certain families.

Clinical experience confirms Paget's words, that the appearance of cancer often follows quickly after "deep anxiety, deferred hope, or disappointment." Cancer is more common in elderly persons than in children or young adults; the malignant growths of bone and other organs, which are not very rare in young children, are almost always sarcomata. It is not more frequent in men than in women, nor in the sickly than in the robust.

The embryonic theory of tumours.—According to Cohnheim, there is but one way in which a new growth can arise in an adult, and that is by a

portion of *embryonic tissue* having become arrested in its development during fœtal life, and having remained shut off until its dormant capacities have ultimately been roused into activity. Such an hypothesis was propounded by Virchow to account for the remarkable fact that enchondromata arise in connection with bones, but never from permanent cartilage; he supposed that a fragment of the original cartilaginous precursor of the bone might remain unossified, and might ultimately form the starting-point of a tumour. Cohnheim maintained that cancers are particularly apt to arise at spots which (as in the case of the orifices of the great mucous channels) are the seat of somewhat complicated processes in the development of the embryo, so that involutions of the external germinal layer may be supposed likely to occur there. The ultimate formation of the new growth he attributed to a failure of "physiological resistance" on the part of the normal tissues around. A somewhat similar notion was put forth by Thiersch in explanation of the liability of aged persons to keratoid carcinoma: he attributed it to the atrophied and inelastic condition of the fibrous texture of the skin at advanced periods of life, whereby he supposed the extension downwards of the epidermis to be facilitated.

There can be no doubt that Cohnheim's theory is readily applicable to certain kinds of tumour. It is probably true so far as the dermoid cysts are concerned; and it may perhaps account for the frequency with which growths arise in the uterus or in the breast of unmarried women in the later years of life, since in such persons the organs in question no doubt contain germs which fail to receive their normal physiological stimulus. In the case of the breast Dr Creighton has worked out a similar idea with extreme care and in great detail.* Having studied fully the normal process of evolution or "unfolding," which the mamma undergoes when preparing for its secretory functions, he finds that the growth of tumours in it may be regarded as a modification of that process, under what he terms "spurious stimulation," occurring at a time when the organ is in a quiescent condition.

It is obvious, though Cohnheim did not seem to think so, that such theories afford no explanation whatever of the mystery of tumour-development in general, or of the striking difference between innocent and malignant tumours. Why a fragment of embryonic tissue, after lying dormant for years, should produce a carcinoma or a sarcoma still remains entirely unexplained.

Moreover, there are facts to show that the theory of dormant embryonic masses is at least not universally true, particularly the relation of tumours to *injuries*, and to various local and accidental lesions. Cohnheim, it is true, altogether denied the traumatic origin of cancers. He cited Boll's statement that in 12 per cent. of the cases of cancer operated on by Langenbeck there had been an injury before the growth was developed; but he declared that such a mode of causation is impossible. He believed that his experiments on animals had enabled him to study all the morbid processes which are capable of resulting from injuries, and that such observations are final. The following cases related by Paget seem to prove the contrary.

A boy was accidentally wounded in one eye, which was sound. Within a few days, a medullary tumour grew from the eyeball; it was removed three weeks later, but it quickly recurred, and destroyed life. Another boy fell and struck his knee; swelling followed, which was at first supposed to be inflammatory; but it increased, and proved to be a large medullary growth

* 'Contributions to the Physiology and Pathology of the Breast,' 8vo, London, 1878.

round the lower end of the femur. A man at his work slipped and strained or broke his fibula; the injury led to pain and swelling, and in the course of eight weeks there was found to be a growth of large size outside and within the shaft of the bone.

Many instances can be cited in which a malignant tumour appears to arise out of some *chronic local lesion*. Thus it is well known that a keratoid cancer sometimes appears in the cicatrix of a burn on the arm or on the hand, where such a growth is otherwise rarely seen. A similar affection of the tongue often follows leucoplacia, or white syphilitic patches, at the end of several years. In the common bile-duct, a carcinomatous growth is very frequently traceable to irritation of its mucous membrane by gall-stones. And in more than one instance a malignant growth in the posterior wall of the urinary bladder has seemed to have been caused by the repeated introduction of catheters.

Cancer of the lip is apparently sometimes due to the irritation of a pipe; "chimney sweep's cancer" of the scrotum was almost certainly caused by the irritation of soot; cancer of the breast is often preceded by the kind of eczema of the nipple known as Paget's disease; and cancer of the glans penis is particularly common in cases of phimosis.

In the same way we may probably explain the predilection of malignant growths for the fauces, œsophagus, pylorus, sigmoid flexure and anus, the parts of the alimentary canal where mechanical friction is greatest.

Perhaps, however, these facts are not so incompatible with Cohnheim's theory as he himself supposed. Injury or irritation of a part gives rise to the formation of a new growth only as an exception; and it is surely not impossible that a dormant fragment of embryonic tissue which happens to be present may find in the injury or irritation the stimulus needed to rouse it into pernicious activity.

The cases in which it is most difficult to admit the embryonic hypothesis seem to be those of cancer in the scars of burns, and (on account of their frequency) those in which cancer follows white spots on the tongue, or in which cancer of the bile-ducts is set up by gall-stones.

SPECIFIC INFECTIOUS DISEASES

AFFECTING THE WHOLE BODY AND ACCOMPANIED BY FEVER

Nunc, ratio quæ sit morbis, aut unde repente
Mortiferam possit cladem conflare coorta
Morbida vis hominum generi pecudumque catervis
Expediam. LUCRETIUS.

Nosology—Bases of classification—General and local diseases—Specific Fevers.

IN arranging the order of diseases in a systematic treatise we cannot attempt a natural, perfect, or scientific classification.

For, in the first place, diseases are not comparable things. One is an anatomical change of structure; another is a pathological process; a third the result of the action of a mineral poison, a mechanical injury, or a parasitic animal or plant; while a fourth is only a frequently recurring series of concomitant symptoms, of which we know neither the material conditions nor the probable cause.

We might arrange diseases by the *regions* they affect; as those of the head, the breast, the belly, and the limbs; or by the *tissues* affected, as bones, joints, mucous membranes, skin; or by the *organs*, as brain, lungs, heart, liver. We should need a separate class for diseases which affect more than one organ, another for those which appear to affect every organ at once, and still there would remain diseases "of uncertain seat."

A *pathological* arrangement would group together diseases in which inflammation was the most important morbid process; those which consist of pyrexia without local inflammation; contagious and miasmatic diseases; degenerations of tissue; new growths—and so on as far as our knowledge of morbid physiology extends—whatever the region, tissue, or organ which these processes affected.

Or we might make an *analytical* arrangement, founded on the most definite and obvious symptoms; and consider successively all diseases which are characterised by a raised temperature, by dropsy, by dyspnœa, by jaundice, by the presence of albumen in the urine.

Lastly, an *ætiological* classification would put in one group those diseases which depend upon the invasion of animal or vegetable parasites, in another those which are produced by lead, mercury, or other poisons, in a third and fourth dietetic and climatic diseases.

But even if our knowledge of every "disease" were as complete as it is of alcoholic cirrhosis of the liver, syphilitic orchitis, aneurysm, scabies, or anthrax—if we had completed the task of Morgagni, and had discovered the seat and the cause of every malady to which the human race is liable—we should still find that no single principle of classification would give satisfactory results.

The object of medicine is to prevent, to predict, and to cure. These objects are sometimes best served by knowledge of morbid anatomy, sometimes by knowledge of processes, and sometimes by knowledge of causes.

Who is to decide whether so well understood a disease as lead-palsy will be best treated along with colic and other effects of plumbism, or with other forms of paralysis, or with wasting affections of the muscles, or as a peripheral neuritis? For different purposes each arrangement would be the best.

Every kind of classification which rests on a firm basis of facts is useful so far as it helps us to remember certain common characters of the objects classified, and mischievous if it is allowed to obscure other common characters upon which other classifications may be constructed.

In a systematic treatise, however, it is necessary to choose some one arrangement, and our choice should be decided by reasons of practical convenience. Our divisions need not be upon a uniform plan, but they should be few and plain. They should depend as little as possible on hypothesis as to the nature and causes of morbid processes, and as much as possible on clinical and pathological facts. And they should agree in the main with those traditional names and arrangements which do not contradict ascertained facts and which are generally accepted by the best authors.

When a clinical group of symptoms is found associated with a definite anatomical change in a given organ, it is best treated along with other "diseases" of that organ. When "it has no anatomy," it is most conveniently grouped with its nearest clinical allies. When it affects several organs, it should be referred to that which is its primary or most important seat. On these principles we make a chapter of diseases of the brain, the cord, or the nervous system generally. We are not afraid to place epilepsy near cerebral hæmorrhage, asthma not far from cancer of the lung, and rheumatism with osteo-arthritis. Diabetes might be classed with diseases of nutrition, with those of the nervous system, of the liver, or of the blood; but it may be reasonably placed in the neighbourhood of renal calculus, because these affections, differing in every other respect, agree in being both recognised by symptomatic changes in the urine. Rheumatism may be considered among the fevers, among general diseases, or among diseases of the joints; but the latter is the place where it is most conveniently compared with gout, and also where a reader would be most likely to look for it.

The general arrangement in the following chapters is local and anatomical, following the organs of the body; while functional diseases are, as a rule, placed with those presenting similar symptoms.

One important exception, however, has been made, in accordance with general practice.

There is a group of "general diseases" which, though variously defined and more or less extended, has been recognised from the earliest times. The type of this group is the disease with which we shall begin, typhus fever, as it was called, when that name only meant a variety of the genus fever. With it were associated other "continued" fevers, so called to distinguish them from the intermittent fevers common in Greece and Italy.

In recent times another group of diseases has been associated with continued fevers, the eruptive fevers or exanthemata: smallpox, measles, and their allies. These were classed with diseases of the skin, not only by Willan and Bateman, but also by Hebra, although they appear separately in the title of his work.*

* Measles and Scarlatina appear in Willan's 'Order Exanthemata' with Nettlerash and Roserash, Smallpox with Scabies, and Chicken-pox with Eczema. Hebra's treatise is entitled "Acute Exantheme und Hautkrankheiten," but Measles and Scarlatina appear after the

The presence of fever is, however, no sufficient criterion in itself. It may be produced by local inflammation or it may be idiopathic, it may be continued or intermittent, accompanied by a rash or not. Since the discovery of the important part played by microphytes in disease, as described above in the chapter on Contagion, it appears probable that idiopathic fevers and irritative pyrexia—perhaps we may add, inflammation itself, or at least purulent inflammation*—are alike due to the reaction of the organism when a brood of microphytes invade the blood and tissues.

Leaving, therefore, the old term "fever" to denote, not a disease or group of diseases, but a physiological state of which pyrexia is a synonym, we may unite the continued fevers and exanthems on the basis of their being contagious, and due exclusively to the entrance of living contagia. In a few cases, as noted in the chapter on Infection, we can identify the actual contagium; and, without venturing to push the argument from analogy, we may affirm that whether the contagium be a living microphyte or not, typhus and enteric fever, measles and scarlet fever, smallpox and chickenpox are each caused by the invasion of a specific particulate contagium.

But not only do they agree in this "specific" (*i. e.* special or peculiar) ætiology, they are all "general diseases." Some, like typhus, are without any local lesion but what is demonstrably the result and not the cause of the fever; while in others, like enteric and scarlet fevers, though local lesions are constant, they are clearly parts of a general process, and not its precedent cause.

Again, these contagious and general diseases agree in running a more or less defined (specific or peculiar) course, limited in time and developing periods of incubation, invasion, ingravescence, culmination, and defervescence.

Lastly, each of these maladies confers remarkable protection from a fresh attack while they do not protect from one another.

We have here therefore the most, perhaps the only, natural and scientific group of diseases, each separate and definable, yet agreeing with one another and differing from the rest.

Have any other affections than the traditional Fevers and Exanthems a right of admission to this class?

Accepting as the best criteria those of exclusive origin by contagion and of "breeding true," we have no difficulty in admitting the typically specific disease Mumps to the list, and, with almost equal claims, Whooping-cough and epidemic Influenza. Erysipelas and Diphtheria may probably be added, but there are difficulties in both cases which will be considered hereafter.

Syphilis never arises but by direct contagion; the contagium is particulate; it breeds true; it affects not only the blood, but, as Sir William Gull well put it, body, blood, and bones; it protects against itself; its invasion is accompanied by slight, occasionally by high fever, and by a characteristic rash. It differs from other specific fevers in the length of its course and in the difficulty of separating this course from what may be called its sequelæ. It also differs in the important fact of being hereditary.

Tuberculosis is like syphilis in being hereditary, and in its variable and

introductory chapters, between Seborrhœa and Erythema. Why dermatologists did not include Typhus is not apparent. It is as much an exanthem as Measles, and Measles as much a continued fever as Typhus.

* The late Prof. Hüter, of Greifswald, proposed to define inflammation as a septic process due to the entrance of microzymes which are so widely diffused that the disease is pandemic.

long-drawn course. It is still doubtful how far it can be called contagious, it does not affect every organ, and it certainly does not protect against itself. On the other hand, a definite bacillus probably occurs in all tubercular lesions, and in well-marked cases of acute tuberculosis the rapid course and general symptoms much resemble those of a contagious fever.

Pneumonia, *i. e.* acute lobar pneumonia, has strong claims on clinical grounds to rank as a specific febrile disorder. It has its micrococcus. It invades, runs its course and subsides like a fever. Its symptoms are scarcely more dependent on the hepatised lung, as to time and severity, than are those of enterica on the typhoid ulcers. But cases of contagion and an epidemic course are both exceptional for pneumonia. It is generally believed to arise from common causes, as exposure to cold; its course, though less variable than that of syphilis or tubercle, is far from constant; it is often secondary to other diseases; and it is not self-protective.

Cholera is another doubtful case. Few would now regard it as a mere local enteritis; such a conclusion would have less foundation than Broussais' doctrine that typhoid fever was only symptomatic pyrexia from ulceration of the bowels. Few doubt that it is contagious, as it certainly is epidemic; and this conclusion is independent of the pathogenic nature of Koch's comma-bacillus; for no diseases are more certainly contagious than typhus and smallpox, yet in neither has the microphyte, if it exists, been discovered. Usually there is little or no pyrexia in cholera, but there is reason to believe that this feature is rather masked than absent.

Two epidemic febrile disorders, contagious and once terribly destructive, have happily been banished from this country—the Sweating Sickness since the sixteenth, and the Oriental Plague since the seventeenth century. The latter without question, and probably the former is, like cholera, endemic and comparatively mild in certain localities; but, when epidemic, they acquire fearfully active properties.

Three specific diseases of the brute creation are occasionally transferred to man by inoculation,—they are, Anthrax or splenic fever, Glanders and Hydrophobia. In the first the microphyte is known and its whole history investigated; it answers every test of being the true contagium of the disease (*v. supra*, p. 13), so that other maladies may be compared with this as a criterion. Glanders is almost as certainly and completely known. The pathology of Hydrophobia is in many ways obscure and it is still classed with diseases of the nervous system, but there can be no doubt that its natural place is not far from Anthrax.

Another remarkable malady, as fatal as hydrophobia, is known by the cumbrous name of "acute yellow atrophy of the liver." This has many of the characters of a specific disease, although, like cholera, it is not as a rule a fever. At present it is best left with other hepatic diseases.

While we must admit, as undoubted members of this class, some affections in which no microphyte has been found, we must exclude others, though more or less certainly bacterial in origin, because they are not "general," or run no definite course, or do not protect. Gonorrhœa and Leprosy are instances. They are more properly grouped, the former with impetigo, some forms of erysipelas and other contagious suppurations (p. 15), the latter with lupus and other infective granulomata (p. 60).

Several of the above diseases are not included in the following section,

either because their pathology is doubtful or because they are more conveniently treated with local diseases.

Rheumatism, though a general disease, and febrile, is not contagious, does not protect, and has neither an exclusive or specific origin nor a constant or specific course.

In the following list are stated in a brief and, of necessity, dogmatic form, the characters of the several diseases which entitle them more or less decisively to a place in this section.

Specific Infectious Diseases

Disease.	Contagious.	Microphyte.	Local foci.	Exanthem.	Extent.	Course.	Protection.
1. Typhus	Exclusively	None known	None	Present	Epidemic	Definite	Complete*
2. Recurrens	,,	Spirillum	,,	Absent	,,	,,	Imperfect.
3. Enterica	,,	Bacillus?	Ileum, spleen, &c.	Present	Endemic	Less definite	,,
4. Morbilli	,,	None known	Bronchi	,,	Epidemic	Definite	Complete.
5. Scarlatina	,,	,,	Throat,&c.	,,	,,	,,	Marked.
6. Rubeola	,,	,,	,,	,,	,,	,,	,,
7. Variola	,,	Micrococcus?	None	,,	,,	,,	Complete.
8. Vaccinia	,,	,,	,,	,,	Endemic	,,	Marked.
9. Varicella	,,	None known	,,	,,	Epidemic	,,	Complete
10. Mumps†	,,	,,	Parotid	Absent	,,	,,	,,
11. Pertussis‡	,,	,,	None	,,	,,	Indefinite	,,
12. Influenza‡	Probably	,,	Bronchi, &c.	,,	,,	Definite	Limited.
13. Cholera	Exclusively	Vibrio?	Intestine	Rare	Both	Definite	,,
14. Plague	,,	None known	Lymph-glands	Petechial	Epidemic	,,	Incomplete?
15. Erysipelas	,,	Micrococcus	None	Present	,,	,,	None.
16. Diphtheria	,,	Uncertain	Throat,&c.	Absent	,,	Less definite	Marked.
17. Syphilis	,,	Uncertain	Throat,&c.	Present	Both	Prolonged	Complete.
18. Tuberculosis	Probably	Bacillus	Various	Absent	Pandemic?	Indefinite	None.
19. Pneumonia‡	?	Micrococcus?	Lung	Herpes?	Sporadic or epidemic	Definite	,,
20. Glanders	Exclusively	Bacillus	Nostrils	Absent	Epizootic	,,	?
21. Anthrax	,,	,,	Spleen	,,	,,	,,	Complete.
22. Hydrophobia	,,	None known	None	,,	,,	,,	,,
23. Ague?	Miasmatic	Microzoon?	Spleen	,,	Endemic	Indefinite	None.

* *I. e.* cases of the same person having two attacks, though they certainly exist, are so extremely rare that they do not affect the practical rules as to infection.

† Described under "Affections of the Mouth and Throat," vol. ii.

‡ Described under "Affections of the Lungs," vol. i.

TYPHUS*

ἦξει Δωριακὸς πόλεμος καὶ λοιμὸς ἅμ' αὐτῷ.
Quoted by THUCYDIDES.

History, nomenclature, and geographical distribution—Incubation—Course: first week, exanthem; second week, crisis and convalescence: death and post-mortem appearances—Complications and sequelæ—Ætiology; contagion of typhus: its supposed spontaneous origin—Diagnosis—Prognosis—Treatment.

SCARCELY more than forty years have elapsed since this disease was finally separated from certain others with which it had been confounded. Yet we can trace back its history. For its prevalence as an epidemic, and the fearful mortality which always accompanies it, make it certain that many descriptions handed down by writers of former times refer to Typhus, unmixed with the other forms of fever from which it is now known to be distinct. Its place in history is next in importance to that of the true or Oriental Plague. It is the common pestilence which has accompanied and followed wars.

The name now in use was first applied to a malady, or a group of maladies, by Sauvages in 1759. Until then it had, from the time of Hippocrates downwards, been employed, in accordance with its etymology, to designate a confused state of intellect, with a tendency to stupor. Most probably the plague of Athens, recorded by Thucydides, was what we now call typhus. However this may be, there is no doubt of the nature of certain epidemic fevers which prevailed in Italy, France, and Hungary in the sixteenth century, and of which accounts were given by Fracastorius of Verona, and other contemporary writers. During the course of the same century occurred the first three of the famous "Black Assizes" in this country, when judges, sheriffs, and jurymen were suddenly attacked with fatal illness, which had spread from the prisoners brought up for trial. One of the older names of the disease is *jail-fever*. Another is *Morbus castrensis* or *military fever*, from the ravages which it has committed among soldiers and camp-followers, from the time of the Thirty Years' War and the siege of Reading in 1643 down to the Crimean campaign. Other names, again, are *spotted fever*, *brain fever*, *putrid fever*, from some of its more conspicuous symptoms. Of late years it has been universally called Typhus in this country and in France; but the Germans are obliged to term it *Typhus exanthematicus* or *Flecktyphus*, from their unfortunate habit of calling Enteric Fever *typhus*, instead of Typhus abdominalis or Ileo-typhus.

On the Continent the disease with which we are now concerned is much less common than in the British Isles. Both in Great Britain and in

* *Synonyms.*—Contagious or epidemic fever; spotted fever, febris petechialis, typhus exanthematicus; brain fever; jail fever, camp fever; fourteen-day fever. *Fr.* Fièvre typhus. *Germ.* Flecktyphus.

Ireland, it has prevailed with great severity on repeated occasions during the last two hundred years. Since the commencement of the present century there have been epidemics of Typhus in 1803, in 1817–19, in 1826–28, in 1836, in 1843, in 1846–48, in 1856, and from 1861 to 1870. It must be noted, however, that in some of the earlier of these epidemics there was a large admixture of cases of another disease, Relapsing Fever, which was not known to be distinct from typhus until 1843, but can even now be recognised by the small mortality which has always attended it.

Typhus is more or less endemic in the poor districts of Edinburgh, Glasgow, and Dublin, and was so until recent years in London. As an epidemic it has again and again left its haunts in cities and invaded the whole country. On the Continent and in the United States its course has been chiefly epidemic, and attendant on armies, especially during the miseries of sieges and of retreat. Typhus is rare even as an occasional visitant in the south of Europe, and it appears to be unknown in India and the tropics generally. It is not uncommon in Northern China.

The disease was introduced into America in 1847 by an infected immigrant ship, and in 1867 by the same means into Australia, but fortunately it has never established itself there.

Typhus is unknown among animals. Mosler injected fresh blood from patients with typhus into the veins of dogs without any result. Zülzer, however, has since been more successful; his injections were made upon rabbits, and he found that when the blood was taken while the disease was at its height those animals died in two or three days, but not if the crisis had been passed.

Entrance of the contagion.—No materies morbi has yet been detected, though there can be little doubt of its existence. It probably gains entrance in most cases by the breath, and successively invades the lymph, the blood, and the tissues through the stomata of the air-vesicles. The contagion is very sure, but readily diluted and dissipated, and probably not very persistent.

Incubation.—This is of variable length. Few cases afford an opportunity of determining it, for the disease rarely follows a single definite exposure to contagion. But Murchison collected for the 'St Thomas's Hospital Reports' in 1871 no fewer than thirty-one instances more or less directly in point. In two of them the effect was apparently immediate, and the same thing has been noticed by other observers: the patient having been conscious of an offensive odour proceeding from a case with which he had come into contact, was at once attacked with headache, prostration, nausea and rigors; and all the other symptoms developed themselves in due course. In one instance the period was not more than two days; in two, only four days; in one, from two to five and a half days; in one (that of Murchison himself) exactly five days; in one not more than six days; in two not more than ten days. Similar short incubation-periods have been given by other writers; thus Lebert, in 'Ziemssen's Handbuch,' states five to seven days; and some cases have been recorded in which the disease followed exactly eight days after exposure. On the other hand, Murchison found that in the larger number of his thirty-one cases the period was longer: in four it was exactly twelve days; in thirteen others it was within a few days of this, on one side or the other; and in four instances it was ascertained to be longer than twelve days; being in one patient not

less than thirteen days, in another not less than fourteen days, in the third exactly fifteen days, in the fourth not less than twenty-one days. The period of incubation would seem then to be very variable; all we can say is that it is a short one compared with that of many specific fevers.

During the incubation the patient appears to be well; sometimes towards its end, there is a little malaise, with headache, pains in the limbs, and loss of appetite. Lebert says that these symptoms may last several days.

Course.—The course of typhus is conveniently studied by dividing it into weekly periods.

First week.—The onset of the disease, from which the first week is reckoned, is generally definite, and sometimes sudden: more so than in enteric though less than in relapsing fever. The patient is attacked with headache and with pains in the back and limbs. He feels chilly, and sometimes shivers; after a while he may perspire, but the chilliness presently returns and he is glad to sit cowering over the fire. He feels weary and disinclined for exertion. He is thirsty, but appetite is completely lost. His tongue is œdematous, pale, and coated with fur, which is at first white, afterwards yellowish. There is nausea, but not often vomiting. The bowels are constipated. The urine is scanty, dense, and high coloured. He is restless and his sleep is disturbed by painful dreams and sudden starts. So far these are only the common early symptoms of any specific fever, and in slighter degree they attend a feverish cold. But then follow more characteristic symptoms.

Every day the patient becomes more prostrate; if unable to seek relief in bed, he totters as he walks, and his hands tremble when he attempts to use them; until, on the third or the fourth day at latest, he can support himself no longer.

From the first the patient's aspect is dull, heavy, and oppressed. The eyes are injected and suffused. The face is of a dusky colour, the flush being general, and not limited to the cheeks as in enteric fever. As the disease advances, the expression becomes more and more vacant and stupid, and it is seldom that the patient himself feels anxiety about his illness. Towards the end of the week there is generally *delirium*, especially at night. The advent of this symptom is said to be earliest in persons who have been intemperate, and in those who have been subject to mental anxiety and fatigue. In some exceptional cases it sets in during the first night, and Murchison speaks of having seen cases which were at first mistaken for mania. But, as a rule, it is only after the lapse of several days that the patient's mind begins to wander. About the same time he generally ceases to complain of headache.

The *temperature* rises rapidly in typhus. Wunderlich stated that it is generally 104°—104·9° on the first evening, and that by the fourth evening it is seldom under 104·9°, generally about 105·8°, and often higher still. But although Lebert has since observed 106·5° on the second evening, experience in England has been that the average figures are much lower than those given by Wunderlich. According to Murchison the highest temperature attained at any period of the disease is generally about 104° or 105°; it is scarcely ever as high as 106°, except in children, and it sometimes fails to reach 103°. The maximum is usually observed between the fourth and the seventh days, sometimes on the third day, and occasionally not until the second week. During the latter part of the first week, when the maximum

has been reached, the thermometer varies little. Murchison remarks that a high range of temperature in the first week generally forebodes severe cerebral symptoms in the second. Lebert observed that a rise in the evening is of comparatively little significance, so long as the thermometer falls each morning; what is really serious is a high temperature without intermission.

The *pulse* does not usually rise above 100 during the first two or three days; afterwards it ranges, as a rule, between 100 and 120. When it is much higher, the case is severe, if the patient is an adult; but in children a very rapid pulse even at the commencement of the disease is not an evil sign. Occasionally the pulse remains below 100, or even 90. Murchison cites instances observed by himself or by others, in which it was not above 40, and sometimes down to 28, for days together. In certain cases however of slow radial pulse, the heart's beats have been twice as frequent as the pulsations felt at the wrist. A slow pulse is not regarded as a favourable sign in typhus.

Respiration during the earlier days follows the pulse rather than the temperature.

The exanthem.—Typhus is attended by a characteristic *eruption*, the mulberry rash, as it was named by Sir William Jenner.

In rare cases this is preceded by a rose rash, which may almost be mistaken for the eruption of scarlet fever. In the museum of Guy's Hospital we have models, illustrating this remarkable roseola, which were taken from two women under the care of Dr Wilks in 1864. The parts represented are the abdomen, and the forearm with the hand; but in each instance the rash is said to have covered the patient. It was of a bright crimson colour, punctated, macular, or diffused. One of the women was a nurse in the hospital, so that she was under observation from the first. The roseola in each case faded before the mulberry rash came out.

The characteristic *mulberry rash* generally appears on the fourth or the fifth day of the fever; sometimes, as in a case admitted into Guy's Hospital in 1874, it may be discovered as early as the third day and sometimes as late as the sixth. Occasionally it is absent; but observations made with great care at the London Fever Hospital in 1864 showed that this occurred in scarcely more than 2½ per cent.—among nearly 2500 cases of all ages. The exceptions were mostly in children. When it does come out in them, it is often peculiarly abundant and well marked, so as to resemble the rash of measles; and in such cases it may be seen on the face, whereas in adults it is generally confined to the trunk and limbs. One usually looks for the typhus rash on the chest and the abdomen; but Dr Buchanan says that the earliest maculæ are to be found on the back of the wrists, the borders of the axillæ, and the epigastrium.

It consists of more or less numerous spots, of indeterminate form, the largest three or four lines in diameter, isolated or irregularly confluent. They may at first be slightly raised, so that one can feel them with the finger; and as they may then be of a somewhat florid colour, it is sometimes difficult, when they are few and separate, to distinguish them at this stage from the rose spots of Enterica. At this time, too, they disappear on pressure. But in the course of a day or two they alter in appearance; their hue becomes darker and more dingy; they are no longer raised; and when the finger is pressed upon them they either remain unaltered or assume a yellowish tint. This change is due to the escape of blood from the vessels into the substance

of the cutis; in other words, the maculæ have become petechial. They remain visible in the dead body if the case should end fatally while they are still present. In many instances they are from the very first of a livid or purple colour and do not fade on pressure.

Within forty-eight hours from its first appearance the mulberry rash is complete. During this time fresh spots may come out, but they are added to the old ones. There is not, as in enteric fever, a succession of crops of maculæ, one set appearing while another is fading away. Murchison satisfied himself on this point in a large number of cases, surrounding every spot with a circle of ink so as to identify it. In addition to the distinct maculæ, there is also a faint, irregular, dusky-red mottling, which looks as if it were more deeply seated, and has therefore been inaccurately called a "subcuticular rash." After the first day or two, no increase of the eruption occurs, but the maculæ persist until the end of the fever.

It is important to notice that the severity of a case of typhus is generally directly proportionate to the amount of eruption, its depth of colour, and the rapidity with which it becomes livid or petechial. Murchison adds that the exceptional cases in which no rash appears have generally a mild course; but according to Lebert they are often severe and sometimes fatal.

Second week.—This period is marked by a gradual aggravation of all the symptoms. The patient is now absolutely sleepless, and he usually passes into a state of continuous *delirium*. Sometimes he is noisy and violent, shouting, talking incessantly, singing, struggling to get out of bed, or fighting with his attendants. When spoken to, he becomes still more excited. In this condition a patient may throw himself out of the window, and some years ago a man was brought into Guy's Hospital for a suicidal wound of the neck inflicted in the course of typhus. After two or three days the delirium becomes quiet and muttering; and in the majority of cases it is so from the first. Sometimes excitement comes on as night approaches, while the rest of the day is passed in a state of stupor and prostration. By the middle of the week there is generally complete unconsciousness. When loudly spoken to, the patient perhaps opens his eyes and stares vacantly; if told to put out his tongue, he may separate his jaws, and leave them gaping, but no other signs of intelligence can be elicited.

Murchison, who himself went through two attacks of typhus, tells us that the imagination is far from inactive during the delirium of typhus. He took a great dislike to a nurse and to a valued friend; and because they once tied him down in bed, he fancied that they intended to murder him, that they were shutting him up in a dungeon, that they followed him to India, Burmah, and other countries which he had visited in former years, and to which he had escaped. Dr Gueneau de Mussey, who caught typhus in Dublin in 1843, afterwards remembered imagining that he was tied down in bed, and gradually consumed by spontaneous combustion, while women were taking water from a pond and pouring it over him. Another fancy was that he saw the front of a particular house in Paris in a state of phosphorescence, and a child suspended by the neck from a window. He also believed that he saw one of his friends killed in the street; and so strong was the impression that even during convalescence he continued to feel concern for his loss.

During the second week of typhus *deafness* is very commonly present; indeed, it begins about the fifth day. It sometimes affects one ear, sometimes both. Its cause is not yet ascertained. Stokes thought that it was

due to softening of the muscles of the ossicula; but to this view Murchison objects that the hearing is too soon recovered during convalescence. For many cases do well in which deafness has been complete; and from the time of Fracastorius there has been a tradition that it is a favourable sign. For this belief, however, there do not appear to be any good grounds; deafness is certainly present in many cases which end fatally.

The *pupils*, in the advanced stages of typhus, are contracted and insensible to light—as minute as pinholes, according to Graves. Jenner first laid stress on this myosis as a distinction between typhus and enteric fever; and Murchison says that, neither during active delirium nor in profound stupor, has he seen dilated and insensible pupils in typhus. Occasionally however when coma comes on, the pupils dilate.

The muscular *prostration*, during the second week of typhus, becomes extreme. The patient sinks down in bed, and lies on his back, unable to raise himself, or even to turn on either side. If the clothes are turned down to look for the maculæ, he takes no notice. One of the shrewd remarks of the last resident apothecary of Guy's Hospital, Mr Stocker, was that to find a fever-patient lying on his side was a good sign. The fæces are passed involuntarily. The urine dribbles away incessantly, so that unless it can be caught in a proper receptacle it soaks the sheets and produces great irritation of the skin. But before it begins to run off it may fully distend the bladder, the muscular coat of this organ being paralysed as well as the sphincter. One must, therefore, never omit to examine the hypogastric region at every visit, even if the nurse tells us that the urine is passed in abundance. Other results of muscular weakness are loss of power to speak, to protrude the tongue, and sometimes even to close the eyelids, so that the cornea may slough.

At this stage of the disease *tremor of the muscles* is almost constantly present. The whole body may be in a state of agitation, especially in old people and in those who have been exhausted by work or by intemperance; indeed, in drunkards a state of delirium tremens often seems to be added to the symptoms of the fever. Sometimes the tremor is limited to the hands and tongue. Murchison speaks of having sometimes seen nystagmus or choreiform spasms of the limbs. Much more frequent are those jerking movements of the muscles of the forearms which are commonly called *subsultus tendinum*. Or there may be twitchings of the face, one corner of the mouth being perhaps drawn up from time to time. Jenner saw two cases in which the face acquired a peculiar expression from a spasmodic action of the inferior recti muscles of the eyeballs in association with the levatores palpebrarum. In each instance the movements were excited when either of the arms was suddenly raised. Another variety of spasm is picking or fumbling at the bedclothes, for which we have the pedantic names of "floccitatio" and "carphology." In more rare cases the fingers are forcibly clenched, or the legs and thighs are bent so that the knees almost touch the chin.

The mulberry *rash* generally remains visible throughout the whole of this period. The maculæ may even become darker and more distinct, and may not disappear until the disease has already subsided. But the faint general mottling vanishes after a few days, and in mild cases, where this is the only eruption, nothing of it may be left at a time when the fever has still several days to run.

The *temperature* during the second week is, as a rule, rather lower than

in the latter part of the first week. Sometimes, indeed, the maximum is not reached until between the seventh and the tenth day. But during this period there is generally a slight fall, to which Murchison attached importance, believing that the cases in which it was absent were grave. Henceforth, and up to the time of the crisis, there are usually slight recurring remissions of one or two degrees. A decided rise during the second week indicates the supervention of some complication, generally pneumonia.

The rate of the *pulse* varies little from day to day, but its general tendency is to become more rapid. Its volume and force decline until in severe cases it is exceedingly feeble or even imperceptible. Sometimes it is dicrotic or undulatory, but Lebert remarks that this is seldom the case, whereas it is almost the rule in enteric fever. Irregularity of the pulse is not uncommon, and sometimes there are intermissions. Stokes pointed out an important sign of that progressive weakness which is the chief danger of the disease, namely, diminution or loss of the heart's impulse, with disappearance of the first sound. For several days, even when recovery is to take place, it is often impossible to feel the heart beating, and with the stethoscope only the second sound can be heard. In other cases a systolic bruit is developed, which no doubt belongs to the class of functional murmurs.

The *breathing* at this stage of the disease is almost always hurried, being at the rate of thirty or forty in the minute. According to Murchison, hypostatic congestion of the lungs should be regarded as a constant symptom rather than as a mere complication. It is, at any rate, present in all severe cases, and after death is scarcely ever wanting, whatever may have been the cause of the fatal issue. It commonly begins about the middle, but sometimes at the commencement of the second week. It is indicated by diminution of resonance over the bases behind, where the respiratory murmur is feeble, and by the presence of coarse râles, which may gradually spread over the greater part of the chest on both sides. Bronchial catarrh is always associated with this condition, and the patient may spit up considerable quantities of frothy tenacious secretion, mixed perhaps with streaks of blood. But sometimes cough and expectoration are entirely absent.

The *tongue*, in very mild cases, may remain moist and furred throughout the whole course of the fever. But usually during the second week it is dry, rough, and brown. When the disease is very severe it contracts into a ball, and becomes covered with a dark brown or black crust; this crust may be irregularly cracked; but the fissured condition of the tongue itself, which is so commonly seen in enteric fever, is rare in typhus. The lips and teeth are covered with a dirty blackish material, commonly spoken of as *sordes*. This consists of an accumulation of epithelial débris, which becomes dark from desiccation, or black from blood or from the remains of beef-tea and other food. An entire loss of appetite lasts to the end of the disease; and sometimes the patient refuses to take the nourishment which is given to him. At this stage he does not seem to suffer from thirst. Murchison met with some instances in which vomiting was a persistent and troublesome symptom, but such an occurrence is exceptional. The abdomen is sometimes flat or even concave, and tympanites is very rare. As a rule there is *constipation*, but looseness of the bowels is not infrequent. The fæces may be of normal consistence, and even if fluid, they are generally dark in colour. Murchison speaks of diarrhœa as occurring in about 5 or 10 per cent. of all cases of typhus. But in 1866 Dr Buchanan said that it had been seen in at least one third of the cases which had come under observa-

tion at the London Fever Hospital during some years previously; he was inclined to refer its greater frequency in the experience of some physicians to the larger amounts of liquid food forced upon the stomachs of their patients. Murchison remarks that when an aperient has been required the bowels often afterwards remain open or even relaxed.

The *urine* generally remains dark in colour during the second week of typhus, but sometimes it is pale and even alkaline. With regard to the amount of urea which is voided it is increased compared with the amount of nitrogenous food taken. At this period of the disease the *chlorides* fail to be excreted by the kidneys, even when the patient is made to take large quantities of common salt. The urine sometimes contains no chlorides at all, but Dr Buchanan says that usually two or three grains are passed in the twenty-four hours, just enough to produce an opalescence on the addition of nitrate of silver. *Albuminuria* is common in the febrile state generally, and in typhus appears to be present in more than half the cases.

Crisis.—Such is the condition of a patient suffering under typhus until about the fourteenth day, when, in favourable cases, a marvellous change takes place. Formerly, what was most often noticed was that he fell into a sound and quiet sleep, from which he awoke a few hours later, rational, refreshed, and cool. But at present the use of the thermometer enables us to watch the process more closely. In some exceptional cases there is a rise of perhaps two degrees the day before the crisis occurs. Much more often the temperature becomes slightly lower during the same period. The crisis itself is marked by a rapid fall to normal or even lower; a difference generally of from 4° to 6° Fahr. The fall may be complete within twelve hours, but according to Lebert it more often takes thirty-six hours and sometimes two or three days. Murchison says that in nearly one half of the cases of typhus the date of the crisis is the thirteenth or the fourteenth day; in more than three fourths it is from the thirteenth to the sixteenth. Lebert gives a much larger proportion of cases as subsiding between the tenth and the twelfth days, some even between the sixth and the ninth.

Complications may occasionally mask the crisis, or prevent its occurrence. They will be considered presently.

Crisis is sometimes accompanied by perspiration, or by diarrhœa, or by the excretion of urine depositing lithates in abundance; but there is no reason to suppose that such symptoms are essential to the defervescence, as was formerly supposed. Afterwards the temperature generally remains normal, except that for a night or two there may be a slight evening rise.

Convalescence advances rapidly. The tongue becomes clean and moist, and the appetite is ravenous. Within three or four weeks the patient often completely recovers his bodily strength and is able to return to work.

A true *relapse* of typhus is exceedingly rare. An instance of it was recorded in 1869 by Ebstein, the interval being twenty-five days. At the London Fever Hospital no such case occurred during twenty-three years after 1855. In that year Dr Buchanan had a nurse under his care who after recovering from an attack of the disease, was taken ill a week later, and went through it a second time: on each occasion there was a mulberry rash. The first fever appears to have lasted a fortnight, the second somewhat longer.

Exitus lethalis—Many cases of typhus do not run on long enough to exhibit a crisis. The mortality from the disease at the London Fever Hospital has been at the lowest 15 per cent. Murchison estimated that, if slight cases and those occurring in children be taken into account, it is about 10 per cent. It differs, however, slightly in different years, and it is said to be generally higher during an epidemic than at other times. Among soldiers in camp, or in a besieged city, the death-rate has sometimes been enormous. In the Crimea one half of the French troops who were attacked is said to have perished; and still more terrible instances are on record. In certain epidemics death has sometimes occurred on the second or the third day, or even after a few hours' illness. Such cases have been described as a special variety of the disease—*typhus siderans*, and the patient was described as *sideratus*, planet-struck. Murchison speaks of having seen several cases end fatally on the sixth or eighth day, usually from pulmonary congestion. But, as a rule, typhus does not destroy the patient until towards the end of the second week.

The exact mode of death varies. Sometimes it is by "typhoid" symptoms and coma; sometimes it is through the lungs by asphyxia; sometimes it is by failure of the heart, the pulse being imperceptible, and the skin cold, livid, and bathed in a profuse sweat. In some of the last-mentioned cases the patient lies for a day or more in a peculiar state, to which Jenner has appropriated the name *coma vigil*. He has his eyes wide open, so that he might be supposed to be awake, but he is absolutely insensible to all that goes on about him, and his face is devoid of expression. Sometimes death is preceded by a rapid elevation of temperature, as in a case at Guy's Hospital in 1873, in which the thermometer registered 108·7°.

Morbid anatomy.—The appearances observed in the bodies of those who have died from typhus are not characteristic of the disease, but are effects of the febrile state through which the patient has passed, and are found equally in cases of erysipelas, pyæmia, and other acute diseases. The *rigor mortis* is brief and incomplete, putrefaction takes place early, there is much cadaveric discolouration of the skin, the *blood* is fluid and dark coloured, and it stains the lining membrane of the heart and of the great vessels. There is often but little emaciation, yet the *muscles* are soft and friable, and when death has occurred at a late period of the fever their fibres are granular or waxy-looking under the microscope. A similar change is found in the substance of the *heart*, and probably often plays an important part in bringing about the fatal issue. The *spleen* is but little enlarged, weighing on an average seven ounces, though sometimes as much as fourteen ounces; but its tissue is very soft, and may be reduced to a mere pulp, which escapes when the capsule is divided. Jacquot is said to have observed a case in which instant death resulted from rupture of that organ. The *liver* is soft and hyperæmic: in an advanced stage of the disease it is often fatty. The *lungs* are in a state of more or less extensive hypostatic congestion. The affected parts are bulky, of a dark red or purple colour, and soft; serous fluid oozes abundantly from their cut surface, and from the opened bronchial tubes. There is, lastly, an entire absence of those intestinal lesions which belong to enteric fever.

Other changes which have been noted are quite independent of the disease. They are common either at a certain period of life, or in dissipated and intemperate persons, such as are apt to succumb to typhus. Thus the brain

has often been found atrophied, with an excess of fluid in its ventricles and upon its surface.

In other instances the *kidneys* have been wasted or in an advanced stage of Bright's disease. In such cases death is apt to be ushered in by convulsions, the occurrence of which in typhus should always lead at once to an examination of the urine; it may be then found to be albuminous, and may contain blood, more or less altered, and casts. Sometimes the renal affection appears to be quite early and recent, and probably is itself secondary to the fever. Murchison speaks of having found the kidneys weigh nineteen, twenty, or even twenty-three and a half ounces. It is to be remembered, however, that albumen may be detected in the urine of a large proportion of cases of typhus in which no cerebral symptoms occur, and which do well. It is usually in small quantities, and it is often present during only a day or two days towards the end of the disease.

Complications and *sequelæ* of typhus are neither numerous nor frequent. Jaundice is extremely rare. Murchison met with only fifteen cases in which it was present. In one of them the liver was in a state of acute yellow atrophy; and in another leucine and tyrosine were found in the viscera as well as in the urine. One well-marked instance of jaundice occurred at Guy's Hospital in 1869. The patient, a man aged forty-nine, died on the fourteenth day of the fever.

Lobar pneumonia is not very uncommon. Sometimes, when the patient appears to die of the severity of the disease, instead of there being merely hypostatic congestion of the dependent parts of the lungs, a considerable part of one of them is found to be hepatised. Either the lower or the upper lobe may be affected in this way. In other cases inflammation of the lung sets in a few days after the crisis. Of about forty consecutive fatal cases examined at Guy's Hospital, no fewer than six had well-marked pneumonia. In one instance an attack of pleurisy occurred a week after the subsidence of the fever; and after death the left side of the chest was found to contain several pints of fluid with flakes of lymph. Murchison has several times seen gangrene of the lung after typhus.

The occurrence of a convulsion is an unfavourable symptom, but even when it is repeated, an absolutely unfavourable prognosis is by no means to be given. Recovery took place in twelve among one hundred and thirty-two cases in which this complication occurred at the London Fever Hospital between 1862 and 1869. In very rare instances, where a fatal issue has occurred, blood has been found effused upon the surface of the brain; but this may have been the result of the convulsions rather than their cause. It is towards the end of the second week that convulsions are most often observed.

In very rare cases acute meningitis occurs as a complication. In addition to the cerebral symptoms which commonly accompany the fever, there is then retraction of the head, strabismus, and dilatation of the pupils. Mania sometimes sets in during convalescence, and may compel the removal of the patient to an asylum; but in the long run it seems always to end favourably. Hemiplegia, with or without aphasia, is an occasional sequela, but in all likelihood it depends upon embolism of one of the cerebral arteries by a fragment of thrombus derived from the left auricle or ventricle of the heart; for clotting of blood in the recesses of these cavities is doubtless of frequent occurrence during the height of the disease. Murchison once saw ulcerative

endocarditis in a case of typhus, with large vegetations and infarction of the spleen. Embolism of the arteries of the limbs is doubtless the cause of gangrene of the feet, which has now and then occurred as a sequel. Bedsores ought hardly to arise in this disease, since the state of coma is of comparatively short duration. Their presence sometimes accounts for the supervention of pyæmia, but in other cases blood-poisoning sets in during convalescence without any such obvious explanation. Thrombosis of the femoral veins is not often seen after typhus. Inflammatory swellings which have been called *buboes*, and compared with those that occur in the plague, occasionally form in the parotid and submaxillary regions, and go on rapidly to suppuration.

When a pregnant woman is attacked with typhus she not infrequently passes through the disease without interruption to the process of gestation, but sometimes a miscarriage occurs between the tenth and the fourteenth days. As a rule she afterwards does well, and the child, if not too immature, is generally saved.

Ætiology.—That typhus passes directly to other persons from the sick is established by the clearest possible evidence. Instances of such an occurrence are commonly observed in every hospital into which cases of typhus are admitted; if into general wards, those who are already patients with all kinds of other complaints are attacked; if into special wards, the medical attendants and nurses fall frequent victims. When cases are left in private houses or lodgings, the disease passes not only to relatives and to other inhabitants of the same dwelling, but also to doctors and clergymen, whose visits are only occasional. On the other hand, the removal of a single case of typhus from the building in which it arose is often effectual in preventing other occupants from taking it.

The diffusion of the disease can often be traced from point to point in a town or in a district. Thus Alison relates how the son of a shoemaker in Edinburgh lay ill with typhus in a room in which his father and two apprentices were at work. Afterwards both of the apprentices were attacked in their own homes, apart from one another, and at considerable distances from the workshop; and there followed seven cases of typhus in one house, and seven in the other.

An epidemic which occurred at Carlisle in 1781 was found by Dr Heysham to have started from a particular house in Richard Gate; one of the persons affected there was a weaver, who on his recovery communicated the disease to his fellow-weavers in a large workshop, and by them it was spread all over the town.

The contagion of typhus is probably exhaled both by the skin and the lungs, and it may perhaps cause the offensive odour which is so perceptible close to severe cases. This odour has been compared to the smell of rotten straw, or to that of mice. Murchison regarded it as *sui generis;* he says that he has known nurses in the London Fever Hospital distinguish typhus from other fevers by it alone. It is given off chiefly during the second week, and there is reason to believe that the contagion is less powerful before that time. The late Dr Perry, of Glasgow, maintained that the disease was not contagious before the ninth day. He found that at the Glasgow Fever Hospital patients who happened to have been sent in for erysipelas, pneumonia, bronchitis, or other inflammatory affections, escaped typhus so long as they remained in the wards for acute cases, but caught it

when they were transferred into a convalescent ward; and he adopted the plan of keeping such patients in the fever wards until they were sufficiently well to go to their homes, with the result that during several months none of them were attacked. Murchison, however, although he admits that typhus is most contagious after the first week, thinks that the spreading of the disease in convalescent wards is due to the patients coming more closely into contact there, but especially to their wearing their own clothes, saturated with the fever-poison before admission. He believes that the human body soon ceases to give off the poison after the subsidence of the fever.

It is, at any rate, certain that clothes and bedding may become vehicles (*fomites*) for the transmission of typhus. Murchison says that laundry-women are especially liable to contract the disease without direct communication with the sick. Barker and Cheyne, in their account of one of the first epidemics, relate that a child discharged from a fever hospital took to another institution a bundle of clothes which had not been disinfected; a woman who opened it perceived an exceedingly disagreeable odour, and in a few minutes became ill with what proved to be the beginning of the fever.* Very few positive instances seem to have been recorded of the communication of typhus by contact with the bodies of those who have died of it; but Murchison, when he was attacked in Edinburgh, had been dissecting in a close room, in which there were many such bodies, and he had never entered the wards of the infirmary, nor seen a case of the disease. On the other hand, at St Bartholomew's Hospital, in 1838–9, the dissecting room received seventeen bodies dead of typhus; but among six students of the hospital who alone took it, four had not dissected at all, and the other two, who had dissected, had been also exposed to contagion in the wards.

It appears that typhus is seldom conveyed by persons not themselves affected. But Murchison relates that in January, 1867, a patient in a surgical ward at the Middlesex Hospital was attacked after she had been there for four and a half months; and she had been receiving daily visits from a nurse who was in close attendance on a patient with typhus downstairs. Again in 1861, great interest was excited by the case of an Egyptian vessel, the "Shiah-Jehaad," from which typhus was introduced into Liverpool, where thirty-one persons caught it. Although Dr Duncan, on the testimony of the surgeons who attended the men on board this ship, thought that they suffered from no other disease than dysentery ('Trans. Epid. Soc.,' 1861), Dr Parkes came to the conclusion that typhus had really been prevalent among them before they arrived in port ('Army Med. Rep.,' vol. ii).

Typhus is not nearly so apt as the contagious exanthemata to be propagated by means of inanimate objects, or of human beings themselves unaffected by it. Moreover, its poison is easily rendered inert by free dilution with air. Some writers have stated that there is greater risk of the disease spreading from the lower to the upper stories of a house or of a hospital than in the reverse direction; and this, if true, would show that the upward currents of air that exist in all inhabited buildings are capable of carrying the infection with them. But experience has abundantly proved

* Haller, of Vienna, thought that dark-coloured materials were more apt to absorb the poison than light-coloured ones; I well remember, I used to notice, when demonstrator of anatomy, that the dissecting-room smell adhered to me more strongly when I wore dark clothes.—C. H. F.

But the 60th aphorism of Dr Keil, of Northampton, is that "Black cloaths *ceteris paribus* draw the least moisture of any" ('Medicina Statica,' 1720).

that it never passes from one house to another through the atmosphere. When the London Fever Hospital was one of a row of houses in Gray's Inn Lane, no case of typhus arose in the others; and afterwards, when it occupied its second site at King's Cross, on the same plot of ground as the Smallpox Hospital, and but a few yards off, Dr Tweedie was able to state that during eight years not one of the officials of the latter institution contracted typhus. So, again, Murchison says that if a patient with this disease is placed in a large, well-ventilated apartment, the attendants incur little risk, and the other residents in the same house none whatever.

Predisposing causes.—The facts stated in the last two paragraphs form links in an argument by which Murchison endeavoured to prove that typhus, instead of being always due to contagion from a previous case, is often generated *de novo* in persons placed under defective sanitary conditions, of which overcrowding is the chief.

It is true of other acute specific diseases, as well as of typhus, that many instances occur of which the origin cannot be traced; but positively to demonstrate the spontaneous origin of a contagious disease, is, from the nature of the case, almost an impossibility. Perhaps the most favourable opportunity that has ever occurred for investigating the point was afforded by the Egyptian ship, the "Shiah-Jehaad," already referred to. She left Alexandria in November, 1860, and, after calling at Malta, arrived at Liverpool on February 16th, 1861. But, according to Dr Parkes, some of the men may have brought the poison with them when they embarked at Alexandria; they numbered 476, and included not only Arabs, but also Nubians and Abyssinians. Of the supposed instances of the spontaneous generation of typhus collected by Dr Murchison, the most important, in consequence of the care and pains which he devoted to its investigation, is perhaps the group of seven cases which arose in Meridian Place, Bermondsey, in March, 1859—a time when the disease was at least very uncommon in London, for during ten and a half months previously only two examples had been seen in the Fever Hospital. But the real force of this and other similar observations lies in the fact that one single condition was traceable in every one of them, namely, that those who were attacked by the disease had been crowded and huddled together in narrow, ill-ventilated dwellings. They were often also destitute and famished; but that this is not so essential to the development of the disease as overcrowding, seems to be shown by the fact that at Dundee, in 1865, an epidemic of typhus was brought about by the inhabitants of the surrounding country flocking into the town in consequence of work being abundant and wages good. The occurrence of typhus in an epidemic form has, indeed, been predicted when, as in 1826 and in 1862, destitution and famine have been very prevalent; and such predictions have been verified by the result. It would be impossible to find a better illustration of the conditions under which, if at all, typhus is generated than was afforded by the "Shiah-Jehaad;" the men on board (who were being carried to Liverpool in order to navigate back to Egypt another vessel, a man-of-war, then in that port) seem to have been crowded together in the most shocking manner; some of them were in a state of starvation; the filth and stench between decks were abominable; and, to crown all, the hatches had been battened down on account of bad weather.

No less an authority than Virchow has endorsed the opinion that the poison of typhus can be generated by the concurrence of such conditions

as these; and the same view has been maintained by the late Dr Hudson, of Dublin. But all the facts adduced by Murchison may be brought into harmony with the opposite doctrine, namely, that the disease is invariably due to contagion from a previous case; for it is well ascertained that impairment of health increases the susceptibility of an individual to the typhous poison, and overcrowding is a powerful predisposing cause when the contagion is present.

It would seem, indeed, that everyone is originally capable of taking this disease, although the fact of having once passed through it affords an almost absolute subsequent immunity. At the London and Glasgow Fever Hospitals all the nurses who have not had the disease before contract typhus within three or four months after entering upon their duties. In 1833 Dr Tweedie stated that with one exception every physician connected with the London Fever Hospital had been attacked with typhus. Similar experience is recorded from the Fever Hospitals of Dublin, Cork, and Edinburgh.

It is sometimes possible to trace accidental conditions which favour the operation of the contagion; one is *intoxication*. Murchison says he has known several instances of persons exposed for months to the poison of typhus in its most concentrated form, who were not attacked until immediately after a debauch. Habitual intemperance probably acts in a similar way: it was once noted that more than one half of the patients admitted for this disease into the Edinburgh and Glasgow Infirmaries had led intemperate lives—but this perhaps was not conclusive evidence.

Other favouring circumstances are excessive *bodily fatigue*, *mental anxiety*, and *want of sleep*. Murchison, in support of the popular doctrine that a dread of typhus increases the risk of taking it, cites the case of an Edinburgh medical student who so feared it that he could hardly be induced to enter a ward in which there were any cases; he was one of the first students to be attacked during the epidemic of 1847. Again, the debility which accompanies convalescence from other complaints predisposes to typhus, and in armies it has been frequently observed to follow scurvy. There is no evidence that typhus is less likely to occur in phthisical persons than in others. Tweedie long ago made the remark that butchers appeared to be comparatively exempt from typhus. Most of the butchers admitted into the London Fever Hospital for the disease have been out of employment and ill-fed.

Poverty and starvation are among the most certain predisposing causes. No less than 95·76 per cent. of all the typhus cases observed at the Fever Hospital during twenty-three years, more than 18,000 in number, had been inmates of hospitals or dependent on parochial relief, and many of them had been on the verge of starvation for several weeks or months. Epidemics in Ireland, in England, and on the Continent have repeatedly been associated with the failure of crops and with the widespread destitution consequent; so that although relapsing fever is the true "famine-fever," that name was formerly given to typhus, and not without warrant.

Another, and the most important, predisposing cause of this disease is *overcrowding*. Beside its obvious influence in increasing the spread of typhus by contagion, there seems to be no doubt that it also augments the susceptibility of individuals to the poison. Thus it is quite possible that it may favour the occurrence of a first case among the occupants of a lodging-house or cellar, without generating typhus directly.

Other so-called predisposing causes probably act only by facilitating contagion. Thus epidemics occur rather in the winter than in the summer because the poor for the sake of warmth block up every hole by which air can enter their wretched dwellings during the cold season. But in some years it has happened that more cases have occurred in July, or in September, than in January. So far as is known, the variations of temperature which occur in temperate climates have no direct influence upon the prevalence of the disease. It is doubtful whether this form of fever has ever been observed in Africa, or in the tropical parts of America, although it was believed by some observers to occur in the gaols of India. If hot countries should prove to be exempt, it will not be possible to attribute the fact to any idiosyncrasy of the inhabitants, like that of negroes in respect of ague. For Africans and East Indians have been admitted into the Fever Hospital with characteristic symptoms of typhus.

It is worthy of notice that sometimes patients themselves attribute the disease to their having "caught cold" or got wet. Murchison says that this has been the case with a considerable proportion of the persons admitted into the London Fever Hospital. In 1856 a young man was taken into Guy's Hospital who gave as the history of his illness that he went for a long walk on damp ground, and felt that he took cold; two days later he had headache and fever, and the typhus eruption followed in due course. In that instance the poison had no doubt already been received, and the relation between the chill and the typhus was one of mere coincidence.

Age and sex.—Typhus may occur in persons at all ages, from one month to eighty-four years old. The quinquennial period at which the disease is most common is from fifteen to twenty; one-half of the cases admitted into the London Fever Hospital have been in patients from ten to thirty.

The proportion of males to females among typhus patients scarcely differs from that in the population generally.

Protection.—A second attack of typhus is as rare as one of smallpox; far more so than a second attack of measles or scarlet fever. It does, however, sometimes occur, and there was a notable instance of it in the case of Murchison himself, the interval between the attacks being ten years. He knew of two other physicians who contracted this form of fever twice, and in one of them it proved fatal on the second occasion. A very remarkable fact, for which he vouches and of which he saw at least six examples, is that, during an epidemic, a person exposed to the poison may have what appears to be an abortive attack of typhus, with fever, dry tongue, and even slight delirium, but no distinct rash; may recover, sometimes after exactly fourteen days, and then a few weeks later may go through a regular attack attended with the characteristic eruption. Such a "*typhisation à petite dose*," in fact, seems to afford no protection whatever.

Diagnosis.—This is seldom, if ever, doubtful when one has an opportunity of watching a case throughout its whole course; but there may be great difficulty in forming an opinion upon a single visit, or if the patient is brought to a hospital a day or two before his death.

(*a*) At the commencement of the disease, what suggests the correct view of its nature is generally the fact that there has been exposure to the specific contagion, or at least that cases have already occurred in the neighbourhood. Otherwise it could at first scarcely be distinguished from

smallpox and the other exanthemata, and not at all from enteric or relapsing fever, some cases of which begin exactly in the same way. As a rule all uncertainty is removed by the development of the eruption, but as a rare exception this may be absent, and sometimes, especially in children, it is by no means easy to say whether the rash is that of measles or of typhus. Lebert makes a point of the harassing cough which belongs to measles, whereas the bronchitis of fever is seldom troublesome. Certain cases which are commonly classed under purpura might also be mistaken for typhus; they are attended with eruptions which rapidly become petechial, and with much febrile disturbance; and they no doubt depend upon blood-poisoning, the exact nature of which, however, is not yet understood. Lastly, the copaiba rash has been mistaken for typhus, as in an instance quoted by Dr Hudson.

(*b*) At the end of the first week the diagnosis of typhus from *Enteric fever* is rarely a matter of serious difficulty, even in children. Apart from evidence of dates and the dissimilar exanthems, the one is epidemic and rapid in its evolution, while the other is sporadic and gradual in its onset and course. The condition of the abdomen and of the pupils is also a help.

Pneumonia, although formerly often mistaken for typhus, and although Murchison says he has seen many instances sent to the London Fever Hospital, can always be discovered by auscultation, which no careful physician would omit before giving a diagnosis of typhus in a patient without eruption. But it may still be a question whether pulmonary inflammation is or is not a primary disease. Murchison says that those cases of pneumonia in which the apex of the lung is first affected are particularly apt to be unattended with local symptoms, and to assume the mask of a "typhoid" condition.

The distinction between *meningitis* and typhus is one of far greater difficulty. Among the symptoms which point to the former disease are an anxious and pained expression of face, intense headache, especially when concurrent with delirium (for in fever pain almost always ceases before delirium sets in), the presence of convulsions early in the case, and the repeated occurrence of vomiting. The patient, moreover, is irritable rather than apathetic, and resents instead of enduring examination.

(*c*) Several further questions arise in the diagnosis of cases which come under observation at a time when the patient is comatose, with "typhoid" symptoms fully developed. Pyæmia may be mistaken for typhus under such circumstances; and if there be erysipelas, jaundice, or delirium tremens, it may be difficult to say whether these diseases are complications of fever or independent. In persons advanced in years, mere bronchitis is sometimes attended with a dry brown tongue, stupor, and other "typhoid" symptoms.

But the condition which is most apt to be mistaken for typhus at this stage is *uræmia*, dependent upon chronic disease of the kidneys. Murchison says that patients have often been sent to the London Fever Hospital whose symptoms were all of renal origin; and at Guy's Hospital in former years the doubts with regard to such cases have sometimes never been entirely cleared up. Conversely, in 1865, a man who was in a surgical ward for stricture became feverish and drowsy, and his illness was attributed to ascending nephritis, until a mulberry rash was discovered upon him; he died, and the kidneys were found to be healthy. It must be borne in mind that the existence of a chronic renal affection, when established by *post-mortem*

examination, affords no proof that typhus was not also present. According to Murchison, however, the thermometer generally affords a sufficient means of diagnosis; the temperature is at or below the normal in cases of Bright's disease, unless complicated with an acute inflammation.

Prognosis.—It is important to remember that the prognosis in typhus does not merely depend upon the severity of the symptoms. The most important consideration is the *age* of the patient. The older he is, the greater is the danger. In children it seems never to be fatal except by some com plication. Murchison found that at the London Fever Hospital (the average typhous death-rate for all ages being from 15 to 19 per cent.) the rate in persons above thirty was 35·39 per cent., in those above forty 43·48 per cent., in those above fifty 53·87 per cent., in those above sixty 67·04 per cent.

Again, typhus is likely to terminate fatally in persons who have been intemperate, who have been exhausted by fatigue of body or mind; or who have been suffering from privation of food. The greater frequency with which these various conditions occur among adult males, as compared with females, is probably the reason why the mortality is higher in them; among children between five and fifteen it is lower in boys than in girls. Murchison remarks that a presentiment of death is a very unfavourable, but not necessarily a fatal, indication. Patients who have gone on struggling against the disease during the first few days often become rapidly prostrate and die. The sooner they are put to bed the better their chance.

With regard to the prognostic significance of particular symptoms, the danger is best measured by the intensity of the cerebral disturbance and by the degree of prostration; the earlier the date at which severe symptoms are present the worse is the prospect. Sleeplessness continued throughout several days is of evil omen. So is the occurrence of convulsions. A very abundant rash is usually a bad sign, especially if the spots are dark and rapidly become petechial. A pulse over 120 is always a serious matter in an adult; and when it exceeds 150 death is almost certain to occur. Relaxation of the sphincters before the tenth day is unfavourable; but towards the end of the second week it is not uncommon in severe cases, which may nevertheless do well. Great lividity of the face and limbs and coldness of the distal parts of the body with profuse sweating are among the most threatening symptoms. A well-marked "typhoid" state, however, with dry, brown tongue, sordes, and subsultus is observed in many patients who recover. Indeed, even in what appear to be the worst cases, one must never give up hope until the last.

Treatment.—We as yet have no means of arresting the course of typhus, or of bringing it to a uniformly favourable termination; but a great deal may be done to increase the chance of a patient's recovery, and many of the most painful and distressing symptoms may be alleviated or checked. Murchison cites observations made both at Philadelphia and at Belfast, from which it appears that the death-rate from this disease was greater in proportion to the duration of the fever before admission into hospital.*

* It must not be supposed that neglect of medical assistance was the sole, even if it was the principal cause of the varying mortality in question; in many instances a fatal issue was doubtless the direct result of removing the patient at an advanced stage of the illness, whereas at an earlier period it might have done him no injury. Murchison says he has repeatedly known patients die from exhaustion after being carried for several miles in a shaking vehicle. But that, with proper precautions, a moderate journey may be effected

When typhus attacks a person in comfortable circumstances he may be safely left at home, provided that his apartment is spacious and well-ventilated, and that he can be properly nursed. But those who are poor should at once be carried to a hospital. The establishment of special institutions for the reception of fever cases dates from the commencement of the present century; but so recently as 1842 many London physicians, among whom was Richard Bright, were in favour of mixing such cases with others in general wards, rather than of setting apart special wards for them, or of collecting them in buildings devoted to that particular purpose. However, so far as typhus is concerned (and the same may be said of relapsing fever) this was certainly a mistake. For (1) it has now been proved by experience that if a sufficient cubic space of 2000 feet be allowed, and if ventilation be well attended to, the death-rate is not greater among typhus cases accumulated in the same ward than when they are scattered. (2) In every hospital in London where such cases have been placed in general wards there have been terrible examples of the spread of fever to other patients. To cite but a single instance: in 1862 one or two cases of typhus were admitted into one of the large medical wards in Guy's Hospital; seven other patients took the disease, and five of them died. What makes the argument stronger is that in a large proportion of medical cases, particularly in those of Bright's disease and of diabetes, the risk of a fatal termination, if typhus should be caught, is very much greater than among healthy persons. Moreover, the free ventilation, which is the chief condition of safety under such circumstances, is directly prejudicial to persons suffering from pulmonary complaints. It would be far safer to admit smallpox cases into a general ward than those of typhus, because protection against the former disease could easily be given by a general vaccination. (3) As far as medical men and nurses are concerned, the danger of the extension of typhus may be greatly reduced by retaining for as long a period as possible the services of those who have once passed through the disease, and by invariably selecting persons under thirty for all vacancies that may arise.

Every large town ought to have a fever hospital, capable of enlargement during an epidemic by the erection of temporary buildings; and all general hospitals should have one or more separate fever wards.

A patient suffering from typhus should be placed upon a spring bed, or upon a hair mattress, with not too many bedclothes. The temperature of the ward or room should not exceed 60°. After the first few days he should not be allowed to get up even to pass his excreta. It is important that his nurse should be strong enough to raise him when necessary, and instructed enough not to lift him into a sitting posture. From time to time he should be turned over to one side or the other, and kept from rolling back by pillows.

Food.—A nourishing diet is of the highest importance. Dr Graves, of Dublin, desired, as his own future epitaph, the testimony, "He fed fevers." So long as the patient can be tempted with food it is important to vary it as much as possible. Beside milk, eggs, and beef-tea, he may have broth made of mutton, veal, or chicken, meat-juices and extracts, jellies, custards, blancmange of isinglass or ground rice, white wine-whey, &c.; and vermicelli may be given in the beef-tea, small pieces of bread or toast in the broth, and arrowroot in the milk.

without risk appears from the fact that, among patients admitted into the London Fever Hospital from the immediate neighbourhood, the death-rate during five years was almost exactly the same as among those brought from the distant parish of St George's-in-the-East.

For beverages we have the choice of barley-water, toast-and-water, lemonade, tamarind-water, currant-water, effervescing drinks, and cold weak tea. Before many days have passed, however, he acquires a distaste for everything but cold water. The fever patient should drink often, in order to keep up free excretion by the kidneys.

Food must be pressed upon him and administered at regular intervals of from one to three hours; to give it more often than this is, according to Murchison, injurious rather than useful. If he be drowsy he should be roused for the purpose, and this can generally be done without seriously disturbing him; but if he should be sleeping quietly after having been very restless, the interval may sometimes be a little lengthened. It is, however, of great consequence that his strength should not be allowed to run down at night, or during the early morning, when the risk of exhaustion is always greatest. When, as is sometimes the case, he clenches his teeth, and obstinately refuses food, it must either be introduced into the stomach by means of a tube passed through the nostril, or, better, by closing one nostril and pouring the liquid nourishment through the other by means of a short funnel; an aural speculum answers the purpose well. If necessary, nutrient enemata or suppositories must be resorted to, and when there is obstinate vomiting there is no other plan open to us.

Stimulants.—The administration of alcohol is a very important part of the treatment. All observers are agreed that alcohol seldom need be given to children, that it is almost always required for patients over forty, and that persons whose habits have been intemperate must have it earlier and in larger quantities than others. The state of the heart affords the best indication; the necessity for stimulants is greater in proportion as the cardiac impulse is feeble, the first sound low, and the radial pulse rapid, compressible, or, above all, irregular. The propriety of continuing to give alcohol may often be determined by its effect on the heart's action. If the pulse becomes quicker than before, it is likely to do harm; if slower, it may be expected to do good. It is most needed during the second week; very seldom before the appearance of the mulberry rash. Low muttering delirium is often controlled by stimulants; a dry, brown tongue becomes moist, and other "typhoid" symptoms become less marked. On the other hand, severe throbbing headache and violent maniacal excitement are often aggravated. A burning dry skin is so far a reason for withholding alcohol; profuse perspiration, especially if the limbs are cold, calls for an increased supply. As a rule it should not be given when the urine, although scanty, is of low specific gravity, or when there is suppression of urine.

Probably there is no direct advantage in employing one liquor rather than another. Some recommend, on various grounds, brandy or whisky or rum, others port wine; Buchanan speaks highly of beer. The amount must vary with the urgency of the symptoms. Murchison thought that it was very rarely necessary to give more than eight ounces of brandy in the twenty-four hours, but some physicians order as much as twenty ounces. Of port wine as much as a bottle may be taken with advantage by patients who are desperately ill. But only small doses should be allowed at a time, repeated very frequently, at intervals of an hour or two; and when the daily quantity is large, the fact that milk mixes with brandy much better than with wine is a good reason for preferring it. When there is great prostration, with cold sweats, hot brandy or whisky-punch or hot wine-whey is recommended by

Murchison. At Guy's Hospital the house-physicians often inject stimulants under the skin in such cases, a practice suggested by Zülzer. Twenty or thirty drops of brandy may be administered in this manner. Ether, carbonate of ammonia, camphor, or musk may also be given by the mouth.

Treatment of symptoms.—The *pyrexia* itself seldom requires direct treatment in typhus. Probably, when the temperature rises high, cold affusion or the wet pack is better than cold baths.

Quinine in large doses of ten or twenty grains, although it lowers the temperature for a time, does no real good. Indeed, the experience of Christison in Edinburgh, of Peacock in London, and of Haller in Vienna was that quinine is positively injurious; and this was the final conclusion of the medical officers of the French army in the Crimean epidemic. Other antipyretic drugs—salicylates, thallin, antipyrin, antifebrin, are probably unsafe from their depressing action on the heart. This question will be again considered under Enteric Fever.

Murchison's favourite medicine at the London Fever Hospital was dilute hydrochloric acid, in doses of twenty minims, with a little syrup and tincture of orange. He often "observed the tongue become moist, and a marked improvement follow the commencement of the acid treatment, at whatever stage it was tried." When there is insatiable thirst, he recommends a very weak infusion of cascarilla or quassia.

The early *headache*, which is often a very distressing symptom, may sometimes be checked by an emetic. When the patient is young and robust, there is no objection to applying three or four leeches to the temples, and they frequently give complete and permanent relief. Or the hair may be cut, and the head be covered with a bag of ice, or a tubular cap through which cold water is running. In other cases a blister to the forehead is of service; or hot fomentations, as recommended by Graves, may be employed, especially in old or debilitated subjects. But in many instances it is advisable to prescribe opium for the relief of headache, particularly when it is accompanied by an inability to sleep.

Murchison says that *sleeplessness* at any stage of typhus, if it continues for two or three nights, is of itself sufficient to kill; and that whenever it lasts for thirty-six hours it should be combated by medicine. During the first week one may almost always give fifteen minims of Battley's liquor opii or a quarter of a grain of morphia at night, following this, if necessary, by another half dose two hours later. But at more advanced periods of the disease there is often danger in prescribing these drugs. When the patient is in a state of maniacal delirium, Murchison recommends chloral hydrate in a dose of twenty grains, which may be once repeated should the first draught not succeed. If this treatment fails, recourse must be had to opium, with which antimony may be usefully combined, as suggested by Graves. The chloral hydrate may also be given in some cases in which the delirium is low and muttering or like that of alcoholic origin; but when the heart's action is feeble or irregular, it does harm. Murchison then preferred opium as a sedative in conjunction with tincture of digitalis and sulphuric ether. Paraldehyde in half-drachm doses may be prescribed with the same object. Other useful sedative drugs are bromide of potassium, Indian hemp, and hyoscyamus. Each must be given in full doses to do any good.

When profound *stupor* appears to threaten the patient's life, Murchison advised that a cupful of strong coffee should be given every three or four hours. He also thought it important to use dry cupping to the loins, and

to apply mustard poultices, or flannels wrung out of hot water and covered with mackintosh, especially when the urine was scanty and albuminous. He believed that he had saved some cases by blistering the scalp with a piece of lint soaked in strong liquor ammoniæ, and applied for five or six minutes under oiled silk. Should high fever accompany coma, cold affusion to the head may sometimes be employed with great advantage.

For the *pulmonary complications* of typhus, ammonia is the chief remedy. But in some cases turpentine is said to be even more decidedly effectual; it is given in doses of fifteen minims every three hours. Mustard poultices should also be applied to the back and sides of the chest.

For *inflammatory swellings*, usually in the parotid region, both Murchison and Buchanan recommend the application of a blister at an early stage, for it sometimes appears to prevent the occurrence of suppuration. When an incision is required, it should be made early.

Convalescence.—After the subsidence of the pyrexia, the patient should be kept upon a restricted diet for the first two days; but upon the third day, if the tongue be clean, he may have a little boiled fish or chicken or the lean part of a chop. If wine or brandy has been given, beer should be substituted while the case is still under treatment, so that there may be no excuse for carrying on the habit of tippling when health is restored.

When pyrexia has disappeared, the patient, as a rule, recovers his strength as well as his appetite with rapidity. Relapses are excessively rare; neither A. P. Stewart, Sir William Jenner, nor Murchison met with a single case. Sequelæ, as deafness and phlegmasia dolens, are almost equally uncommon—in striking contrast to their frequency after enterica.

Moreover, while a tedious convalescence often leaves the typhoid patient weaker for years afterwards, typhus, like acute pneumonia, leaves no harm behind it. Hence the joys of returning strength are unalloyed by misgivings for the future. The contest with this disease is severe and often perilous, but it is neither protracted nor indecisive. *Horæ momento cita mors venit aut victoria lœta.*

RELAPSING FEVER

Λιμόν τε καὶ λοιμὸν γενέσθαι.—HERODOTUS.

History, nomenclature, and distribution—Incubation, onset and course—Relapse—Convalescence—Death and morbid anatomy—The spirillum—Famine as a predisposing cause—Diagnosis—Prognosis—Treatment.

TYPHUS, as described in the preceding chapter, is only the residue of the larger group of typhous "continued" fevers. The most important advance made by a long series of researches during the first half of the present century was the differentiation of enteric fever. At the same period a third fever also was discovered which is generally known as Relapsing or Recurrent Fever,* from its regularly consisting of two or more attacks separated by intervals of apyrexia.

According to Murchison, the distinction between this affection and typhus was first drawn in Ireland in 1826, when they both prevailed at the same time; but there is evidence that it also formed part of the epidemic of 1817–19, and of several earlier Irish epidemics, as far back as one recorded by Rutty as having occurred in 1739. Being far less fatal, and of shorter duration than typhus, it was in 1826 regarded as a mild variety of that disease. But in the year 1842, when it next appeared, Dr Henderson, of Edinburgh, brought forward good grounds for believing that it was a distinct fever and due to a different poison. The same view was strongly upheld by Jenner in 1849–51. The arguments used by these writers were chiefly three. 1. That the symptoms and course of the two diseases were different. 2. That even when they prevailed together in the same town, one could never be traced to infection from the other. 3. That neither of them afforded protection against a subsequent attack of the other. To these arguments may now be added (4) the fact that in relapsing fever the blood always contains a microphyte, which is absent in typhus; so that there is now no doubt of the specific and independent character of relapsing fever.

Since the publication of Henderson's and Jenner's papers, it has been ascertained that the same person often has relapsing fever more than once, so that we could not expect this disease to protect against typhus. But it has still been thought that typhus protects from famine fever. Dr J. C. Steele (then of Glasgow, now for many years our esteemed Superintendent at Guy's Hospital) remarked that, in the epidemic of 1848, persons who had previously suffered from typhus were not attacked by relapsing fever; and the same thing is said to have been noticed recently in epidemics at St Petersburg and at Breslau. Dr Henderson, however, related six cases in which typhus occurred first and relapsing fever afterwards; and Murchison says that of thirty-one persons who contracted relapsing fever in the London

* *Synonyms.*—Febris recurrens, typhus recurrens. Bilious remittent fever, seven-day fever, famine fever.. *Fr.* Fièvre à rechutes. *Germ.* Rückfallstyphus, Hungertyphus.

Fever Hospital in 1868–69 no fewer than thirteen were known to have had typhus.

At the beginning of the great epidemic of 1847–48, and also in 1851, it was particularly noticed, both in London and in other large towns of Great Britain, that most of the patients were destitute Irish, many of whom had recently left their own country; afterwards the English or the Scotch were attacked. Whether in 1842 the disease was derived from Ireland is not clear. The latest epidemic, that of 1868–71, seems to have had its origin in the east of Europe. There is no doubt that in 1847 relapsing fever had prevailed in Upper Silesia. With this exception it was unknown on the Continent of Europe, until in 1863 it was observed in Odessa, and in 1864 at St Petersburg. Next, in 1867, it broke out for the second time in Silesia, and in the following year it prevailed in Berlin and in other German towns, as well as in Breslau. In 1868 it reappeared in England. The first case, that of a woman of Irish birth who had been for several years in London, was brought into the Fever Hospital on the 4th of July. Four days later, another case occurred in the person of a Polish Jewess, who lived near to the Irishwoman. Three weeks later there arrived from the street in which the Jewess lodged a girl who had lived all her life in London. The only other persons who are known to have been attacked in London that year were eight German Jews; they all were admitted into the German Hospital at Dalston. But in October, a severe outbreak occurred at Tredegar in South Wales. In the autumn of 1869 relapsing fever became epidemic in several of the large cities of England; in London the disease reached its height in December of that year, and then gradually declined until June, 1871, when it finally disappeared. In Breslau it was again epidemic in 1872–73. In September, 1869, it appeared at Philadelphia, and in November at New York. The patients were chiefly poor Irish or Germans, and it seems to be almost certain that the contagion was imported from Europe, although its origin could not be discovered. On two or three former occasions it had been introduced into the very same cities by Irish emigrants, but it never showed any tendency to spread among the American population.

With regard to the occurrence of relapsing fever in Africa and in Asia, information is imperfect. Griesinger observed it in Egypt in 1851.* There is reason to believe that it has repeatedly prevailed in India, and the well-marked epidemic of 1877 at Bombay was carefully studied by Dr Vandyke Carter ('Med.-Chir. Trans.,' vol. lxi).

Incubation.—The period of incubation for this disease appears to vary; Murchison says that it sometimes breaks out immediately after exposure to contagion, sometimes not until fourteen days have elapsed. The usual period observed in this country has been four to ten days; in Silesia it was longer —a fortnight to three weeks. During the interval, no symptoms whatever are present; only in some very exceptional cases is there malaise.

Onset and course.—The attack generally begins with remarkable suddenness. The patient, while engaged as usual during the day, or perhaps on first waking in the morning, is seized with chilliness or with rigors; his head aches, he feels giddy, he has pains in the back and limbs, and his skin becomes burning hot. He grows rapidly worse and at once takes to bed.

* He saw a fever in Egypt which he called Bilious Remittent, and afterwards visiting London saw cases of undoubted Relapsing Fever and identified the two diseases, but it is not certain that he was correct.

One of the distinctions from typhus is that it is giddiness rather than prostration which prevents his keeping about. He is sometimes able to walk to the hospital two or three days after the seizure.

The thermometer indicates a very rapid rise of temperature. This begins even before the rigor, and within twelve or twenty-four hours it reaches 104° or 106°. The pulse, too, becomes quickened much earlier than in typhus; it is scarcely ever found below 110, and not infrequently it reaches 140 on the second day. The respiration is not quickened in proportion to the pulse and temperature. The tongue is moist and covered with a white or yellowish fur. There is great thirst. Appetite is generally wanting, but sometimes it remains good, or even voracious, the patient being able to take solid food without any harm resulting. In other cases, however, there is much nausea, and even vomiting. Pain, with tenderness on pressure, is often present in the epigastric and hypochondriac regions. By percussion increased dulness of the liver may be detected, and the spleen usually becomes decidedly enlarged, so that its edge reaches far below the costal cartilages. Jaundice sometimes occurs on or after the third day; in some epidemics it has been observed in one of every four or five cases, but generally not oftener than in one of eleven or twelve. It is now and then very bright, and the urine may be loaded with bile pigment; but the fæces appear always to remain dark. The face is flushed and the eyes are injected, but the countenance is not usually dusky, nor is the expression stupid and confused as in typhus.

As a rule, there is no exanthem; but several observers have noticed a roseola in certain exceptional cases, and in the epidemic in Silesia of 1857 a rose-rash on the second or third day was the rule. Murchison, who met with this in eight out of about six hundred cases, says that the rash consisted, of small spots, or of a reddish mottling, sometimes resembling measles, but more often undistinguishable from typhus at an early stage; yet always disappearing under pressure, and fading after a few hours or within three or four days at the latest. It came out sometimes during the first attack, sometimes in the relapse; and either as early as the third day, or immediately before the crisis. Petechiæ are not uncommonly present; no doubt fleabites have frequently been taken for them, but Murchison says that large numbers may come out after the patient's admission into hospital. The surface of the body often remains very dry, but sometimes on the second or third day there is profuse sweating which may last for hours without relieving the patient. Sudamina may be developed in large numbers, and are probably the cause of desquamation of cuticle which frequently occurs later on.

When relapsing fever occurs in a pregnant woman, gestation is almost invariably brought to an end, whatever may be its period; but it is curious that the abortion or miscarriage is not seldom delayed until the relapse. The child, if not stillborn, survives only a few hours. Here, again, there is a marked contrast with what occurs in typhus.

In the regular course of the disease, the patient's condition undergoes but little alteration for about a week. The temperature and the pulse remain high, though oscillating upwards and downwards a little, and the pulse-rate sometimes reaches 150, 160, or even 180. The patient continues to suffer from severe headache, often of a throbbing character, and from intense pains in the muscles and joints, much increased by movement. Murchison remarks that relapsing fever is altogether attended with far more

suffering than typhus; persons who have passed through both diseases invariably look back upon the former as the worse. Moreover, sleeplessness is almost always a marked symptom, and the mind generally remains clear although there may be a little delirium towards the end of the attack.

Crisis.—On the fifth or the seventh day as a rule the fever suddenly subsides. Sometimes, however, the crisis takes place as early as the third day, sometimes not until the tenth. Just before it, the fever often reaches its *acme*, touching a point higher than at any previous time; there may even be a rapid rise through as many as 4°. In some exceptional instances maniacal delirium suddenly comes on at this time; the patient screams and struggles violently, and passes his evacuations under him, but within a quarter of an hour becomes calm again and has no recollection of what has occurred. In other cases the crisis is said to be ushered in by epistaxis, diarrhœa, or the appearance of the catamenia. But the characteristic sign of the crisis is profuse sweating. The change is wonderfully complete. In the course of a few hours the temperature becomes normal, or even lower than normal, having fallen probably 8° or 10° without a break; Murchison cites one case in which there was a range of 13° in six hours, and another in which the difference amounted to 14·4° in twelve hours. At the same time, the pulse drops from 120 or more to about 70, the tongue becomes clean, and the patient loses all his pains. Lebert remarks that the period from evening to morning is that at which the crisis is most apt to occur. Litten has seen it sometimes preceded by a *pseudo-crisis*, the temperature falling below normal, but within twenty-four hours rising to as high a point as before, again to fall on the following day.

Interval.—During the *interval* which follows, the patient feels perfectly well. At first he is rather languid and exhausted; but his appetite is good, he quickly regains his strength, he gets up and walks about, and sometimes insists upon leaving the hospital and returning to work. His temperature for two or three days is almost always lower than in health; but afterwards it becomes normal. On the other hand, the pulse is at first rather above the natural standard; but at a later period it is often remarkably infrequent, perhaps not more than 44 or 50. Murchison remarks that in such cases it may rise to over 100 when the patient assumes the erect position.

Relapse.—In exceptional cases this apparent convalescence is in reality the end of the disease. But, as a rule, it is abruptly terminated, when it has lasted seven days, by a sudden reappearance of all the symptoms which had belonged to the first attack. Sir Robert Christison related a striking anecdote in reference to his colleague, Dr Hughes Bennett, who took relapsing fever at the commencement of the epidemic of 1843, when its characters were known only by the older members of the profession. "When he had detailed his case," being then supposed to be convalescent, "I told him he had sustained an attack of my old acquaintance . . . whose face I had not seen for a good many years; that he was not yet done with it, and that he would have another attack, commencing with rigor, on the fourteenth day" (of the disease). "Dr Bennett, surprised—I will not say incredulous,—replied that the relapse had no time to lose, as there were only three or four hours of the fourteenth day to run. It did, indeed, lose no time, for I must have scarcely reached home from his house before the rigor set in with violence." Sometimes, however, instead of occurring on the seventh day of the interval, the relapse begins on the second, or the third,

or the fifth day; sometimes it is postponed for several days, and even as late as the twenty-fifth day. Lebert says that it usually sets in at night.

This second attack is generally shorter than the first one, lasting three days, or not more than one or two; but sometimes it is prolonged to five days, and even to seven or eight. In the details of their symptoms and in their severity the two attacks may resemble one another exactly, but in some patients the relapse is the worse, especially when the original attack was very mild; in others it is far less severe, and it may be so slight as almost to escape notice. When well marked, it ends in a sudden crisis, just as before.

Recovery.—The patient now generally passes on to a permanent recovery, but sometimes there is a third attack, which begins between the twenty-first and the twenty-fourth days of the disease; it is almost always mild, and seldom lasts more than forty-eight hours. There may even be a fourth or a fifth attack.

Convalescence after relapsing fever is slow, as compared with that after typhus. The patient is a long time in regaining his strength. Notwithstanding that the duration of the disease up to the second crisis is only about eighteen days, he is seldom able to return to work within six weeks.

Sequelæ.—Relapsing fever sometimes leaves traces behind. Severe articular pains often continue during convalescence, and effusion into the knee- or ankle-joint has now and then been seen.

Another affection, which sometimes does not set in for several days, weeks, or even months after the subsidence of the fever, is a form of ophthalmia. This is described as beginning with an amaurotic stage, in which the retina, or perhaps the choroid, is alone attacked; afterwards, in what is termed the second or inflammatory stage, when the more superficial structures are involved, there is intense pain in and around the eye. It fortunately is rare for both eyes to be affected, for recovery is always tedious, and there is sometimes permanent loss of vision.

Protection.—As already remarked, relapsing fever seems to confer little or no immunity from subsequent attacks. All writers record instances in which persons have taken the disease two, or even three, times in the same epidemic.

Fatal event.—It is only in exceptional cases that relapsing fever proves fatal. Murchison states that in this country the mortality has been 4·03 per cent. In Bombay, however, Dr Vandyke Carter estimated it at 10 per cent. In persons advanced in years the disease is decidedly more dangerous than in younger persons.

Death occurs sometimes by sudden collapse. At and after the crisis the pulse is often weak, small, or irregular, and there may be temporary impairment of the first sound of the heart and of its impulse. These symptoms usually soon disappear, but the liability to their occurrence probably affords some explanation of the fact that a patient who may have had a mild attack, and who may have appeared to be doing perfectly well, is now and then found pulseless, cold, livid, and unconscious, and dies in a few hours. This sometimes takes place at about the period of the first crisis, but sometimes during the interval. In three such cases, observed by Murchison, the heart was found fatty and dilated. Another mode of death is by suppression of urine leading to convulsions and other cerebral symptoms. In some instances of this kind, recorded by Dr Henderson as far back as 1843, Dr Maclagan

discovered urea not only in the blood, but also in fluid from the cerebral ventricles. It is probable that in these cases the kidneys were diseased and that albuminuria was present during life; but the urine may contain albumen, and even blood, in relapsing fever, without any serious consequences resulting. Again, there is a variety of the disease attended with typhoid symptoms, and invariably accompanied by jaundice, which often terminates fatally; this Griesinger described as a distinct "bilious" form of fever. Among his symptoms are hæmorrhages into the skin and from various mucous surfaces, including that of the stomach, so that the vomited matters may be black, like those of yellow fever. The interval is imperfectly marked, the pyrexia running on more or less continuously from the first attack to the second. As, however, Heydenreich, in St Petersburg, and Carter, at Bombay, have each shown that the spirillum was present in cases of this kind, there is no doubt that they belong to relapsing fever.

Anatomy.—The *spleen*, if death occurs during the attack, is found to be more or less soft and much enlarged, more so than in either typhus or enteric fever. Küttner is said to have found this organ four and a half pounds in weight. It sometimes contains embolic wedges. In the one case which came to an autopsy at Guy's Hospital during the epidemic of 1869–71, the spleen, which weighed fifteen ounces, had a remarkable appearance. It contained a number of small yellow softened patches or abscesses, the largest being of the size of a horse-bean, which seemed to follow the branchings of minute veins. Dr Moxon could discover no thrombi in any of the vessels, even with the aid of the microscope. Very similar appearances were observed by Litten, at Breslau, in the epidemic of 1872–73. The *liver* is also increased in size. As a rule, the *kidneys* are gorged and swollen, and the epithelium in the renal tubules is in a state of cloudy swelling.

In some cases relapsing fever proves fatal through some secondary complication. Thus pneumonia has, in certain epidemics, been rather frequent, as, for example, at Breslau in 1872–73. Lebert says that it was generally double, that it occurred chiefly in those who had been intemperate, and that it sometimes set in during the attack, sometimes in the relapse. It has been known to lead to pulmonary gangrene. Bronchitis is commonly present, but is seldom severe. In St Petersburg hæmorrhagic pachymeningitis is said to have been observed. The spleen has sometimes ruptured, discharging a quantity of blood into the peritoneal cavity, and causing death by rapid collapse. In other instances an embolic block in this organ has broken down and set up fatal peritonitis or pleurisy. Another cause of peritonitis may be dysentery, which has been a frequent and a very dangerous complication to some epidemics. The occurrence of abortion is sometimes fatal by profuse hæmorrhage, or by rapid sinking. Inflammatory swellings or "buboes" in the parotid regions or in the groins are said to have been often the cause of death at St Petersburg, but in England they appear to have been more frequent in cases which recovered.

The pathogenic microbe.—Relapsing fever is remarkable as the first specific disease in which the contagion was discovered and its pathogenic significance demonstrated. This microzyme is morphologically a vibrio (p. 14),

and has been named *Spirillum Obermeieri*,* after the late Dr Obermeier, of Berlin, who first described them in 1873, although he had observed them as far back as 1868. It seems doubtful whether the form found in the blood of relapsing fever is identical with that which had previously been observed in water containing vegetable matters (*Spirochæta plicatilis*, Ehr., Cohn) and in the saliva of healthy persons (*S. denticola*). Dr Vandyke Carter says that the dimensions of an organism of this kind which he found in the water of a tank at Bombay were much larger than those taken from his fever patients ('Med.-Chir. Trans.,' vol. lxi).

Its ordinary appearance is that of a delicate, homogeneous, spirally-twisted filament; its length is from $\frac{1}{2000}$ to $\frac{1}{600}$ of an inch, or equal to from one and a half to six times the diameter of a red blood-disc (*circa* 15—40 μ). It is never still, and its motion is compounded of a rotation on the long axis, a progression forwards or backwards, and a lashing movement, which lengthens it out and causes the twist for a time almost to disappear.

All observers admit that the presence of the spirilla is coincident with the attacks of relapsing fever, and that during the interval or intervals it cannot be detected. Birch-Hirschfeld, however, once found it for two days after the second crisis, and several writers, including Litten, have stated that it is seldom to be seen during the first two or three days of the disease.

Heydenreich, of St Petersburg, who has made what seems to have been a careful series of investigations, declares that while it appears before the thermometer begins to rise, it ceases to be discoverable before the commencement of the crisis. He has specially inquired into the influence of heat upon this organism outside the body, and he finds that it remains alive, as evidenced by the persistence of active movements, much longer when it is kept at a temperature of 60° or 70° F. than at blood-heat, whereas at fever temperatures it dies more quickly still. His hypothesis accordingly is that the pyrexia, which is itself caused by the existence of this organism in the blood, proves directly fatal to it. In all probability the filament breaks up into a number of minute granules, and some of them may perhaps constitute the germs from which fresh crops of spirilla are afterwards developed. Heydenreich has occasionally seen some of the filaments beset with granules so as to resemble a necklace, and the late Dr T. R. Lewis, of Calcutta, speaks of having once noticed a beaded appearance. If the germs remain alive in the blood throughout the whole of the interval, they must possess the attributes of *Dauersporen*, for the spirochæta itself dies at the temperature of the blood in about twenty hours.

Even during a single attack, Heydenreich finds that there are extraordinary fluctuations in the numbers of these microphytes from day to day. He therefore supposes that successive generations are more or less constantly produced throughout the fever. Sometimes after they have been present for two or three days, they suddenly cease to be discoverable; but a few hours later they reappear in greater or less abundance. Their recognition in the blood, at the end of an interval, has repeatedly enabled him to foretell the approach of a relapse, which, however, he admits may be so slight as to be scarcely noticeable. On the other hand, Dr Carter says

* Others refer it to the genus *Spirochæta*; for Ehrenberg in 1833 distinguished two genera of *schizomycetes* under these names, the difference between them being that *Spirochæta* possesses greater flexibility than *Spirillum*. This distinction is preserved in the nomenclature of Cohn and Flügge, but the two "genera" are closely allied and the names are applied almost indifferently to the microbe discovered by Obermeier.

that at Bombay he sometimes detected the spirochætæ at periodic dates, when there was absolutely no rise of temperature; while in other cases a one-day febrile attack would occur at the proper time for a relapse, without the blood containing the organism. This observer remarks that the numbers of them must often amount to hundreds of millions, dozens being seen in the field of the microscope at the same time. It even seems likely that they may become aggregated together into dense masses with blood-corpuscles, and seriously interfere with the circulation. In fatal cases they cease to be discoverable after death. None of the solid organs have hitherto been found to contain them. Heydenreich could not detect them in the urine, nor in the conjunctival secretion, in the fluid from the pleura, the intestine, or the bronchial tubes.

Spirochæta (*v. Spirillum*) *Obermeieri* has been successfully cultivated out of the body, but for a long time all attempts to reproduce the disease failed. Injections of blood infected with it into the circulation of dogs, rabbits, or guinea-pigs were made by Obermeier himself without result, and the same negative result followed inoculation of sheep. Nor did he find that the disease was propagated by the penetration of minute quantities of blood from patients with relapsing fever into scratches upon the hands of healthy persons. Dr Carter has since recorded the fact that a few days before he himself was attacked he had scratched his finger in making an autopsy upon a fatal case. Finally he succeeded in inoculating monkeys with the spirillum taken from the blood of patients suffering from relapsing fever. The organisms were found after death in the viscera of the animals experimented upon ('Lancet,' 1879 and 1880). This completed the chain of evidence, and we may henceforward regard this vibrionic fission-fungus as the contagium vivum, and the sole cause of relapsing fever, according to the criteria laid down on p. 13.

Mode of spreading.—Direct contagion from the sick is the chief mode of propagation of this disease, and it is exceedingly apt to pass from hospital patients to nurses and clinical clerks. In 1843 the post of house-physician in a fever hospital at Edinburgh had to be filled six times in five months, owing to successive incumbents being attacked. In the London Fever Hospital, during the years 1869 and 1870, twenty-seven of the nurses and officers, and five patients admitted for other diseases, contracted relapsing fever. In 1870 a nurse from the Fever Hospital was transferred to St Mark's Hospital for fistula; and about the same time a wardmaid from St Mark's paid a visit to the Fever Hospital. These two women both fell ill at St Mark's, and gave relapsing fever to five other persons there. Two striking instances of direct contagion were recorded by Mr Reid, of Glasgow, in 1843. (1) At Dalmarnock Colliery there was a large building consisting of three stories, which was entered by separate stairs and contained forty families; some Irish people brought relapsing fever with them into a single apartment on the uppermost flat, whereupon it spread from room to room, and ultimately descending a stair, attacked twenty-two individuals in the space of two months. (2) Into a house of two apartments, in which eleven human beings were lodged, a person from a neighbouring village introduced the fever; every person there fell ill, but all the occupants of the next house, separated only by a brick partition, escaped, although they were almost as closely crowded together.

That the disease may be transported to a distance by infected clothes seems to have been proved by two cases recorded by Dr Parry, of Phila-

delphia, in 1870; and in 1843 it was noticed in Edinburgh that a large number of laundry-women contracted relapsing fever, although they had no other communication with the sick than washing their clothes and bedding.

Litten has related how a mason, who himself remained well, but who slept in a street in Breslau in which the disease was prevailing, carried the contagion to his mother, who lived where no case had before appeared.

The facts of this Breslau epidemic of 1872–3, which are to be found in the 'Deutsches Archiv' for 1874, strongly support the conclusion derived from experiment, that the spirillum (or its spores) is the sole and constant agent in spreading the disease. In all probability they are given off either with the breath or from the skin, and are received into the air-vesicles with the inhaled air. Litten has clearly shown that at Breslau they were not conveyed in drinking-water.

Predisposing cause.—The cases observed by Litten in Silesia seem to have belonged to the poorest classes, with the exception of the medical men who took the fever in the hospital; and English writers have generally regarded as the cardinal fact in the ætiology of this disease its occurrence in those who have been starving. It is the *famine fever* of Ireland. Murchison cites instance after instance to prove that those who have suffered from it in England and in Scotland have, with certain exceptions, been in a state of extreme destitution. When it occurred in Silesia in 1847, the inhabitants, in consequence of a succession of bad harvests, had been reduced to subsist on clover, grass, mushrooms, and the roots of trees. Carter states that it was brought to Bombay in 1877 by the peasantry flocking into the city from famine-stricken districts. History shows that as Typhus is the pestilence of war, of camps, and of sieges, so Relapsing fever is the pestilence of famine.* Relief of extreme destitution in districts where relapsing fever was prevailing has repeatedly been followed by the subsidence of the epidemic. Starvation renders persons more susceptible to the contagion, either by weakening the power of the living cells of the body to cope with the invading microbes or by favouring the development of the latter more directly.

Age and sex.—Relapsing fever may attack persons of all ages, from five months to seventy-five years, but the majority of cases are between the ages of fifteen and twenty-five. There are more male than female patients, the reason probably being that more men than women are beggars, hawkers, and vagrants.

Diagnosis.—It is only at the commencement of relapsing fever that its diagnosis in ordinary cases presents any difficulty. Murchison remarks that during the first two days one may be scarcely able to distinguish it from smallpox; but in consequence of the frequency with which it prevails epidemically at the same time with typhus, the early differentiation of those two

* Murchison believed that the specific poison of the disease might be generated *de novo* in the human body as the result of inanition. He cited the observations of Holland and of Donovan to prove that persons in a state of starvation suffer from delirium, stupor, and coma; that they may have a quick pulse, a dry tongue, flushing of the face, intolerance of light, and neuralgic pains over the body, and that they exhale putrid odours from the skin. He believed it to be a point in favour of such a view that tramps have often been found to bring the disease with them into London after sleeping by the roadside under hedges. Dr Carter also says that one of the earliest cases he saw in Bombay was that of a destitute lad who, while tramping up from the famine district of Poonah, was seized with fever in the upland country, and was barely able to crawl into the hospital on his arrival at Bombay. But it is obvious that in none of these instances could the reception of the disease by infection be positively disproved.

diseases, so unlike one another in the danger which they entail, is important in practice. How useful for diagnostic purposes the detection of the spirochætæ may be, has been shown by Dr Carter at Bombay, where there was often difficulty in distinguishing the effects of malaria from the more continued varieties of relapsing fever, corresponding with the "bilious typhoid" of German writers. This observer states that so multiform were the phases and degrees of the spirillum fever that about 25 per cent. of his cases could properly be termed irregular.

Prognosis.—Most patients attacked by relapsing fever recover. It is far less fatal than typhus or enterica. Out of 441 cases collected by Murchison from the records of the London Fever Hospital only 11 proved fatal; and in the great Scottish epidemic of 1843, of 6300 cases only 260 died. On a basis of over 14,000 cases he estimates the mortality at less than 5 per cent. As usual in epidemics, the worst cases come first and the disease becomes gradually milder. Age is the most serious adverse circumstance, as in typhus and enteric fever. Hæmorrhage, a petechial rash, delirium and scanty urine are the most grave symptoms.

Treatment.—This is not so successful as might be hoped, for we have as yet no means of preventing the relapse. With this object quinine and arsenic have been employed both in England and in Germany in vain. At Breslau carbolic acid, and in America the sulphites, the hypo-sulphites, and the preparations of chlorine have been used with no better result. It remains to be seen whether the salicylate of soda, or the sulphocarbolates, or perchloride of mercury, or some other of the parasiticides of antiseptic surgery may be more effectual. According to Murchison patients often find great comfort from frequent sponging of the surface with cold or tepid water, and from cold affusion on the head. An emetic at the first onset of the disease affords much relief to the severe pains in the hypochondriac regions. Throughout the febrile attacks castor-oil, or some other mild aperient, should be given when necessary. Murchison also recommended the systematic administration of diuretics, by which he believed that it was often possible to prevent one of the chief dangers of the disease, the supervention of uræmic poisoning. He directed that from one to two drachms of nitrate of potass, one drachm of dilute nitric acid, and half a drachm of tincture of digitalis should be taken in divided doses in the twenty-four hours. He allowed rather a liberal supply of food, and to persons beyond middle age he administered stimulants about the period of the crisis, especially when he suspected that the heart was weak. He often found it necessary to give opium for the relief of headache, or of the muscular and arthritic pains which cause so much distress, but states that the hydrate of chloral proved a useful substitute. Lebert recommends frictions with a liniment of chloroform and oil for alleviation of the pains in the limbs.

On account of the contagiousness of relapsing fever, it is important that patients should be taken to a hospital as early as possible. They should be placed together in wards set apart for the purpose, since Litten seems to have shown that the accumulation of a large number of cases together neither augments the mortality of the disease nor increases to an appreciable extent the danger of its spreading to medical attendants and nurses.

ENTERIC FEVER*

Ac dum prima lues udo sublapsa veneno
Pertentat sensus, atque ossibus implicat ignem
Necdum animus toto percepit pectore flammam.
VIRGIL.

History of the recognition of Enteric Fever and of its distinction from Typhus—Its extent—Nomenclature—Incubation—Clinical course and symptoms—Recovery—Abortive cases—Fatal cases—Complications—Sequelæ—Relapses—Protection—Diagnosis—Morbid anatomy—Ætiology—A contagious disease, always produced from a previous case—Methods of contagion: by air, water, &c.—Predisposing causes: soil-water, climate, age—Prognosis—Treatment—Food, stimulants, drugs—Cold baths and antipyretics—Treatment of other symptoms—Convalescence—FEBRICULA.

ALMOST at the beginning of the present century, the study of morbid anatomy in Paris was rewarded by the discovery that in the bodies of those who died of fever in that city the intestines were inflamed and ulcerated. The true bearings of such observations were not, however, at once fully appreciated. Broussais and others maintained that a "gastro-entérite" was the essential cause of "continued fever." Presently the suggestion was made by Serres and Petit that the intestinal lesions were specific, and resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of smallpox. Bretonneau, of Tours, noticed in 1826–29 that they were specially localised in the solitary and the agminated lymph-follicles of the intestine. He endeavoured to introduce the name "dothienentérie" (δοθιήν, a pustule, and ἔντερον, intestine) for the fever in which they occurred. This clumsy term was used by his pupil Trousseau, but it never met with general acceptance. Unfortunately, much greater success attended a proposal made by Louis, in 1829, to designate it "fièvre typhoïde." This proposal was, at least in England, universally adopted; and it greatly impeded a right understanding of the matter. Up to that time, all the French physicians who had written upon the intestinal affection had assumed that the same lesion would be found in every form of the disease known by the various names of typhus, putrid fever, bilious fever, gastric fever, jail fever, &c. But when the morbid anatomy of patients who had died of fever came to be investigated in Scotland and in England, the bowels were often found to be healthy. For a time the difference was supposed to be accidental, or of little consequence. But gradually it became apparent that two distinct diseases had been confused together. The credit of indicating the clinical distinctions between them is divided among a number of observers; Gerhard, of Philadelphia (1836), Lombard, of Geneva (1836), Shattuck, of Boston (1839), and particularly

* *Synonyms.*—Typhoid Fever, Gastric, Pythogenic, Infantile remittent Fever, Slow nervous Fever of Huxham (1739), Fall Fever (U.S.A.).—*Fr.* Fièvre typhoïde. *Germ.* Typhus abdominalis or Ileo-typhus.

A. P. Stewart, of London (1840), deserve mention.* Louis, in the second edition of his work, published in 1841, expressly announced that his "fièvre typhoïde" and the typhus of English writers were very different. There were, however, many who maintained the opposite view, that the two forms were identical. The ultimate issue of the controversy still appeared uncertain when, in 1849–51, Jenner published a series of researches, based upon observations made at the London Fever Hospital. The peculiar value of his papers lay in the proofs which he brought forward of the fact that the different forms of fever owe their origin to distinct specific causes. During two or three years he investigated with great care every instance in which more than one patient was brought from the same house; and he found that the later cases invariably corresponded in character with the first one, even where both kinds of fever were prevalent in London at the time. All subsequent writers, whose opinions carry weight, have adopted the doctrine for which Jenner contended, and there no longer remains any doubt about the matter.†

Soon after Jenner's researches were published, Dr Austin Flint, in 1852, showed that the same distinction between typhus and enteric fever was manifest from a comparison of clinical and anatomical facts in the United States, thus confirming the views of Gerhard in Philadelphia, and of Shattuck in Boston.

It has naturally been a point of interest to inquire whether the presence of enteric fever can be recognised in the description given by medical writers in former centuries: and the answer is decidedly in the affirmative.‡ In John Hunter's Museum there still are two preparations showing the characteristic intestinal lesions; and a well-marked case was dissected by Morgagni. It appears, too, that the disease is identical with the "slow, nervous fever" which Gilchrist, of Dumfries, described in 1734, and which Huxham, in 1738, distinguished from "putrid malignant" fever. In works of a still earlier date it must probably be looked for among the "remittent fevers," attributed to the same cause as that of ague. The mistake of confounding enteric fever with malarial fevers was commonly committed in the marshy districts of England up to a very recent period; and in India they are even now separated with difficulty. These facts would in themselves suggest that in former times, when the marsh-miasm was much more widely spread than at present, the distinction would fail to be recognised; and instances in point are probably to be found in the writings of Baglivi (1696) and Lancisi (1718), who recorded cases of fever at Rome in which the intestines were ulcerated, but which they identified with the "hemitritæus," *i. e.* semitertian, a form of ague mentioned by Galen.

Even at the present time a supposed compound disease, due to both malarial and typhoid poison, is generally recognised in America, and by some physicians in India, and has been named typho-malarial fever.

* Dr Stewart, from his comparison of the fever seen in the wards and deadhouse at Glasgow with that seen in the wards and deadhouses of Paris (1836—1839), concluded that the differences between the two diseases were "so marked as to defy misconception, and to enable the observer to form with the utmost precision the diagnosis of the nature of the disease and the lesions to be revealed by dissection."

† Dr Wilks ('Guy's Hosp. Rep.,' 1855 and 1856), Dr Peacock ('Med. Times and Gaz.,' xiii, 1856), Sir Thomas Watson ('Lectures,' 4th ed., 1857), and Dr Murchison ('Continued Fevers of Great Britain,' 1862), were among the earliest who adopted Jenner's conclusion.

‡ An interesting example of the possibility of interpreting historical records by the light of modern science is afforded by an admirable essay in which Dr Norman Moore has satisfactorily proved, from the memoranda of Dr Mayerne, physician to James I, that the disease of which Henry, Prince of Wales, died in 1612, was enteric fever.

Enteric fever is endemic in all parts of Europe and Asia, in the United States, and in Australia. It is common in the tropics, and, though less prevalent, is far from infrequent in Scotland, Norway, and Iceland. In fact, it may be called pandemic in its distribution.

It is doubtful whether enteric fever occurs in more or less modified forms in any species of animal. Mr Bland Sutton communicated to the Pathological Society ('Trans.,' 1885 and 1889) a paper on the occurrence of a disease in monkeys and in beavers, which on anatomical grounds he identified with the typhoid fever of man. The symptoms observed during life were diarrhœa and hæmorrhage. He quoted M. Serres, who recorded an epidemic of what he regarded as enteric fever among the monkeys in the Musée d'Histoire Naturelle in Paris; the symptoms observed were diarrhœa, increased frequency of pulse, and fever, ending almost always in death.

Nomenclature.—Louis' name of "typhoid fever" is an unfortunate term, since the object in giving a title to a disease really distinct from typhus, but liable to be mistaken for it, is to mark the difference between them, rather than the resemblance. Moreover, the same word has long been fitly used to designate a group of symptoms, consisting of stupor with muttering delirium and a dry, brown tongue, which are really *typhoid*, since they are like what is seen in typhus. The two meanings lead to much confusion. The term *enteric fever* or *Enterica* (sc. *febris*) appears to be very suitably substituted, as indicating the fact that the disease is attended with intestinal lesions—though not always with intestinal symptoms.* It has been adopted in the 'Nomenclature of Diseases,' published by the Royal College of Physicians, and is ordered to be used in the official returns made to the Registrar-General.

Incubation.—This period appears to vary within somewhat wide limits. The date at which a patient receives the poison can seldom be directly fixed. At Guildford, in 1867, an epidemic was traced to the fact that contaminated water was supplied on a single day, the 17th of August; a large number of cases came under medical observation on the 3rd and the 4th of September, so that, allowance being made for the gradual development of the symptoms, the incubation probably lasted twelve or fourteen days. On the other hand, instances are recorded in which the disease has broken out within four or five days after the patient has been exposed to sewer gas, or after drinking contaminated milk, or arriving in an infected locality. With regard to cases of apparently much longer incubation there is great theoretical difficulty. Persons sometimes have not fallen ill until three weeks or longer after leaving a place in which there is reason to believe that they must have taken the fever; but the question is at what date the intestinal lesions began to develop themselves in such cases. It has been conjectured that the incubation is shorter when the poison is inhaled with the breath, longer when it is swallowed in drinking-water.

Course.—The beginning of enteric fever is generally slow and insidious. The patient feels depressed and weary, more and more so every day; he has headache, and giddiness, and pains in his back and limbs; he loses appetite, and sometimes vomits. Diarrhœa comes on of itself; or, if he takes an aperient, his bowels remain relaxed. After five or six days he becomes so ill that he

* Murchison proposed as an alternative name, "pythogenic fever;" but this was never accepted, and it involves an erroneous ætiological theory.

has to give up work and take to his bed. In such a case the duration of his illness must be reckoned from the day on which he first felt ailing. There are, however, some cases in which marked symptoms set in with chilliness, or even a rigor, so that medical assistance is at once sent for. If, then, on inquiry, it appears that the patient has been unwell for a few days previously, the disease is said to have had a *prodromal stage*. It is clear that we ought to count the whole duration of such cases; for the lesions are often found after death to be further advanced than had been expected from the clinical history. It may even happen that malaise and other slight symptoms run on for two or three weeks, or longer still, and that afterwards a definite illness begins, the subsequent course of which is not shorter than usual. These instances create a difficulty from which the late Dr Irvine proposed to escape by assuming that the definite attack is in reality a relapse, and preceded by an almost latent primary fever. In every case of this kind, when the patient was ailing before he fell seriously ill, one must be prepared in case of death to find some of Peyer's patches in a state corresponding with an advanced stage of the disease. But it is probable that some of these cases begin as ordinary diarrhœa, and that the disordered state of the bowels renders them more obnoxious to the specific contagion.

In some very exceptional cases, enteric fever sets in with sudden maniacal delirium, so that the patient's removal to a lunatic asylum may appear to be necessary until the real nature of his case is manifested.

According to Wunderlich, the *temperature* during the first three or four days rises in a zigzag fashion: from morning to evening there is an ascent of about 3° F., from evening to morning a fall of about 1°; so that each evening the thermometer stands at about 2° higher than on the evening before; and he declared this course to be in itself diagnostic; enteric fever might generally be excluded if the temperature rose during the first two days to 104°; if (at least in a patient under middle age) it did not rise between the fourth and the sixth day to 103·1°; if it stood at the same level on two successive mornings or on two successive evenings; or if it was ever lower than at the same hour on the previous day. He admitted, indeed, that this stage of the disease comparatively seldom comes under medical observation, but he seems to have overlooked the fact that the exceptional cases in which its onset is marked by definite symptoms are almost the only ones in which the thermometer is likely to be used during the first few days, so that they afford no proof that similar results would be obtained in cases that begin in the usual insidious manner. Jenner remarked to the Clinical Society in 1875 that in private practice there were found to be numerous exceptions to one of Wunderlich's rules, and this certainly accords with the writer's more limited experience in the matter. In 1879 a patient in Guy's Hospital, convalescent from pleuritic effusion, was attacked with enteric fever. He first felt unwell on April 2nd, and his temperature immediately rose from 98·6° to 102·6°; next day he had rigors, on April 4th there were characteristic stools, on April 8th rose-spots appeared. In 1878 a girl was admitted who had been carefully watched from the third day of her illness by Dr Ingoldby; the morning and evening temperatures were on that day 103·4° and 105·2° respectively, on the fourth day they were 103·4° and 104·2°, on the fifth day 103·4° and 102·3°, on the sixth 103·5° and 104·2°.

As the disease advances, the patient's state becomes gradually more and

more serious. He grows weaker from day to day, his mental faculties are more obscured, and he manifests less concern about his own condition. Yet the maximum temperature to be attained in the whole course of the fever is often registered by the fourth, the sixth, or the eighth evening. From that time the thermometer indicates only trifling variations during the next ten or fourteen days, and in the morning it generally stands one or two degrees lower than in the evening. The skin may either be dry or moist. Profuse sweats are by no means infrequent, especially at night; and are sometimes accompanied by an abundant eruption of sudamina, especially towards the end of the second and in the third week, and this again leads to desquamation of the cuticle of the chest and abdomen, the importance of which lies in the fact that its cause may be misunderstood.

The *pulse* is not always very rapid; its beats are generally from 100 to 110 a minute, but sometimes they remain at the normal rate, or are even less frequent. Murchison lays stress on the liability to variations in the pulse-rate at different periods of the day, sometimes in correspondence with the fluctuations of the temperature, sometimes independently. Changes of posture also affect it much more than in health; but the most important peculiarity of the pulse is its soft, compressible character, with a dicrotism which may be so marked that an unskilled observer may actually count it at twice the frequency of the heart's beats. These features are well shown by the sphygmograph.

The *breathing* is more rapid than in health. In many cases bronchitis is present from an early period, but otherwise the respirations are not quickened in the same ratio as the pulse until hypostatic congestion begins to set in.

The *aspect* of a patient with enteric fever is in general that of languor and weariness; the face is not dusky nor is the aspect so stupid as in typhus. There is usually pallor, with a circumscribed pink flush on one or both cheeks, especially towards evening or when food or stimulant has been recently given.

The *tongue*, even at first, while it is covered with a white fur, has commonly a bright red tip and edges. In the course of the second week it often becomes clean, and its whole surface may then be of the same vivid colour and smooth, as though it had been glazed. It sometimes remains moist, but in severe cases it gradually becomes very dry; and deep painful transverse fissures often form in it.

Anorexia and thirst are common to this and to almost every other febrile complaint; nor is much stress to be laid upon nausea and vomiting, although they are often complained of at the commencement of the illness.

The *spleen* becomes enlarged in the course of the first week, and by the end of the second week it is often twice its normal size. Sometimes its edge can be felt below the ribs, but in most cases its extent can only be mapped out by percussion. Even this method of detecting enlargement of the spleen often fails, so that as a negative symptom it is of little clinical value. With resonant lungs and a full tumid abdomen, the organ may be many ounces heavier than natural, without there being any appreciable percussion-dulness over it.

Epistaxis is of rather frequent occurrence, especially soon after the commencement of the disease. It has been known to be so profuse as to destroy life.

Exanthem.—At the end of the first week or early in the second there

appears the *rose-rash*, which is the one symptom of enteric fever that is almost if not quite pathognomonic. It is sometimes preceded by a diffused scarlatina-like eruption which comes out two or three days sooner, and when attended with a slight sore-throat this has led to a mistaken diagnosis. The rose-rash itself is generally, of all rashes, the least conspicuous; to an untrained observer it seems absurd to attach significance to the presence of small red spots hardly larger than pin's-heads, the whole number of which may not exceed a dozen. Yet, when well-marked, they are practically conclusive of the presence of enteric fever. No doubt it is often impossible to say of individual papules whether they are true rose-spots or what Murchison terms "ordinary pimples;" but besides their disappearance under pressure, their colour and the absence of the signs of inflammation of a sebaceous gland, we depend upon their short course and the eruption of a new crop of similar spots. The regions in which they are most often looked for are the lower part of the chest and the front and sides of the abdomen; but they also occur on the back, although a careful physician will seldom be curious on this point. In an epidemic among the French troops at Montpellier (1870–71) Dr George Turner observed that in several cases the rose-spots could only be found on the lumbar regions, and Murchison has seen them limited to the back. In exceptional cases they are scattered thickly over the whole of the trunk, and even upon the face and the limbs. The date at which they first appear is usually between the seventh and the twelfth day, sometimes as early as the fifth or sixth day from the commencement of symptoms, sometimes not before the fourteenth. Liebermeister speaks with hesitation as to their ever being absent throughout the whole course of the disease; but it is certain that, in children especially, one fails to observe them in many cases in which careful search is made every day. They can not only be seen but felt, as rounded, smooth, solid papules; their colour, which is pink or rose-red, disappears beneath the pressure of the finger; they never become petechial; after death they are invisible. In some exceptional cases it is said that their summits are vesicular; they appear in successive crops, so that although the duration of the rash as a whole may be from one to three weeks, each individual spot never remains for more than three, four, or five days, sometimes not longer even than twenty-four hours. It has sometimes been noticed that they have come out in large numbers after a warm bath, or that they have first shown themselves on a part that had been reddened by mustard.

Diarrhœa.—The occurrence of diarrhœa is of considerable diagnostic significance. Sometimes, indeed, it is not present at any period of the disease. The proportion of such cases probably varies in different places and in different years; but Murchison, as the result of twelve years' experience, placed it at one fifth. In many other instances the bowels cease to be relaxed when the patient has been ill for a few days, or they first are loose during the third or the fourth week. Louis stated, and subsequent experience has confirmed it, that, as a rule, the severity of enteric fever is proportionate to the urgency and persistence of the diarrhœa. But it is to be observed that there is no necessary relation between the symptom in question and the extent of the peculiar intestinal lesions. It was thought by Addison that diarrhœa was more constantly present when the colon was ulcerated than when the morbid change was confined to the glands of the ileum, and the same fact was observed by the late Mr Busk at the Dreadnought Hospital; but the rule is certainly not without exceptions. The number of the

evacuations is generally about four to six in the twenty-four hours; sometimes much more. The stools have an appearance which has been compared with that of pea-soup, and which, although not uncommon in other intestinal affections, is probably seen in no specific fever but this. Their colour is yellowish, almost like that of yellow ochre. Addison used to lay stress upon their being almost identical with the normal contents of the small intestine, and he supposed that they were hurried on through the colon and rectum, and discharged without having undergone the usual changes there. Albumen is present in considerable quantity in typhoid stools. The reaction is alkaline, and there are numerous crystals of ammonio-magnesian phosphate. Blood may often be recognised microscopically or in the form of small clots, even when the patient has been ill less than a week, so that ulceration is not likely to have begun. Abundant intestinal hæmorrhage is a serious complication, to be afterwards described. In doubtful cases Dr John Harley recommends searching during the third week for fragments of sloughs from Peyer's patches. He advises that the stools should be strained through a net, and that the matters caught upon it should be washed, and then examined by a lens; flocculent shreds may thus be obtained, in which the remains of intestinal follicles can be plainly recognised. In 1873 a man died in Guy's Hospital, who two days before his death passed a slough an inch and a half long, in which muscular fibres were discerned; at the autopsy the corresponding ulcer was found to be two inches in length, and the peritoneum was exposed in its floor.

Another sign of intestinal disturbance is, in some cases, the production on gentle pressure (which must be applied with extreme caution) of a gurgling sound in the right iliac fossa. There may also be more or less tenderness in this region, and the patient may complain of pain there or in other parts of the abdomen.

During the second week of the disease the bowels generally become distended with gas, so that the abdomen assumes a rounded form. Sometimes the distension is extreme. This tympanites or "meteorism" is a grave symptom. Murchison says that the colon is the chief seat of the accumulation, and that the bulging is therefore at the sides rather than in front.

The *urine* is at first scanty and high coloured, and may remain so throughout the whole course of the disease, but after a time it may become copious, pale, and of a low specific gravity. Towards the end of the third week it often contains albumen in small, and sometimes in large, quantity; and in exceptional cases there may be hæmaturia.

Cerebral symptoms are almost absent in some mild cases of enteric fever. In the course of the second week the headache subsides, and the patient may seem to have but little the matter with him. There is, indeed, no doubt that before the thermometer came into use persons were often wrongly allowed, and even persuaded, to leave their beds with the disease still upon them. There is not always much muscular prostration. Murchison says that forty-four out of one hundred patients under his care were always able to sit up, and to get out to the night-chair; but this should never be allowed, however apparently mild the attack.

In the third week, however, in most cases, and in many much earlier, the patient becomes altogether helpless, and lies upon his back, unable even to turn over. His hands and tongue are tremulous, and occasionally he cannot even put out his tongue. Jenner believes that a disproportionate intensity of *tremor*, as compared with other nervous symptoms, is of signifi-

cance, pointing to the presence of deep ulceration of the intestine, such as is likely to lead to grave perforation or to dangerous hæmorrhage; and Murchison was of the same opinion. In some rare cases the limbs or the trunk become rigid, or the back of the neck is retracted, or there is strabismus, or spasm of the pharynx, or trismus. Murchison's large experience yielded him only six cases of enterica complicated by epileptiform convulsions.

There may be all degrees of *delirium* in this disease, from the slightest rambling or moaning during sleep up to the most violent maniacal excitement, under which the patient screams and shouts for hours together. Liebermeister remarks that one may sometimes fail to appreciate the extent to which the mental powers are enfeebled; a man may answer questions as to his actual condition clearly, but when asked how long he has been ill, what is the day of the week, what the month, what the year, he may be altogether unable to reply. Active noisy delirium (which is more common in enteric fever than in typhus) is always a dangerous symptom. A condition resembling delirium tremens is comparatively infrequent, even in persons who have been intemperate. Sometimes the patient unexpectedly jumps out of bed, and may throw himself out of the window; or if not properly watched he may cut his throat, as in a well-known case of suicide at Newmarket which occurred during delirium at a late period of enteric fever. As Gairdner observes, the patient may lie for days perfectly still and quiet, apparently understanding everything that is said or done, but unable to articulate intelligible replies.

The *pupils* in enteric fever are almost always dilated, as Jenner first remarked; but Murchison observes that where there is complete unconsciousness they may be as narrowly contracted as in any case of typhus.

Resolution.—When recovery is to take place the fever sometimes, though very rarely, subsides by crisis. A medical friend of the writer in whom the temperature from morning to evening had been ranging from 101° to 103° with almost absolute regularity, was found, on what was calculated to be about the twentieth evening, to have a temperature of only 101·1°; next morning it was 100°, in the evening 99·3°; on the twenty-second morning it was 98·3°, and for several days afterwards it remained slightly below normal. But the rule is that at some period of the disease, generally towards the end of the third week, the morning temperatures begin to fall, while the evening temperatures rise to the same level as before; so that on the daily chart a series of acute zigzags is formed. After a few days, these become still more exaggerated, there being perhaps a difference of 4° or 6° between the temperatures of a single day; but the absolute height of the evening rise now begins to decline in its turn, so that each day it is about 1·5° or 2° lower than on the previous day; and thus, towards the end of the fourth week, a temperature is reached which is normal or slightly below normal. This *intermittent* type of pyrexia at the termination of the disease is so characteristic that a correct diagnosis has been based upon it alone, in the case of a child about whose earlier symptoms no information was attainable.

Many cases, however, particularly in children, are convalescent several days before the end of the third week; while, on the other hand, there are not a few instances in which the fever runs on for four, five, or even six weeks without any definite change. In a woman who was under treatment

at Guy's Hospital in 1874 the temperature at the expiration of six weeks was still 103·8°, then it fell suddenly, and within two days became normal. It is in such cases that, if death occurs after several weeks' illness, one finds all stages of the lesions in Peyer's patches and solitary glands down to early swelling. There is therefore reason to doubt the statement that fever protracted for more than four weeks is generally due either to some complication or to non-cicatrisation of intestinal ulcers formed in the early period of the disease. Murchison met with several instances in which fresh rose-spots appeared as late as the thirty-fifth day. Some observers think that the right view to take of such cases is to regard them as examples of relapse occurring "intercurrently;" that is, that before the primary attack comes to an end, a second one begins and carries on the fever without apparent break.

During the stage of subsidence of the fever the pulse sometimes remains quick, and the temperature is often subnormal for several days. This is often a period of much suffering to the patient who, as his mind becomes clear, complains more and more of weakness and of prostration and of the impossibility of finding a comfortable posture for his wasted body and limbs. The loss of weight during an attack of enterica is often very considerable, amounting to twenty pounds or even more.

The *convalescence* from enteric fever is slow in comparison with that from typhus, even when its course is uninterrupted by a relapse or any of the various accidents such as will be presently described as complications and sequelæ. Many weeks elapse after the subsidence of the pyrexia before the patient is fit to resume the active duties of life.

We have still to describe (1) cases of enteric fever which run an abortive course or are exceptionally slight, (2) the mode in which death is directly brought about, (3) complications, some of which may indirectly be fatal, (4) sequelæ, and (5) relapses.

Slight and abbreviated cases.—The symptoms of enterica are sometimes from first to last so slight that there is the greatest difficulty in persuading the patient that he is really ill, and in preventing him from going on with his usual work. Such cases are often seen in hospital out-patient practice in the second and third week, and the clumsy but expressive name of *ambulatory typhoid* has been applied to them. Their duration may be as long as that of typical cases, or it may be slightly shorter. In many of them the affected intestinal glands probably subside without breach of surface, but that this is not always the case is shown by the occasional occurrence of perforation. For latent enteric fever is one of the very few causes that may give rise to acute peritonitis without warning, from the floor of a single ulcer giving way.

Again, not a few cases of enteric fever, attended with well-marked symptoms, subside in the course of the third week. Probably the intestinal lesions are comparatively slight, and the swelling of Peyer's patches and solitary glands disappears by absorption without ulceration. But since it is only by the rarest accident that their condition can be verified by an autopsy, it would be unsafe to make this statement absolute, especially as in other forms of the disease there are so many exceptions to the correspondence between clinical symptoms and anatomical lesions.

Cases which terminate before the sixteenth day, however, require special study, because their real nature being overlooked, they are apt to be set

down as examples of "simple febricula," or of a non-specific gastric or intestinal catarrh. Yet it is unquestionable that such cases depend upon infection with the poison of enteric fever, and they often form part of "house-epidemics." Jürgensen, for example, mentions an outbreak near Kiel, in which fourteen out of twenty persons had an abortive form of the disease. Moreover, in addition to malaise, headache, and anorexia, many cases present rose-spots, diarrhœa with characteristic stools, and enlargement of the spleen. The spots are said to appear at an exceptionally early date, sometimes on the second, generally by the fourth or the fifth day. Moreover this abbreviated variety of enteric fever appears to be particularly definite in its onset. In seventy-four of eighty-seven cases collected by Jürgensen it was sudden, and in forty-two it was attended with chilliness or even with a rigor. The temperature generally rises quickly, and it may be 104° by the second or the third day; indeed, Liebermeister says that he has seen cases in which the thermometer rose to 106° in the axilla, and which yet subsided between the fifth and twelfth days. But more frequently in these short cases the temperature does not exceed 102°, 101° or even less. Its subsidence is generally rapid, without presenting the marked zigzags seen in the commoner and more severe form of the disease, and resolution is completed in from one to three days.

Lastly, enteric fever is sometimes not only of short duration, but also of very slight severity. Liebermeister argued that it may be altogether unattended with pyrexia, appearing as a slight non-febrile catarrh of the intestinal canal, but this would be difficult of proof. The slight as well as the abbreviated cases are most often observed among children.

Fatal cases.—The proportion of deaths to recoveries in enteric fever varies in different cities and at different times to some extent, but perhaps less than might have been expected. Murchison speaks of having seen the disease much more fatal in one village than in another a few miles off, and he seems to have been struck by the fact that several members of the same family have sometimes died of it at long intervals and at distant places. It does not appear, however, in malignant outbreaks, such as those of measles and of scarlatina. At the London Fever Hospital the average death-rate from 1848 to 1870 was 17·26 per cent. of all cases admitted, the extreme figures in particular years being 28·42 and 12·82. For purposes of comparison, in estimating the effects of special modes of treatment, it would, however, be necessary to exclude all cases which ended fatally within forty-eight hours of admission; and this would reduce the average death-rate to 15·82 per cent. Statistics from various sources given by Murchison show a range of mortality from 11·37 to 32 per cent. At Basle, the average death-rate from 1843 to 1864 was 27·3 per cent. At Munich for several years before and after 1880 a very low mortality was observed, and ascribed to treatment by baths; but the patients were soldiers, selected healthy young men, under the most favourable circumstances. At Hamburg, according to Senator, the mortality on 937 patients (1874–77) was 7·2 per cent., and on 568 patients after treatment by cold baths was introduced 7·3 per cent. At Berlin, on nearly 13,000 patients of all ages and under various treatment, the mortality was 14·5 per cent.

According to Goltdammer the mortality from enteric fever in the German army is only 10 per cent., in the Austrian 26·8, in the Italian 28·3, and in the French 36·5 (?).

How difficult it is to judge of the normal mortality, uninfluenced by

treatment, even among patients of about the same age, in the same locality, and of the same habits and mode of life, is shown by the following results obtained by Eichhorst in the General Hospital at Zurich in three successive years. In 1884 there was an epidemic of enteric fever, and of 411 patients fifty-six died (13·5 per cent.); in 1885, of 164 patients only seven died (a little over 4 per cent.); and in 1886 there were only 91 cases, of which five were fatal (nearly 5·5 per cent.).

During ten years (1879–88 inclusive) the number of cases of enterica treated in Guy's Hospital was 415; and the number of deaths seventy-two. One of these was from accidental poisoning by morphia, so that the mortality from the disease was 17·1 per cent.—15 per cent. for men, 21 for women.

The causes of death may for convenience of description be divided into two classes: the general effects of the fever, and special complications. These, though often only aiding in the fatal result, not infrequently cut a case short which was running a favourable course. The greater number of deaths are due to failure of the heart's action, particularly in the pulmonary circulation. This leads to congestion of the posterior and lower part of the lungs, the most dependent in the supine posture of fever; and the mere length of enterica as compared with typhus or scarlatina, makes this result more frequent. The hypostatic congestion, or hypostatic pneumonia as it is called, is not ordinary hepatization but a combination of hyperæmia, œdema, and sometimes hæmorrhage, with more or less lobular catarrh and pulmonary collapse.

The weakness of the heart is no doubt aggravated by severe and unchecked diarrhœa or by repeated hæmorrhage from the bowels, in addition to the inability of the patient to take suffieient food, and the direct effects of the febrile process on the muscular tissues. Death by failure of the heart and engorgement of the lungs, somewhat vaguely described as *asthenia*, commonly occurs in the third or fourth week, and occasionally later still.

Sometimes a condition more strictly so named supervenes after the temperature has fallen, and the diarrhœa ceased. There is no evidence of serious obstruction of the lung, either in rapid breathing and cyanosis during life, or in pulmonary congestion after death; but the patient, instead of regaining strength, lies in a state of complete prostration with shallow breathing and fluctuating pulse for hours or even days before death. He may, however, rally from this condition and recover after all, or it may return after apparent improvement.

Death from *syncope*, by sudden failure of the heart, cutting off the supply of blood to the brain and systemic circulation generally, is rare in enteric fever, but would doubtless be frequent if we did not feed our patients with extreme care, and prevent them sitting up in bed until convalescence is established.

Cases are sometimes cut short by *coma* within the first, and more often in the course of the second or by the beginning of the third week. No morbid appearances are discoverable in the brain or in its membranes, except in extremely rare cases of meningitis.

Death from hyperpyrexia is a very rare event in enteric fever.

Complications.—In a considerable number of cases death is brought about indirectly, by affections which can only be regarded as complications. They are more numerous and varied than in any other acute specific disease.

Foremost among these come certain *abdominal* affections arising out of the intestinal lesions.

One complication is severe *hæmorrhage* from the bowels. Its frequency is difficult to estimate, because more or less blood may generally be found in the evacuations, if it is looked for from day to day. But Murchison says that in 3·77 per cent. of his cases it occurred to the extent of more than six ounces. As a direct cause of death it was noted seven times (in four men and three women) among 415 cases at Guy's Hospital, according to an analysis made by our present registrar, Dr E. W. Goodall.

The colour of the blood may be either bright red or dark, and it may be fluid or partially clotted; if it has been retained for a time in the intestinal canal it is dark brown like chocolate. When it is passed during the second week, it is due to a general venous oozing from the swollen Peyer's patches—unless indeed the real date of the commencement of the disease should have been earlier than was supposed. But at later periods it often comes from an artery exposed during the separation of the sloughs. We have more than once seen one particular ulcer deeply blood-stained or with a clot attached to its floor; and Jenner, in a similar case, injected water into the superior mesenteric artery, and found that it escaped from one of the ulcers. In other instances, however, after four or six weeks of illness, a number of ulcers are found all equally reddened, or the whole mucous membrane of the last two feet of the ileum intensely congested, soft, and swollen. Probably intestinal hæmorrhage is sometimes an indication of a scorbutic condition; for we have seen it accompanied by epistaxis, and by the appearance of purpuric spots upon the legs.

When the amount of blood lost is large, the patient exhibits the usual symptoms of profuse hæmorrhage, and becomes blanched and cold. The rectal temperature also is suddenly lowered, a point of importance, as it may afford the earliest indication of what has occurred, before any of the blood has been discharged.

Liebermeister has found the part of the bowel which contains the blood dull on percussion. According to him hæmorrhage diminishes the rapidity of the pulse, and is often attended with a marked alleviation of the cerebral symptoms. But these effects are transitory.

Hæmaturia has been observed alone or along with intestinal hæmorrhage, and also epistaxis.

There has been a remarkable difference of opinion with regard to the influence of intestinal hæmorrhage upon the course of the disease. Graves, and after him Trousseau, declared that it was not unfavourable; but probably the real basis of their opinion was the fact that many patients recover, or, in other words, that it is not often directly fatal. For the statistics of Murchison and Liebermeister show conclusively that the mortality among cases in which this complication occurs is far higher than the average death-rate of the disease.

It is possible that the cases in question are altogether of greater severity than average cases, independently of their being attended with hæmorrhage. As Murchison remarks, the deep ulceration which opens an artery is very likely to extend still further; and thus it is well established that hæmorrhage, when it subsides, is often followed after a few days by fatal peritonitis from perforation of the bowel.

A considerable loss of blood must impair the patient's power of resisting the disease, and promote cardiac failure. Murchison says that he has

repeatedly seen patients who had been doing well die unexpectedly of syncope a few hours after copious hæmorrhage. A case in point occurred at Guy's Hospital in 1879; the patient, a child aged six, went on favourably for a week after the hæmorrhage and then suddenly expired. Dr Goodhart found considerable dilatation and some degree of fatty degeneration of the left ventricle of the heart. The change was perhaps only such as is frequent in uncomplicated cases; but the hæmorrhage probably intensified it, and gave to it a special clinical significance.

It is worthy of notice that hæmorrhage seems never to afford the earliest clinical indication of an enteric fever hitherto latent, as is often the case with perforation.

The other chief abdominal complication of enteric fever is *peritonitis*, from *perforation* of the bowel. Its frequency is very great, especially in England. Thus, whereas Liebermeister gives it as the cause of death in 8 per cent. of fatal cases of the disease, Murchison found it present in nearly 20 per cent. of fatal cases, and in no fewer than 3 per cent. of all his cases, including those which ended in recovery. All writers are agreed that it more often occurs in males than in females, and in seventy-three instances observed by Murchison, the proportion was as fifty-one to twenty-two. In the 415 cases analyzed by Dr Goodall, 22 (*i. e.* 5·3 per cent. of the whole number, and about 30 per cent. of the fatal cases) died from perforation, and of these 12 were male and 10 female. The aperture is sometimes exceedingly small and rounded, but in other cases, as Dr Bristowe stated in vol. xi of the 'Pathological Transactions,' it is linear, showing that it was due to laceration; and stress must be laid upon the fact that its occurrence is sometimes directly traceable to disturbance of the bowel during defæcation, by the administration of an enema, in the act of vomiting, or in sitting up to take food; or it may be due to irritation from solid fæcal matters, or to the presence of pent-up gas. Again, in certain cases, the gangrenous process affecting a Peyer's patch extends directly through the whole thickness of the bowel, and then a large slough may drop out, leaving a hole of considerable size. One may even find a number of such holes at different spots. Liebermeister observed that when gas escapes freely into the peritoneum, it allows the liver to fall backwards from the ribs, so that the percussion-note in the right hypochondriac region becomes tympanitic. Sometimes a large quantity of fæcal matter is extravasated, and thus round-worms have been found free in the peritoneal cavity.

The seat of perforation is almost always in the lower end of the ileum within a foot or two of the valve. It is said to be sometimes in the cæcal appendix or in the colon, but no instance of either kind appears to have occurred at Guy's Hospital within the last thirty years.

The date at which this terrible complication is more apt to occur is in the third, fourth, or fifth week of the fever. Cases have been recorded in which it has been as early as the eighth, ninth, eleventh, or twelfth day of the patient's illness, but in all probability the disease was really more advanced. Perforation is often the cause of death when there has been profuse diarrhœa, and all the symptoms well marked; but it is sometimes the first sign that anything is amiss with the patient, when the course of the disease has been altogether latent. So, also, it may occur at a very advanced stage, when two, three, or even four months have elapsed, and this may be the case not only when the fever has been unusually protracted, or when there has been a definite relapse, but even when convalescence

has apparently been established, and when perfectly formed and healthy fæces have been passed. In two instances that have occurred at Guy's Hospital, each at the end of the sixth week, perforation has been distinctly traced to the ingestion of improper food. One patient was seized with pain very soon after eating two raw apples, another while eating water-cresses.

The symptoms of perforation are identical with those of a sudden attack of peritonitis from whatever cause, but it is important to note that enteric fever yields a large proportion of cases in which peritonitis begins insidiously, so that it may remain altogether unnoticed. In a case observed in 1864 at Guy's Hospital, the chief sign of an unfavourable turn in the patient's illness was his sudden refusal to take food, after which he died in a few hours. Perforation and consequent peritonitis are often found at a *post-mortem* examination when they had not been suspected.

It has been much discussed whether recovery after this complication is possible. The doubt lies in the difficulty of determining whether there is an actual perforation in any given case of peritonitis; but Liebermeister says he has had four cases of recovery in which the abdominal cavity contained pus, and several instances have been recorded in which, death having occurred from some other cause, a perforation has been found sealed up by adhesions, or at least closed off from the general peritoneal cavity. In other cases a circumscribed abscess has formed, which has discharged itself either externally or into the bowel.

In a case of severe enteric fever which occurred in a boy of ten, who lay for many weeks in Philip Ward, during the winter 1888–9, there were one morning all the signs of perforation,—sudden pain, meteorismus, and collapse, with thready pulse and sighing respiration, and extreme local tenderness. A hopeless prognosis was given, but he was treated by laudanum and frequent small doses of brandy, and very gradually the symptoms improved, until he completely rallied, and finally made an excellent recovery.

Acute peritonitis does not invariably start from an intestinal ulcer. Occasionally the mere spreading of a putrid inflammation to the serous surface may light up peritonitis without any actual perforation taking place. It may be set up by sloughing of a swollen mesenteric gland, as in a case recorded by Jenner, in which recovery took place, so that the real nature of the complication would not have been known if the patient had not afterwards died of erysipelas. It has also sometimes been due to extension from an inflamed and ulcerated gall-bladder, or from a suppurating embolus in the spleen.

Such embolic masses in the spleen are not infrequently found after death from enteric fever. They are doubtless the result of plugging of branches of the splenic artery with portions of clot that had formed in the almost stagnant blood in the dilated left cavities of the heart. In the same way embolism of one of the cerebral arteries, causing hemiplegia, with or without aphasia, may arise either during the course of the disease, or later when convalescence has already occurred.

Other complications of enteric fever have their seat in the respiratory organs. A certain degree of *bronchitis* is almost always present, but sometimes it becomes so severe as to constitute a very important part of the disease. Apart from the hypostatic broncho-pneumonia already mentioned as a frequent occurrence, lobar *pneumonia* with true hepatisation, is far more

common than in typhus, and sometimes passes on to gangrene. Pleurisy, too, is often seen, and may lead to empyema.

Ulceration of the *larynx* is present in a considerable number of cases, but it seldom or never produces symptoms during life, and will therefore be described among the anatomical lesions found after death.

Another complication of enteric fever is *parotitis*. This, however, is much less frequent than in typhus. It sometimes affects one side, sometimes both. It may either subside after a week or two, or it may lead to suppuration, with extensive brawny induration and purulent infiltration of the side of the neck as far as the sterno-mastoid muscle.

The occurrence of enteric fever often leads to the reopening of fistulous openings which had healed up, with consequent *necrosis* of extensive portions of bone. Apart from previous disease, periostitis is not an uncommon sequel: according to Paget it most frequently affects the tibia, next the femur, and then the ulna and cranium. In patients who have been suffering from soft chancres he has seen wide-spreading gangrene under the influence of an attack of fever. In two cases at Guy's Hospital an ordinary gonorrhœa has led to sloughing of the penis or of the scrotum under similar circumstances.

As already remarked, in some very rare cases *meningitis* is believed to occur as a complication of enteric fever.

Lastly, we have repeatedly observed renal complications of enteric fever which do not seem to be common, but which have more than once led to a fatal result, and are therefore of practical importance.

Among our 415 cases of the past ten years, two (both in women) were fatal from *suppuration of the kidneys*, and two others from the same cause had been previously recorded. In two cases there was cystitis, sometimes perhaps caused by retention of urine, but this condition, with the attendant dangers of catheterism, is less common in women than in men. In one of these cases (November, 1888) we found the bladder and uterus normal; and in another (Philip Ward, May, 1888) there was no retention of urine, no stricture, and no pyelitis.

In two other patients (also women) acute *tubular nephritis* was found after death, and in one of them, who was under the writer's care in August, 1886, this appeared during life to be the fatal complication. In a third case the patient, a boy of fourteen, happened to be the son of one of the women who died from suppurative nephritis in the hospital a week before (November, 1888). Mother and son had come in with typhoid fever, and both died with uræmic symptoms but from different renal lesions, for in the case of the latter there was anasarca and severe diarrhœa, and acute tubal nephritis with catarrhal colitis were found after death.

4. *Sequelæ*.—In certain cases, affections which are described as complications of enteric fever do not begin until the patient is already convalescent. It is therefore impossible to draw a fixed line between them and the sequelæ of the disease, which themselves may set in before it has come to an end. What really justifies the distinction and renders it useful in practice, is that the latter often run on for many weeks, and obviously constitute the sole obstacle to the restoration of health.

This is the case with *bedsores*, which are apt to form over the sacrum and hips unless great care is taken to prevent them, and which sometimes destroy life by exhaustion, or indirectly by setting up pyæmia.

Another sequela is *thrombosis* of the femoral vein, with the attendant liability to pulmonary embolism.

In some cases enteric fever is followed by *marasmus*. Murchison says that there is not always a repugnance to food; the patient may eat well, but what he takes is not assimilated, and slight errors in diet cause flatulence and diarrhœa. Yet the temperature is normal or below normal, and if death occurs, perhaps after many months, nothing can be discovered except an unusually smooth appearance of the mucous membrane of the ileum, and a shrivelled condition of the mesenteric glands.*

Writers generally speak of pulmonary *phthisis* as frequently occurring after enteric fever. But it is a remarkable circumstance that after searching the records of *post-mortem* examinations at Guy's Hospital, Dr Fagge failed to find a single case in point.

Paraplegia has been recorded by Nothnagel and others as an occasional sequela. It is uncertain whether the lesion in these cases is in the cord or in the peripheral nerves.

5. *Relapses.*—The return of the temperature to normal is not necessarily followed by the restoration of the patient's health; enteric fever is apt to relapse. The frequency of such an occurrence seems to differ in different places: at Basle, Liebermeister met with it in 8·6 per cent. of 1743 cases; Murchison says that in the London Fever Hospital it was observed in 3 per cent. of 2591 cases; the figures given by other writers vary from 11 to 1·4 per cent. Among our 415 cases from 1879 to 1888 at Guy's Hospital there were 46 relapses, *i. e.* about 11 per cent. As a rule, there is an interval of some days between the termination of the first and the beginning of the second attack. Murchison states it as eleven days on an average; Liebermeister found that among 111 cases it was less than four days in twenty-seven, from five to seven days in seventeen, from eight to fourteen days in thirty-five, and still longer in thirty-two. The late Dr Irvine, however, in a valuable paper in the 'Medical Times and Gazette' for 1879, maintained that the average interval is not longer than five days. He believed that a relapse sometimes begins before the original illness has come to an end. As already remarked, this may explain those cases in which enteric fever seems to run on indefinitely for six or seven weeks or longer. It is not very uncommon for the termination of a relapse to be followed after a second interval by a second relapse, and this, again, may be succeeded by a third, and even by a fourth. There is scarcely a more important use of the thermometer in clinical practice than in the detection of such secondary attacks of fever. For it sometimes happens that the patient is unaware that anything is amiss with him, and yet to allow him to get about and to return to ordinary diet is to expose him to fearful risk. Thus at Guy's Hospital in 1876, a man was apparently going on favourably through convalescence, when on the twenty-ninth day his temperature was found to be 104·2°; he looked ill, though he declared that he did not feel so; and a few days later he died of perforation of the intestine. In other instances the thermometer indicates but a very moderate degree of fever, ranging from 100° to 101° or 102°, but nevertheless taking

* When diarrhœa has persisted after the subsidence of the fever, it has been supposed that the ulcers have remained unhealed, or (to employ the usual phrase) have passed into an "atonic" condition. But although Murchison supports this statement with the weight of his authority, I am not sure whether it rests on strict pathological evidence; Dr Wilks long ago taught me to doubt its accuracy, and I have never myself met with any example of it. I remember one case in which a patient was admitted into Guy's Hospital, whose sole complaint was pain in the right iliac fossa, which appeared to have been left behind by an attack of enteric fever; after a few weeks this pain subsided.—C. H. F.

a perfectly typical course. Dr Irvine suggested that when a relapse has been supposed to occur many weeks after convalescence, there have really been one or more intervening attacks which escaped notice. The duration of a relapse is by most writers said to be shorter than that of the original attack. Murchison found it to be on an average sixteen days. Dr Irvine, however, maintained that it is typically twenty or twenty-one days. Cases in which it appears to be longer he explained by the hypothesis of an intercurrent second relapse, interrupting the middle of the first relapse, and subsequently running a regular course. According to this observer the temperature in a relapse generally rises pretty steadily, until on the fifth day it attains its maximum; it remains high until the ninth or the tenth day, when it undergoes a marked fall of from 2° to 6°; directly afterwards, however, it rises again, but it henceforth shows daily remissions, which at length bring it to an end.

It is now well ascertained that relapses of enteric fever are attended with a renewal of the intestinal lesions, although Trousseau maintained the contrary. We have had at least nine cases fatal during relapse at Guy's Hospital within the last twenty-two years, and in every one of them recent morbid changes have been found. As a rule, some Peyer's patches are in a state of early swelling, or have partly formed sloughs, while others show clean ulcers or cicatrices; but in one instance the floors of the patches are said to have been smooth and bare, while their edges showed the pink tumefaction of commencing disease.

The symptoms of a relapse do not differ from those of a primary attack. Rose-spots are said by Murchison to appear somewhat earlier; in twenty-two out of thirty-eight cases they were visible on the third, the fourth, or the fifth day. In all of Dr Irvine's cases the stools were characteristic in appearance. The patient is often delirious and insensible; and, considering the weakness resulting from his first illness, one is surprised that he does not more often succumb. As a fact, however, relapses are seldom fatal, except by some complication, such as perforation, from which recovery is practically impossible. The only one of 46 relapses which ended ill during ten years at Guy's Hospital was one of perforation.

The cause of the liability of enteric fever to relapse is still imperfectly understood. It cannot be due to a fresh infection with the specific poison from without, for the patient is often in a hospital, far removed from the original source of his disease, and little exposed to other infection.

Very often the time at which he falls ill for the second time is when he has just begun to take solid food, or when he has once or twice been out of bed. But other instances cannot possibly be thus accounted for; and at the best, such an apparent exciting cause can only be regarded as setting up the symptoms of a morbid process which would otherwise have remained latent; since, if the patient happens to die a few days later, the intestinal lesions are found to be so far advanced that they clearly must have begun before the obvious relapse set in. For instance, in 1876, a case ended fatally on the sixth day of relapse, and several of the Peyer's patches had already ochrey yellow centres. Such cases probably afford the key to the whole question. In August, 1861, a man was admitted into Guy's Hospital with bronchitis and emphysema. A month later he was attacked with enteric fever, which ran a regular course and from which he recovered, so as to be able to sit up. But early in October the weather became very cold, whereupon his bronchitic symptoms underwent rapid aggravation, and

in a few days they proved fatal. At the autopsy, the lower Peyer's patches were found to be roughened and flocculent, as if sloughs had separated from them; but high up in the ileum one or two patches were still somewhat raised, and several solitary follicles showed an early stage of the affection. This case seems to prove that after an attack of enteric fever has to all appearance passed off, intestinal lesions may smoulder on without giving rise to any symptoms.

Dr Maclagan some years ago suggested that sloughs thrown off from the patches first diseased might perhaps infect other patches in their turn. Such a hypothesis is difficult either to prove or to disprove. But with the analogy of spirillum fever to guide us, we may suppose that the relapse of enteric fever is due to a reinfection of the blood with a virus (doubtless consisting of specific organisms) derived from patches thus recently diseased.

Protection.—Notwithstanding the phenomena of relapse, there is a marked immunity from a second attack of the disease among those who have once finally recovered. In our 415 cases there was only one of a second attack, and that happened five months after the first: after death, from perforation, beside the recent ulcers healed scars were seen in the ileum.

Murchison quotes an observation made by Gendron and Piedvache, that after an interval of many years a particular house or locality became for the second time the seat of an outbreak, whereupon it spared those who had previously suffered, but attacked almost everyone else. Murchison, however, had himself met with several exceptions to this rule, and he cites others.

Diagnosis.—The diagnosis of enteric fever may either be easy or difficult. In some cases a single examination of the patient may enable one to speak positively of the nature of his illness, especially in the second or third week; in some others the most careful clinical observation, from day to day, may leave one to the last in a state of doubt.

At its commencement the disease can never be determined with certainty, although strong suspicions may be excited if there have been other cases in the same house, or the same neighbourhood. In the absence of other cases, the most common mistake is to call the illness a "bilious attack;" and great harm is often done by the aperient dose which follows. The known fact that the onset of enteric fever is generally insidious is apt, when it begins suddenly, to suggest the idea that the case is rather one of typhus, of some exanthem, or of influenza. Liebermeister lays stress on the rarity of nasal catarrh and of sneezing at the beginning of enterica; and another point is that it is very seldom attended with herpes of the lips. As already mentioned, cases of enteric fever have been at first set down to mania.

The occasional early roseola should never be mistaken for scarlet fever, for it does not appear until the fourth or fifth day. Murchison has known a copious eruption of rose-spots attributed to smallpox, in spite of their late appearance and very different distribution.

The mulberry rash of *typhus*, with its petechiæ, its dull diffused mottling, its earlier advent, and its much wider and more abundant distribution, is so different from the scanty crops of rose-spots on the clear skin of the tumid abdomen, that since the distinction between typhus and enteric fever was established mistakes between them are very rare. It must, however, be remembered that typhus is sometimes accompanied by diarrhœa and enterica by constipation. Even where no rose-spots appear one can often feel confident

of the nature of the disease from the course of the pyrexia, the presence of an enlarged spleen, and the characteristic appearance of the stools. But in most cases one's opinion is in reality mainly founded upon negative considerations, and, as Liebermeister remarks, he who is most sensible of his liability to errors is least likely to commit them.

About the end of the first week (that is, within two or three days of the time when the case generally comes under medical observation) a positive diagnosis can, as a rule, be given.

The diseases which are liable to be confused with enteric fever without eruption, or before it has appeared, or when it has been overlooked, may be divided into two groups.

First come certain general diseases. Of these the most important in many countries, although not in England, is *ague*. In India and many parts of America the difficulty of distinguishing the remittent forms of marsh-poisoning from enteric fever, whether in individual cases or throughout a district, is so great that, unless opportunities should arise for making autopsies, mistakes seem almost inevitable, and probably this has led to the hypothesis of a "mixed" form of "typho-malarious fever."

In this country the most important disease simulating enteric fever is *general miliary tuberculosis*, without marked symptoms indicating localisation in any particular organ. But although it is quite true that at an early period of a case it is often impossible to say whether a patient is entering upon one or the other of these diseases, yet our *post-mortem* records at Guy's Hospital show very few, if any, fatal cases in which a correct diagnosis has not been made. It has recently been the writer's fate to supply this deficiency. A boy was ill of enteric fever, which was without diarrhœa or rash, and accompanied by severe broncho-pneumonia. It ran a protracted course, with cyanosis and high irregular temperature, and we regarded it as tuberculosis, and not enterica, up to the time of death. A most remarkable instance of persistent obscurity is recorded by Senator in the 'Berlin. klin. Wochenschrift' for 1881. A man, aged forty-two, was admitted into hospital on October 25th, 1880. He had not been quite well since the beginning of September, but his illness had been more marked for about ten days before his admission. His pulse was 80 to 84. His temperature was 100·4°, and for some days it ranged from 101° in the morning to 103° in the evening very regularly. On October 29th the spleen was found to be enlarged, and on October 31st distinct rose-spots appeared, and fresh ones again on the following day. On November 1st and 2nd there was repeated epistaxis. The pulse, still 80 to the minute, was now plainly dicrotic. Signs of bronchial catarrh appeared, and increased greatly up to November 11th. The bowels were open without diarrhœa. On November 21st suppuration of the left parotid occurred, with discharge of pus through Steno's duct. The diagnosis, about which up to this time there had been slight doubts, was now finally given in favour of enteric fever. Yet after the patient's death, which occurred on November 22nd, miliary tubercles were found in the lungs and in other organs, while the appearances characteristic of enteric fever were altogether wanting. The ophthalmoscope had been used with negative results. A correct diagnosis of this case was surely impossible.

The second general disease that in temperate climates is often mistaken for enteric fever (or the fever for it) or is *pyæmia*, when its starting-point is some deep-seated affection, and when it affects, not joints, but internal organs. Murchison saw several cases of pyæmia from caries of the petrous bone, of

which the course was very like that of enteric fever. At Guy's Hospital, in two instances at least, this mistake was actually made. In each of them the source of the mischief was latent disease of the lumbar or the dorsal vertebræ, there being secondary abscesses in the lungs and the kidneys, and one in the heart. The variations of temperature are in pyæmia always much greater and not unfrequently touch or fall below the normal, which does not occur in enterica except from severe hæmorrhage or from perforation.

Trichiniasis has often been set down as enteric fever by those who have not seen it before; but with due care this error might probably be avoided.

Again, many local diseases may be overlooked, and their effects attributed to enteric fever, if a positive diagnosis be rashly made in the absence of rose-spots.

Foremost among these is a cerebral affection, *tubercular meningitis*, to be afterwards described. An important point, which has been insisted on by Jenner, is that in enteric fever headache ceases before delirium begins; the two symptoms are not present simultaneously as in meningitis, unless indeed this is actually present as a complication.

Before the clinical thermometer was in use, enteric fever was sometimes mistaken for hysteria; in a diabetic patient it has been set down as diabetic coma; and in one who was the subject of lead-poisoning, uræmia, from granular disease of the kidneys, has been mistaken for enteric fever.

Among thoracic affections, *tuberculosis of the lungs* must especially be borne in mind. Enteric fever is often accompanied with bronchitis; and even when miliary tuberculosis produces marked thoracic symptoms it may be difficult to say whether they are not rather due to this complication of fever. Formerly *acute phthisis*, causing a rapidly spreading consolidation of one or of both lungs, was sometimes mistaken for typhoid fever; and a similar error was even made with regard to *simple pneumonia*, when there was no pain in the chest, nor cough, nor expectoration. Even now, although the routine use of the stethoscope keeps us from overlooking the presence of a pulmonary lesion, we may sometimes be in doubt whether it is the primary disease or merely a complication of fever.

Ulcerative endocarditis has often been mistaken for enteric fever; the discovery of a cardiac murmur should in most cases suffice to prevent this error, but in exceptional cases the heart-sounds remain normal. Moreover, a patient already the subject of valvular disease, might fall sick with pyrexia and enlarged spleen, which might be due to enteric fever or to septic endocarditis. Even the occurrence of hæmaturia or hemiplegia would not be decisive.

Various abdominal affections are liable to be confounded at the bedside with enteric fever. Foremost among them is *tubercular peritonitis*. In the absence of the positive signs of this affection its diagnosis from enteric fever may be impossible. We have had more than one case of which the nature has still remained doubtful after several weeks of illness. Again, two or three instances have also occurred at Guy's Hospital in which *acute diffused inflammation of the colon*, generally of diphtheritic character, has been mistaken for enteric fever, there having been no symptoms of dysentery, so far as could afterwards be ascertained.

Other diseases which have failed to betray their presence during life have been *typhlitis*, *perinephric abscess*, and *abscess of the liver* secondary to ulceration of the colon, or (in another case) to suppuration in the broad

ligament of the uterus, with an opening into the intestine. Of these affections typhlitis is the most likely to be mistaken for enterica.

Finally, it is most important to remember that some patients omit to complain of *affections of the genito-urinary organs* sufficiently severe to cause profound constitutional disturbance, and that others conceal them from mistaken modesty. Sir William Gull used in his lectures to speak of cases of extravasation of urine to which he had been called as to typhoid fever, on account of the stupor, muttering delirium, and a dry brown tongue. Some years ago Dr Fagge was asked to see a girl who had been suffering from febrile symptoms, with a very quick pulse. It was not until she had been sent home that she mentioned to her mother that she had severe pains in micturition. There was then found intense diphtheritic inflammation of the labia, and she narrowly escaped with her life. In these cases, as in tuberculosis, and pyæmia, and septic endocarditis, the condition is "typhoid" and it is "fever," but it is not due to the enteric contagium.

Anatomy.—In typhus and in relapsing fever the "general" or "constitutional" character, which is one of the notes of the whole group of specific fevers, is unmodified by any predilection of the contagion for fixing itself in one part more than another. Blood and lymph, solids and liquids, bones, and viscera, all are alike invaded by the poison, and its effects only differ in accordance with the functions of the several organs.

But in several specific fevers we find, in addition to the universal "intoxication" with the poison, that it fixes itself peculiarly in certain *foci*, as we may call them, where it produces definite local lesions. Thus measles, we shall see, particularly affects the respiratory mucous membranes, and scarlet fever the throat. But nowhere is this localisation of the disease so remarkable as in enteric fever. In fact, so striking and clinically important are the local lesions that it was possible for Broussais and his school to regard them as the primary disease, and the fever as merely a symptomatic result. Probably the "typhoid deposits," as they used to be called, in the intestine, are to be regarded as infective granulomata, produced, like the products of tubercle and leprosy, by the local action of specific bacilli.

The most important lesions of enterica are situated in the lymphatic follicles of the intestine, both agminated and solitary. These first become injected and swollen, so that they project further above the level of the surrounding mucous membrane than is naturally the case, even in children. As a rule they rise to the height of one or two lines only; but in some exceptional cases, according to Hoffmann, they may be three eighths of an inch thick. Louis distinguished *plaques molles* from *plaques dures;* the difference between them is, however, merely one of degree, the hard plates being those in which the change is most rapid and intense. After a few days the redness passes of, and the diseased follicles become pale, so white that they have been compared to a thin layer of a soft medullary growth, and in the early days of morbid anatomy were even described as encephaloid cancer of the bowel. The microscope shows that there is a very abundant formation of new cells, which are considerably larger than leucocytes, have large nuclei, and often exhibit signs of multiplying by fission. These cells, besides distending the lymph-follicles, infiltrate the intervening fibrous septa, and may even spread into the mucous membrane above, and into the interstices of the muscular coat and subserous tissue beneath.*

* I have never seen them forming grey granules on the peritoneal surface, as is

The next occurrence is generally ulceration. This seems sometimes to begin as an abrasion of the surface of the diseased follicles, and gradually to extend through their substance. But much more often the whole of the infiltrated tissue, or at least a large part of it, dies in a mass. It then forms a soft, shreddy, flocculent slough, which is of a bright ochre-yellow colour, apparently from imbibition of bile pigment from fæcal matter. Presently the slough is detached, either entire or in fragments. There is then exposed the floor of an ulcer, in which the transverse muscular fibres are often plainly visible, while the gut is so thinned that it is translucent when held up to the light. The muscular fibres themselves may undergo destruction to a greater or less extent, so as to lay bare the subserous tissue—to the imminent risk of perforation into the peritoneal cavity. The edges of the ulcer are thin, soft, and rounded; and when examined under water they are seen to be slightly undermined. Its form at first corresponds with that of the lymphatic organ in which it took its origin; if in a solitary follicle, it is small and rounded; if in a Peyer's patch, it is usually elongated in the direction of the axis of the intestine. In the latter case, too, its position is always remote from the line of attachment of the mesentery. In these respects, as well as in the characters of their floors and edges, the ulcers of enteric fever differ from tubercular ulcers. At advanced stages of the disease it not seldom happens that they are found to be elongated transversely to the axis of the intestine by secondary ulceration, their form being in fact the reverse of that which generally characterises them. At length their floors become covered with a thin grey layer of granulation-tissue, and they heal by the gradual growth of mucous membrane from their edges towards their centres. At Guy's Hospital five cases are recorded, in which death occurred from some other disease within a few months after recovery from enteric fever; in two of them the patches were of a blackish colour, with slaty margins; but in the others the cicatrices seem to have been white; and in one of them it was only on close examination that a deviation from the normal appearance, consisting in a slight unevenness of surface, could be recognised. There is never any thickening or puckering of the affected tissues after this disease, so that it cannot lead to narrowing of the bowel.

Enteric fever by no means affects all the lymph-follicles of the intestines equally. In a large number of cases the solitary follicles entirely escape; in many others those of the ileum are alone attacked; and when those of the large bowel suffer, it is often only in the cæcum, or in the ascending colon; in a very few instances the morbid change extends even to the rectum. It seems, in fact, to spread from the ilio-cæcal valve as from a starting-point. Precisely the same thing is observed with Peyer's patches; the highest of them are very seldom, if ever, affected. As a rule, the lesion is limited to patches within two or three feet of the valve, and sometimes it does not reach beyond a few inches. In very exceptional instances the agminated patches of Peyer remain unaffected, and the solitary follicles bear the whole brunt of the disease.*

described by Hoffmann. But it is not uncommon for adjacent Peyer's patches to become fused together by an extension of the morbid growth, which thus may affect the whole circumference of the lower part of the ileum for some inches above the valve.—C. H. F.

* This apparent caprice of distribution in different cases cannot but suggest the inquiry whether there may not be some in which no glands suffer at all, so that, if the nature of the disease were otherwise doubtful, the criterion afforded by morbid anatomy would fail. The nearest approach to this, with which I am acquainted, is afforded by a specimen which

The morbid process is almost always farthest advanced in the follicles close to the valve; and very often every stage is present, from clean ulcers below, through ulcers containing scattered shreds of slough, to those in which sloughs have just been formed, and from these, again, to patches which are only swollen and excoriated. Sometimes, indeed, the progression is not perfectly regular, a patch less affected being seen at a lower point than one which is more affected. Most pathologists think that these differences depend upon the fact that the follicles are attacked, not simultaneously, but in succession. Dr Moxon, however, held that the morbid change is of less severity, rather than of later date, in the higher patches. He thought that the greater tendency to destruction of those which lie at the end of the ileum is due to the irritating contact of the intestinal contents, held back upon them by the valve. There can be little doubt, however, that in some of the more protracted cases the glands are affected successively and at considerable intervals of time.

But the swollen patches, instead of sloughing or ulcerating, sometimes entirely subside, as was long ago pointed out by Chomel and Louis. They supposed that the morbid material in the follicles underwent softening, and that they ruptured so as to allow it to escape into the bowel. Hoffmann still says that this occurs, and that the follicles become stained in consequence of a little hæmorrhage, so that the Peyer's patches acquire a dotted appearance, the *état pointillé* of French writers. This condition, which has also been compared to a shaven beard, is not peculiar to enteric fever, nor is it uncommon in those who have died from other causes and at all ages. It would be too arbitrary to assume that there was always an attack of typhoid fever which occurred so long ago as to have been forgotten. However this may be, there seems to be no doubt that subsidence of the swollen glands often takes place in a very different manner, namely, by the cell growth in the follicles disintegrating, and undergoing absorption like an inflammatory exudation.* As a rule absorption is found to have taken place in certain patches only, or even in parts of them, while elsewhere there are sloughs or ulcers. Thus in 1862 Dr Wilks examined a case in which, while the solitary follicles had each a depression in its centre, some patches appeared to be in a state of retrogression, parts of them only being slightly raised and nodular.

It is a matter of some practical importance to determine as accurately as possible the dates at which, reckoning from the beginning of the fever, one may expect to meet with the several stages of the intestinal lesions.

I showed at one of the meetings of the Pathological Society in 1875. The only lesions in the intestine were the following:—"One ill-defined purplish-red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water." I think it is by no means unlikely that in mild cases of enteric fever, such as could never prove fatal except by some accident, the intestinal lesions are often very slight, and may possibly in rare cases be altogether absent.—C. H. F.

* Among cases which end in recovery it is perhaps not uncommon for all the patches and all the solitary glands which are affected to take this course, but when death occurs at an advanced stage it very rarely happens that some are not found ulcerated. I have only met with one instance of the kind; the patient died on the twenty-first day, and the patches were found swollen, raised, and reddened, but marked by irregular depressed lines and spaces, so that they had a reticulated appearance. They were not "medullary" in character, as would probably have been the case if the affection had been early; there was no trace of sloughing or ulceration. I preserved the specimen in spirit for the museum, but after a few weeks it showed nothing.—C. H. F.

According to Liebermeister, the first week is occupied by swelling and infiltration of the lymph-glands of the bowel; during the second week they either slough or begin to subside; in the third week any sloughs that may have formed become detached, so that by the twenty-first day all the ulcers have clean floors; during the fourth week they begin to heal, but the process is often not completed until a much later period. This statement is easy to remember, and in many cases it may be true, but it certainly is not so universally applicable as to enable us, from the anatomical appearances in a given case, to state positively the duration of the patient's illness. According to Trousseau the swelling of the intestinal glands does not begin to appear until the fourth or fifth day; according to Chomel and Louis not until the seventh or eighth day; but Bristowe and Hoffmann found them enlarged in cases fatal on the fifth and fourth days respectively; and Murchison relates one instance, that of a girl who died forty-seven hours after being suddenly attacked with vomiting and fever, in which the solitary follicles were of the size of hemp-seeds or split peas, Peyer's patches being also similarly affected. Is it not, however, probable that the disease in that case began earlier than its symptoms? In 1871 a man died in Guy's Hospital, who had been definitely attacked twelve days before his death with shivering and aching pains in his limbs, so that he was obliged to go home, but at the autopsy his intestine contained extensive ulcers, all with clean floors except one, which had a few fragments of slough still adherent to it. Surely the lesion must have been in progress before his illness began.

In fact, one is led to the belief that during the period of incubation the invading bacilli produce a local "infective granuloma," and that this remains latent until they (or their spores) leave the intestinal follicles by the lymphatic channels, and overspread the entire organism in swarms.

On the other hand, it is not uncommon, in cases fatal at the end of six or seven weeks of continuous fever, to find sloughs still adhering to several of the patches, while others are merely swollen and medullary looking. But probably the lesions are there of different dates, and have been developed in successive outbreaks at intervals of a fortnight or longer. In the case of a boy who had been ill for five weeks, and who died in Guy's Hospital on October 26th, 1879, Dr Goodhart describes the glands as fleshy-looking, and as just beginning to ulcerate, so that he would have supposed the disease to be at about the eighth or the ninth day. Doubtless some other glands had been affected from the first but had afterwards subsided.

Another lesion in enteric fever has its seat in the *mesenteric lymph-glands*. This does not appear to be absolutely constant; at least, in our records of autopsies there are two cases, one of them fatal on the seventeenth day, in which the glands are said not to have been enlarged. It may be present at a very early period of the disease; thus, in Hoffmann's case, fatal on the fourth day, the glands were already doubled in size. No doubt it results from absorption by the lacteals of contagion from the affected parts of the intestines, and in many instances the lower glands, corresponding with these parts, are alone attacked; but sometimes the upper mesenteric lymph-glands become also involved, and even (as in a case inspected in 1878) glands in the portal fissure. As a rule, the enlargement is moderate, the diseased glands not being larger than hazel-nuts, but they may be as big as horse-chestnuts. Their substance is white, or grey, or pinkish, with spots

of hæmorrhage. Under the microscope they are found to contain large cells, some of which have two or more nuclei, like those in the intestinal follicles. Suppuration sometimes occurs, or partial sloughing, and under such circumstances a cheesy mass may result if the disease subsides; but caseation, or the deposition of calcareous salts, certainly does form part of the ordinary retrograde process.

The *spleen* is found as a rule enlarged and soft, but there are exceptions to the rule. Even in young subjects who died at the height of the fever, it has been found at Guy's Hospital to weigh only six, five, or even four ounces. Bacilli have been repeatedly found in this organ, and have been referred to the specific pathogenic species. Indeed, one enthusiastic pathologist has proposed to insert a small trocar and cannula into the spleen during life in order to diagnose enteric fever, by finding its bacilli in the drops of splenic blood thus obtained.

The *larynx* is sometimes found ulcerated. Hoffmann observed this in twenty-eight cases out of two hundred and fifty. It has generally been set down among primary lesions of enteric fever, but the absence of a specific cell-growth seems important. Probably the explanation of its occurrence lies in some subtle association in morbid tendencies between the larynx, the lungs, and the intestine, of which we have an example in phthisis. As a rule there is a sharply defined ulcer over the base of one or both of the arytænoid cartilages. This lesion very rarely gives rise to symptoms, but it may cause hoarseness, or aphonia. In some instances, however, the morbid action involves deeper structures, and it may even be attended with necrosis and exfoliation of the cartilage, as in a case inspected in 1879. Hoffmann once saw the cricoid cartilage almost completely denuded. The entrance of air may, under such circumstances, be greatly obstructed. Laryngeal perichondritis by Dittrich has been assigned (in other cases as well as in fever), a mode of origin like that of bedsores. And since, according to Ziemssen, modern antipyretic treatment has much diminished the frequency of laryngitis in enteric fever, it seems fair to conclude that the pyrexia is in some way concerned with them. In another of Hoffmann's cases tracheotomy was rendered necessary by the formation of a polypoid blood-clot, which hung down into the interior of the larynx. Again a laryngeal ulcer may endanger life by eating its way deeply into the connective tissue. In a case that occurred many years ago to Dr Wilks subcutaneous emphysema arose from this cause; the patient was a boy aged twelve. A similar instance, in a girl four years old, has since been recorded by Ziemssen. In his report of this case it is specially stated that the seat of the ulcer, which led down to the necrosed cricoid and arytænoid cartilages, was below the cords, and as the child had had much bronchitis there can be no doubt that the cough had forced air through the ulcer into the connective tissue. In two cases the writer has seen sloughs in the larynx of a bright yellow colour, exactly like that which in the intestine is attributed to the imbibition of bile. In one of them there were two linear ulcers, situated one on each side of the epiglottis, a position which is described by Hoffmann as frequent.

Sometimes a thin pellicle of lymph has been found lining both the interior of the larynx and the epiglottis, and still more rarely, a membranous layer resembling that of diphtheria. Whether the specific poison of diphtheria has then been in operation it is difficult to say, but Murchison relates an instance in which fluids escaped from the nostrils when the patient attempted to swallow them, exactly as in that disease.

Zenker, in 1864, pointed out two kinds of degeneration in the fibres of voluntary *muscles*; some become granular, others undergo conversion into a glassy-looking substance, in which no striæ can be recognised, and which splits up transversely into discs. As he himself remarked, however, the same lesion occurs likewise in other febrile diseases, if sufficiently severe and protracted. It is especially marked in the adductors of the thighs and in the recti of the abdomen. Muscles so affected are said to be sometimes obviously altered in appearance, being of a yellowish-grey colour, but this admits of doubt. They may during life be so much softened that they become extensively lacerated; thus, in 1870, a patient died of enteric fever in Guy's Hospital both of whose recti were torn across, and much blood was extravasated into them and into their sheath; and in another case, in 1866, the inner part of one rectus was found to be not only ruptured, but in a state of suppuration. Hoffmann has shown that similar forms of degeneration occur in the tongue, accounting perhaps for the tremor of its movements.

The *heart* is then found to be soft and pale, and sometimes its right ventricle is dilated, or even its left ventricle. Its muscular fibres are commonly more or less granular, and may have lost their transverse striation. In one instance Hoffmann detected in them the glassy change just described as occurring in the voluntary muscles. Among 159 cases in which he examined the substance of the heart it was more or less altered in 103.

The *lungs* are found in the same state of "hypostatic pneumonia" as was described under Typhus (*supra*, p. 111).

The *liver*, again, has been described by many pathologists as undergoing a diffused change; it is soft, and on section it has a pale or "clayey" look. Under the microscope its cells are seen to be granular and disintegrating.* The *kidneys* are often of a greyish colour, and have their epithelium granular. Perhaps this causes the albuminuria which we have seen to be sometimes present.

Wagner and other German pathologists describe the frequent occurrence of *minute grey nodules*—sometimes visible to the naked eye, sometimes microscopic—in the substance of the liver and of the kidneys. Hoffmann noted their presence in thirty-eight among two hundred and fifty cases. A similar observation was once made at Guy's Hospital by Dr Goodhart, in 1879, in the case of a girl, aged seventeen. If it stood alone, one might of course suppose that acute tuberculosis was accidentally developing itself at the same time, just as occurred in a child who died a few weeks later of scarlatinal nephritis; but against such a view is the fact that the lungs in the case of fever were free.

Hoffmann believed that he detected changes in the *brain*. He describes the nerve-cells in the great basal ganglia as deeply pigmented.

Lastly, he found an enormous overgrowth of cells in the acini of the salivary *glands* and of the pancreas. He says that those structures feel unusually hard and dense.

In cases which recover, all these various lesions are doubtless repaired.

* I doubt whether this can account for jaundice in those very exceptional cases in which it is present. It is worthy of remark that the occurrence in the gall-bladder of a colourless mucus, or of a muco-purulent fluid, after an acute illness, is not to be taken as showing that there has been a deficient secretion of bile in the liver. Probably it only indicates that for several days before death there was no storage of bile, in consequence of the very short intervals at which food was taken.—C. H. F.

The process of regeneration of muscular fibres has been traced, and according to Hoffmann the liver-cells are reproduced by proliferation, of which there is evidence in the great excess of nuclei which these cells contain when death happens to occur at advanced stages of the disease.

Ætiology.—It is now generally admitted, after years of controversy, that Enteric Fever is specific in its origin as well as in its course, *i. e.* that it is always the result of infection from a preceding case of the disease, and that it always "breeds true." The phenomena of contagion and incubation, of pyrexia running a definite course, and of subsequent protection by sterilisation of the organism—all agree with those seen in other specific febrile diseases, and lead by analogy to the belief that here also we have to deal with the effects of invasion, and multiplication of a *contagium vivum*, and probably of a microphyte belonging to the group of schizomycetes. But hitherto the demonstration has not been complete. Various bacilli have been discovered in the intestines of those who have died of Enteric Fever, and have been described by Recklinghausen (1871), Klebs (1880), Klein (1880), Eberth (1883), and Friedländer as the typhoid microbe. But none of these claims satisfied even the preliminary criteria stated above (p. 13). More recently, however, Koch noticed and Gaffky has investigated* the occurrence of what is said to be a distinct rod-like microphyte, short and thick, distinct in its size, form, reactions, and the presence of spores; and this has been identified not only in the characteristic lesions of the ileum but also in the mesenteric glands, in the spleen, and elsewhere in the tissues. It has also been reported as occasionally present in successful cultivations from the urine and the fæces. Its presence in the blood appears to be less easy to demonstrate, but it is reported as occasionally present after death in the portal blood (Gaffky), and during life in that obtained by pricking the rose-papules, in rather more than half the experiments, by Neuhaus, by Rütimeyer, and by Fränkel, and Simmonds (quoted by Eichhorst). Attempts to inoculate various animals with a pure cultivation of this bacillus have hitherto failed.

Conveyance of the disease.—Enteric Fever has sometimes been said to be an *endemic* malady, but although it may prevail in certain districts rather than others, yet its diffusion is not, like that of ague, independent of the intercourse of human beings.

Among the most famous examples of *direct contagion* are those recorded by Dr William Budd, in his well-known paper on the epidemic in and near North Tawton, Devonshire, during the autumn of 1839. Three persons left that village after having taken the fever. Two of them went to Morchard and gave it, one to his two children, the other to a friend, and he again to his two children and to his brother. The third went to Chaffcombe, seven miles off, where ten others were attacked in turn, and two of these carried the disease to fresh places, with the result that several more cases occurred at each of them.

In 1875 Sir William Jenner, in his Presidential Address to the Clinical Society, stated that he had twice known enteric fever contracted by students who diligently took temperatures before the registering thermometer was in use, so that they many times a day put their heads almost into the beds of patients suffering under the disease. In 1871 Dr Collie, of the Homerton Fever Hospital, expressed his belief that certain cases

* 'Mitth. aus. d. k. Gesundheitsamte,' 1881–82, and Syd. Soc. Collected Papers, 1884.

among the attendants at that institution were caused by direct infection, either from the freshly-passed evacuations of patients or from their lungs or skin.

But striking as such occurrences naturally appear to the observer who watches their progress, there are strong grounds for rejecting this interpretation of them. Murchison tells us that during nine years, from 1861 to 1870, cases of enteric fever were treated in the same wards of the London Fever Hospital with various non-specific febrile complaints, to the number of 3555 of the former class, and 5144 of the latter. The same night-chairs were used by both sets of patients, and the employment of disinfectants was exceptional. Yet enteric fever was not contracted by one of those who were under treatment for other diseases. In the 'British Medical Journal' for 1879 Dr Shirley Murphy has brought down to 1878 the experience of this hospital in regard to the occurrence of enteric fever among the nurses and other attendants. During twenty-four years only nineteen persons engaged in the institution were attacked by it. "Of these, ten were in no way connected with the enteric-fever patients or the enteric-fever wards. Of the other nine, one was a laundry-maid, whose duties would bring her into contact with the soiled linen of the patients, but who otherwise was not in contact with them. Of the remaining eight there were special circumstances connected with drainage which would probably account for fever, leaving four for whose attacks there was no explanation given." In this time 5569 patients with enteric fever passed through the wards. Is it conceivable that if the disease were directly infectious there would not have been more numerous instances of its spreading? Murchison cites a few instances in which nurses contracted enteric fever immediately after attending upon patients suffering from it; but the fact is not even presumptive evidence of direct infection if the patient remains in the place where he was attacked. So, again, the circumstance that several inmates of a house are affected in succession proves nothing, unless the first case was introduced from elsewhere.

Dr Budd maintained as early as 1856 that the intestinal discharges of enteric fever were incomparably more contagious than the breath or other excreta of the patient. He drew an analogy between the specific cutaneous eruption and the lesions of the intestinal follicles. This doctrine had been previously taught at Munich by von Gietl, and to express this very relation the word *enanthem* had already been coined in Germany as a correlative to the term *exanthem*. Further, it is now supposed that fresh typhoid stools do not contain the poison which is afterwards developed in them. We shall hereafter see that the same thing is true of cholera; in that disease the "rice-water" discharges are probably innocuous when first voided, and become virulent subsequently. Dr Cayley, in his 'Croonian Lectures' for 1880, expresses his belief that this change may occur within twelve hours; for in the Middlesex Hospital patients have apparently caught enteric fever from using closets in which pans were placed containing stools from other cases set apart for inspection. Probably the development of the poison may occur even in fæcal matters smeared upon linen or sheets, for washerwomen have often been observed to take the disease after washing the clothes and bedding of patients. Biermer, in one of the 'Clinical Lectures' published by Volkmann, says that he has met with several instances of this. Cayley mentions two cases of enterica which attacked patients in the wards of the Middlesex Hospital, and which were traced to dried discharges upon the bedding of a typhoid patient in a neighbouring bed. Murchison relates the case of a woman who brought to her house in Warbstowe, on the Cornish

moors, the bedding of a sister who had died of enteric fever at Cardiff, in Wales. She remained free, but her sister, who was employed in hanging out the clothes, took the disease; and it spread from her as a centre.*

That the contagion of enterica is usually conveyed by the "typhoid stools" and thus by sewage there is every reason to believe. If it be the bacillus, it may germinate and multiply outside the human body, and there is no apparent reason why it should not survive, under favourable conditions, for an indefinite period. What, again, is more likely than that the living organism which produces enteric fever should sometimes remain for years in a dormant state, multiplying itself just enough to escape extinction; and that then, when more favourable conditions supervene, it should suddenly undergo immense development? Such an interpretation alone seems to apply to a fact which Murchison himself adduces; namely, that he has seen single cases of enteric fever arise in the same house again and again at intervals of a year or longer. Thus between 1849 and 1857 six cases were admitted from a certain house into the London Fever Hospital; one in June, 1849, one in October, 1851, one in February, 1854, one in November, 1855, one in November, 1856, one in July, 1857. It is unlikely that the disease was six times generated *de novo* in a single building during eight years, or that its specific poison was six times introduced from without. But if the poison was there all along, it may well have undergone excessive development from time to time when the conditions were especially favourable.

Accepting the evidence that the *contagium vivum* of enterica leaves the body in the stools and is thus conveyed to sewers, the question remains how it gains an entrance into the human body, and this is of great practical moment.

The virus may be transported by the air. Many instances have been recorded in which the disease was caused by exhalations from drains, or sewers, or water-closets, but probably this mode of infection is exceptional.

It may be conveyed by drinking-water. Such is the origin of the small epidemics that frequently occur among the inhabitants of a village, who derive their water-supply directly from one or more surface wells, into which sewage finds its way through a porous soil.

At Wicken Bonant, in Essex, the disease prevailed in 1869, and Dr Buchanan investigated its origin for the Privy Council Office. He found that there was a broad division among the people in regard to the sources from which they obtained their water. One hundred and eighteen persons used private wells, and among them there was only one positive case of fever; eighty-eight drank the water of one well called the "parish well," and no fewer than forty of them were attacked. Now, this well was situated four or five paces distant from a brook-channel which ran through the place. At the upper end of the village the brook always contained water; but lower down the channel was dry during the greater part of the year, the water being carried beneath the surface in a stratum of gravel, to reappear as a stream at the bottom of the village. That there was a direct communi-

* Murchison, although he agreed in teaching that the stools of patients suffering under the disease are at first incapable of propagating it, and become so only when they have undergone a change, supposed that the change in question is not the development of a specific microbe, but a chemical decomposition. He held that it was possible for the intestinal discharges of persons not affected with enteric fever to be decomposed in a precisely similar manner, and so to give rise to the disease *de novo*. Indeed, he proposed the name of *pythogenic fever* ($\pi\acute{\upsilon}\theta o\mu\alpha\iota$=*putresco*) in place of the objectionable term "typhoid fever."

cation between it and the parish well was evident from the fact that in times of flood, when the channel was full, the water in the well ran to a corresponding height and became discoloured. On June 24th the first case of fever occurred in the person of a boy, who lived in a cottage about thirty-five yards above the well. He had much diarrhœa, and his stools were thrown, without being disinfected, into a privy which stood almost on the edge of the channel. At this very time the soil water in the village was falling, after abundant rains which had taken place a month before, and pools of water were to be seen here and there in the channel. A month after the boy's illness the persons who made use of the well began to fall ill with the fever. Can it be doubted that the well-water had become impregnated with the specific poison? Previously, on May 30th, two cases of fever had been imported from London into a house, of which the sewer opened into the brook two hundred and fifty yards above the well.

At Page Green, in the parish of Tottenham, a great many cases of enteric fever occurred in 1864 and 1865. Dr Seaton investigated the matter and found that whereas there was to some houses a supply of water from the works of the Local Board of Health, the occupants of many other houses drank water from shallow surface wells. In three instances, in consequence of the families having removed from the place, he could not learn from which source the drinking-water had been taken, but in all other cases, with the single exception of one child, it was ascertained that those who were attacked had used well-water. Some of them had had the water the Local Board distributed to their houses, but had been in the habit of borrowing water from their neighbours' wells, because it was bright and pleasant, whereas the other was hard, turbid, and red, from rust in the pipes. When, however, the well-waters were analysed by Prof. W. A. Miller, he declared them to be quite unfit for dietetic purposes.

At Terling, in Essex, between the beginning of December, 1867, and the end of February, 1868, there occurred an epidemic of enteric fever, upon which Dr Thorne reported. It was of extraordinary severity: amongst a population of nine hundred persons, at least two hundred and sixty were attacked during the first two months; there were in all forty-one deaths, and so panic-stricken was the village, that it was necessary to discontinue the tolling of the church bell at deaths or funerals. Whether the disease was introduced from elsewhere could not be ascertained, for there had been isolated cases during previous years. But the extension of it was clearly traced to contamination of the drinking-water by sewage. The cottages were supplied, singly or in groups, by shallow surface wells, sunk in a loose and porous gravel. Round about them, but at a higher level, there were numbers of manure-heaps, cesspools, and privies, the ordure from which was often spread out for yards over adjacent fields. During the autumn the water in the wells had been unusually low; doubtless, therefore, the filth had accumulated in the soil; so that it was washed into the wells in very large quantities, when, towards the end of November, a sudden rise of water took place. Among seventy-one persons, living on the outskirts of Terling, who procured their water from ponds, only six cases of fever occurred.

In Caterham, during the fortnight which ended February 2nd, 1879, there occurred forty-seven cases of enteric fever; and, at the same time, no fewer than one hundred and thirty-two cases were observed at Redhill, eight miles distant. In each town the persons attacked were using the water

furnished by the Caterham Waterworks Company, but considerable numbers of persons who derived their supply from other sources escaped altogether. On the other hand, cases occurred at the Earlswood Asylum and in other places which also used the Company's water. Now, this water is drawn from chalk wells more than 500 feet deep, and it had a deservedly high reputation for wholesomeness. Every point in regard to its sources, storage, and distribution was carefully inquired into, but for a long time in vain. At last, however, the attention of Dr Thorne was drawn to the fact that in January, 1879, the Company had been constructing an adit, at a depth of 455 feet from one of their old wells, to a new bore which was then being sunk. A number of men had been employed upon this duty, and one of them, it was found, had been ill and had left work in the course of the month. He was sought out, and on inquiring it appeared clear that he had been suffering from a mild attack of enteric fever, which began on January 5th, and which he had probably acquired at Croydon, where he had spent December 25th and 26th. He had much diarrhœa, the bowels acting at least two or three times during each shift of eight or twelve hours, and in accordance with the usual practice under such circumstances he made use of the buckets by which the excavated chalk was being raised to the surface. He denied that he had ever relieved himself in the adit without waiting for a bucket, but it seems to be almost certain that in some way his fæces passed into the water of the well in which he was working and gave rise to the epidemic. The poison must have been diluted to an extraordinary degree.

At Caius College, Cambridge, a local outbreak of the disease occurred in November, 1873, which was traced by Dr Buchanan, with very strong probability, to a precisely similar origin. Twelve out of fifteen cases in students at the college were among the sixty-three residents in Tree Court, a part of the building which had been erected only four years previously with every care as to sewers, drains, and water-pipes. Now, Tree Court had an independent water supply direct from a high-pressure main. This supply was intended to be constant, but there had in fact been a complete intermission of it on two occasions shortly before the outbreak. After such intermissions the water had been noticed to come in with a rush, like "soda-water," evidently in consequence of its having become mixed with air, which had been sucked up into the pipes. Within the Tree Court buildings there were two water-closets, one in the basement of the porter's lodge, the other on the first floor of one of the staircases. The tap of the lower one, or that over an adjoining sink, if left open during the intermission of water supply from the main, would have allowed water to drain from the whole pipe system of the court; that of the upper one would under such circumstances have permitted of the free entrance of air. This air, however, would have been mixed with sewer-gas from an unventilated sewer in Trinity Street, which, at the very time under consideration, was receiving the excreta of patients ill with fever in other parts of the town. The effect of recharging the pipes with water must necessarily have been to distribute sewer-gas in solution to every part of the building. It was, indeed, positively ascertained that not merely air, but water impregnated with fæcal matter, had been sucked up into the supply-pipe of the upper water-closet, for that pipe was lined with a brownish deposit, containing phosphoric acid and a large proportion of intermixed organic matter.

Dr George Turner, speaking from his experience as an Officer of Health,

remarks :—"In the country amongst the cottagers Typhoid Fever appears to spread almost like Measles or Scarlet Fever. A patient returns home ill from the town, and her relations become infected. This is because usually the supply of linen is short; sheets, &c., when stained by excreta, are not immediately changed, and the air of the dwelling, usually a small one, is polluted; but it is more frequently brought about by a scarcity of washing utensils; the same bucket takes the water which has been used for washing the patient's clothes to the privy or cesspit, and then serves for a fresh supply of water, often of drinking-water. I have seen typhoid spread in a village because the family just infected dipped the bucket which had been used for taking away slop-water into the common dipping well, and thus polluted the whole water supply."

Milk may be contaminated with the enteric contagion.—This is clearly shown by the following remarkable instances.

The first epidemic that was traced to such an origin occurred in Islington in 1870. It was investigated by Dr Ballard. Between July 3rd and September 10th the occupants of sixty-seven houses were attacked, one hundred and sixty-seven individuals, of whom twenty-five died. It was a most remarkable circumstance that the district affected was included in a semicircle, with a radius of a quarter of a mile, drawn immediately on the north side of the line of the North London Railway from a centre almost upon this line. There was no fever in the area contained in the corresponding semicircle south of the railway, which here passes through a cutting. This, of course, at once suggested that human intercourse was in some way concerned in spreading the disease. The right clue was first hit upon by a lady, whose family was attacked; and a little inquiry convinced Dr Ballard that, far-fetched as such an idea had appeared, there was much probability in it. The milk vendor whose milk was suspected had himself fallen a victim to the epidemic, but his father, greatly to his credit, readily consented when applied to to give a list of the customers. It was then found that the dairy supplied one hundred and forty-two families, a very small proportion of those who lived within the semicircle. In no less than seventy among the hundred and forty-two families there had been cases of enteric fever. The way in which the disease picked out the customers of the dairy in particular streets and rows was most striking. In one long road and a street running from it the milkman supplied three families; two of them were affected. In a crescent with twenty-five houses he supplied four families; they were all attacked. In a new neighbourhood, where there were about seventy houses, he supplied four families; three had the disease. In a square with fifty-nine houses he supplied four families; all had it. On the other hand, there were scarcely any cases among those families who had invariably bought their milk from other sources. As might have been expected, women and children were attacked in much larger numbers than men. The source of infection was traced, with much probability, to the water of an underground tank in the cowyard. It was not proved that water from the tank had been used to dilute the milk, but the pails were washed out with it, and some might have been left in by accident.

In the summer of 1873 an outbreak of enteric fever occurred in St Marylebone, and in certain parts of St George's (Hanover Square) and of Paddington. It affected among others the family of Dr Murchison, who quickly became convinced that the only probable mode of introduction of the disease into his house was by the milk supply. This was confirmed by

a minute investigation made by Mr Netten Radcliffe and Mr Power. It was shown that nine tenths of the two hundred and forty-four cases to which the inquiry extended were in households which consumed milk from a particular service of a particular dairy. Certain ramifications of the same milk supply extended to the east end of Regent's Park, to Belsize Park (Hampstead), and to St Anne's (Soho), and in these districts also enteric fever occurred among the consumers. There was a special incidence of the disease upon women and children, and many striking instances are noted in which those members of a family were attacked who were in the habit of drinking milk, while others escaped who did not do so. The milk which appeared to convey the poison was a special kind, sold as "nursery milk," and taken from three or four cows, set apart for the purpose at Chilton Grove Farm, in Buckinghamshire. Now, on the 8th of June the occupier of this farm had died in the fourth week of an attack of enteric fever. His evacuations, instead of being thrown into the common privy, were buried in an ash-heap outside the farm buildings. Subsequently, however, it turned out that this was the very worst thing that could have been done with them. For there was a well close by, the water of which was used for dairy purposes, although not for drinking or cooking, as it had been noticed to have a disagreeable taste. Excavations made for the purpose showed that there had been a line of soakage into the well, along the foundations of a wall, of the filth from a pigsty which formed a pool in immediate proximity to the ash-heap above mentioned. If due intervals are allowed for the gradual penetration of the matters containing the poison of enteric fever through the soil, and for the incubation of the disease, the date at which the outbreak in London began—during the last days of June and the first days of July—corresponds exactly with this theory of its origin.

It is possible that meat may under certain circumstances convey the contagion.—At Kloten, near Zurich, six hundred and sixty-eight persons were attacked in July, 1878, all of whom had partaken of some veal provided for a festival of the choral societies, and derived from various sources, but partly from two diseased calves. A few other outbreaks, also attributed to diseased meat, are referred to by Dr Cayley. It is, however, doubtful whether the lower animals are liable to enteric fever (cf. *supra*, p. 136). The so-called typhoid fever of pigs is an entirely different affection.

Finally, it must not be supposed that it is always, or even generally, possible to trace to their source isolated cases of enteric fever, especially when they occur in large towns like London. Probably there are many chances of infection, from sewer-gas in air, and from impurities in water, which no inquirer, however acute, could trace out. The number of cases admitted into the London Fever Hospital varies from year to year much less than might have been expected. Dr Cayley believes that they are not dependent upon the presence of the contagion of the disease in water taken from the Thames; for although there can be no doubt that the poison frequently passes into the upper part of the river from the towns and villages on its banks, he thinks it is always destroyed by exposure to the air and by other agencies, among which vegetable life is probably one of the most important.

Predisposing causes.—Buhl, of Munich, applied the observations of von Pettenkofer on the relation of the soil-water to cholera to the case of enteric fever; he showed that when the soil-water in that city (as measured by the depth of water in the surface wells) is falling, the number of cases of enteric fever increases; when it is rising, the number of cases diminishes. That the

facts are so is generally admitted, but Buhl's interpretation is inadmissible. It was that the falling of the soil-water enables air to penetrate more deeply into the ground than before, and so brings about changes in the organic matters there which result in the giving off of a pathogenic poison. But this is inconsistent with everything that is known of the way in which enteric fever spreads; and no doubt Liebermeister and Buchanan are right in supposing that the observations on soil-water simply illustrate its communication by means of drinking-water. It must be added that in no other place but Munich has a fixed relation been found between the soil-water and the spread of enteric fever. In the case of particular epidemics, as notably at Terling in 1868, the disease has broken out with great severity precisely when the wells have been high.

It is certain that *climatic influences* affect the prevalence of enteric fever. In the London Fever Hospital there have been far more admissions during dry and hot summers (*e. g.* 1865, 1866, 1868, 1870) than in damp and cold summers (*e. g.* 1860, 1872). Each year there is an increase of the disease during the four autumn months from August to November, while it is less frequent from March to May inclusive. Similar observations have been made in Berlin and in Basle, and there can be no doubt that the cause is the heat of summer on the one hand, and the cold of winter on the other; the effect being, however, not immediate, but retarded by two or three months.*

In Australia, as in the United States, enteric fever is most common in the late autumn and after a hot summer. In Victoria (January being mid-summer) the disease is most prevalent in March and least so in November.†

All such conditions play but a secondary part in the ætiology of the disease; their effect is merely to favour, or to hinder, the operation of its real cause.

Age, &c.—Certain circumstances, however, remain to be stated, which affect the power of individuals to resist the contagion of enteric fever. Chief among these is age. The disease is far more frequent in persons between fifteen and thirty than in those who are either younger or older. Under two years old it is very uncommon, but in 1864 Murchison showed at the Pathological Society the intestines of an infant six months old who had been attacked at the same time with her mother. In the last ten years, ending 1888, we have had in our wards 7 cases of enteric fever in patients between three and five years old, 103 between five and fifteen, 241 between fifteen and thirty, 42 between thirty and forty, and 13 above forty years of age. The youngest patient was three and a half years old, and he recovered; the oldest was fifty-four, and he died.

During childhood the liability to the disease increases from year to year, probably owing to increased exposure to contagion. After the age of twenty the liability begins to decline, after thirty more rapidly, and beyond forty very few cases occur.‡ However, Dr Wilks once found enteric ulcers in

* At Munich, however, the maximum prevalence of enteric fever is in February, much too late for even the deeper layers of the soil to retain the heat of autumn.

† From a table given at a meeting of the Intercolonial Medical Congress on January 11th, 1889, by Dr J. G. Carstairs, of Geelong, Victoria, it appears that in Melbourne the mortality is least in November; it rises in December, to attain a maximum in March; falls very slowly in April and May, and has a sudden decline in June. The hottest month in Melbourne is January, when the mean temperature of the air averages 66·2° F.; so that, as with us, the maximum mortality from typhoid obtains about two months after the hottest season of the year is passed. In Melbourne, too, an excess of summer heat is followed by excessive prevalence of the fever.

‡ The proportion of cases over 40 in the three continued fevers observed at the London

the ileum of an old woman of seventy. Liebermeister gives a tabular statement of the proportion of cases at varying ages at Basle, corrected according to the numbers of persons at the corresponding ages in the population generally. This consideration must not be forgotten, nor yet the fact of protection acquired by having already passed through the disease in youth. But it may be that the atrophy of the lymphatic organs in old age, including those of the ileum, may be an anatomical condition which is unfavourable to the reception and multiplication of the enteric microbe.

There does not appear to be any constant predominance of one sex over the other among patients suffering from enteric fever. Among our 415 patients at Guy's Hospital there were 272 male to 143 female, but this is not a constant ratio. There seems to be a certain degree of immunity among women in pregnancy, after labour, and during lactation.

French writers have stated that students, servants, and foreigners are especially liable to contract this disease when they first come to live in Paris; and Murchison has shown that more than 6 per cent. of the patients admitted into the London Fever Hospital have arrived in London within three months.

Prognosis.—The forecast in enterica depends partly upon the condition of the patient before its invasion, partly upon the symptoms which gradually develop themselves as each case goes on, and partly on early and judicious treatment.

The general prognosis with respect to *age* is, as in typhus and small-pox, most favourable for children above infancy and for young adults; least so for elderly patients. But Murchison's statistics show that the mortality does not vary at different periods of life to the same extent as in typhus; and even in old people it is not much more than twice as high as in young adults. The disease, however, is comparatively rare above forty, and not common under five. Almost all the very slight cases occur in children. At Guy's Hospital the mortality under five was 1 in 7, too small a number to afford a guide. Between five and fifteen it was rather less than 12 per cent., between fifteen and thirty a little more than 16 per cent., between thirty and forty a little more than 26 per cent., and above forty nearly 50 per cent.

Enteric fever is very dangerous in drunkards, in those who are very fat, and in those who are affected with Bright's disease. Murchison believed that the death-rate is not augmented by a state of poverty, and that it is probably greater among the upper classes. In pregnant women abortion or miscarriage almost always occurs; but as a rule the patient recovers.

The prognosis afforded by symptoms is exceedingly liable to be unexpectedly altered by the supervention of some grave complication. So that one cannot too strongly insist that no case, however mild, is altogether free from danger. Apart from complications, the death-rate in young adults is found to be greater in proportion as the maximum temperature—usually an evening temperature—is higher, and by the end of the first week the fever has generally reached its highest point, or nearly so. Still more important are observations on morning temperatures; the more marked the morning

Fever Hospital was as follows. Out of upwards of 3000 cases of typhus, nearly 74 per cent. were under, and more than 26 per cent. over 40; out of 437 cases of relapsing fever the corresponding numbers were 82 and 17·5, and out of 1772 cases of enterica 95 and 5.

remissions the more favourable the case. A considerable fall is a good sign, unless it is due to hæmorrhage or perforation. Fiedler found that the disease proved fatal to more than half of those patients in whom the temperature in the morning reached 105·4°, and to every one, with a single exception, in whom it reached 106·25°.

But individuals differ in the extent to which a continued high temperature is injurious, and the best measures of such difference are afforded by the state of the pulse and by the degree of mental disturbance. Liebermeister put together the cases that had occurred at the Basle Hospital, and shows that, the average mortality being 16 or 17 per cent., no fewer than 40 per cent. of those patients died when the pulse reached 120, and 80 per cent. of those in whom it exceeded 140. He also arranged his cases according to the intensity of the delirium and coma, and corresponding variations were found in the death-rate.

Next to the general severity of the febrile process as shown by the temperature, the pulse, and the nervous system, we have regard to the condition of the *heart* and of the *lungs* in forming a prognosis. A feeble pulse and faint systolic sound, and very rapid breathing with cyanosis, are both grave symptoms.

Severity of the *diarrhœa* is also an unfavourable symptom, and soon tells upon the strength. But some of the cases which are entirely without this symptom are far from favourable, and Sir William Jenner has said that he prefers moderate diarrhœa to constipation in enteric fever. Extreme tympanites has long been justly regarded as a very unfavourable sign.

The significance of *hæmorrhage* has been already discussed: there is no question that large or repeated bleeding from the bowels is a very grave symptom, not only in itself, but as indicating deep ulceration.

Of all symptoms the signs of *perforation* are the gravest.

Treatment.—The first and one of the most important points is that the patient from the very beginning of the fever should be kept at complete rest in bed. Not the least advantage of the habitual use of the thermometer in daily practice is that the discovery of pyrexia in what seems a trifling disorder gives ground for insisting that no work shall be done until the temperature has fallen to the normal level. Men are apt to do themselves irreparable injury by struggling on day after day, even if they do not try to "walk off" their illness, or go away for a change of air and a holiday, when they ought to be lying quietly in bed. The members of our profession show no more judgment than others in this respect; Liebermeister has known medical men go on seeing patients after having themselves noted their temperature at 104° on the previous evening. Sir William Jenner, speaking at Birmingham in 1879, declared ('Lancet,' 1879, ii, p. 715) that some of the worst cases of enteric fever which he had ever seen appeared to owe their gravity to the circumstance that the patient had travelled, after having begun to feel ill, in order to reach his home. He added that he scarcely ever allowed a patient to be removed from the place where he was seized with the disease, if his residence was at a distance.

The sick room should be large and airy, and provided with a fire or with a door opening into another room, so that it can be well ventilated without draughts. The danger of a fever patient catching cold is probably much exaggerated.

Diet.—The food throughout the whole course of the disease should be

entirely liquid. The staple ought to be milk, of which two, three, or four pints may be supplied each day; it should be given in regular portions every two hours, or every hour, or even oftener; as the case goes on it soon becomes necessary to persevere with the feeding systematically throughout the night. If curdled milk is rejected by the stomach, or if it appears undigested in the evacuations, it should be boiled, or lime-water may be added in the proportion of one part in three, or the milk may be diluted with barley water; and if it still disagrees, it should be peptonised with *liquor pancreaticus*, which prevents a curd forming.

Other articles of food which may be allowed are beef-tea, skimmed mutton broth, blancmange (made with isinglass and milk or cream), custard, and calf's-foot jelly. Beef-tea is apt to cause increased diarrhœa. It is therefore well to avoid the routine use of broth or extracts made from beef. Veal-tea and chicken broth are less likely to act on the bowels. When there is no diarrhœa, but constipation, beef-tea is rather indicated than not, and its valuable effect on the heart should not be lost. Jenner advises that some strong essence of vegetables should be added to the broths. He permits a little strained fruit juice to be taken, but does not let his patient suck grapes because of the chance of the seeds slipping down the throat; even particles of the pulp of fruits may cause irritation of the bowel.

Pure water may be taken without stint. Toast and water, or lemonade, or barley water with lemons may be preferred, or red or black currant jelly in water, or strained tamarind tea; but before many days the tongue becomes furred and dry, the patient can taste nothing, and cold water is the most grateful drink. Iced water generally quenches the thirst less. Ice to suck can do no harm, but it often rather aggravates than relieves the parched mouth and throat. Painting the tongue with weak glycerine is a better remedy, and the lips should be well smeared with cold cream or vaseline so as if possible to prevent painful fissures.

Stimulants.—The rules for the administration of alcohol in enteric fever in which most experienced physicians are agreed are as follows. Young and healthy patients may go through well-marked attacks with perfect safety when no stimulants are administered throughout. When, however, the pulse becomes very weak or irregular, when the first sound of the heart becomes faint, or when there is great prostration shown by sinking in bed and sluggish reflex action, by the motions passed unconsciously, the eyes half open, the attention scarcely to be roused—then, whatever the age of the patient, stimulants must not be withheld. Most patients above twenty or twenty-five years of age will need alcohol before the disease has run its course, but even here it is generally better not to begin its administration at once, but to wait for indications in the circulation, the breathing, or the general condition. All cases of fever in patients above forty are grave, and stimulants should be freely given from the first.

When the condition of the circulation, or pulmonary congestion, or general depression of the patient's powers demands alcohol, it should be given every two hours or every hour, sometimes for a short time more frequently still. Children generally take brandy better than any other form of stimulant, diluted and sweetened. Young adults often do better with wine, especially red wine, such as burgundy and port. Elderly patients are sometimes wonderfully benefited by champagne. The egg and brandy mixture of the Pharmacopœia is a valuable medicine, and sometimes taken when other forms of stimulant are refused. Whatever form is found best

suited to the case, it should be given as a medicine, *i. e.* in definite doses and at definite intervals.

Drugs.—Almost all observers are agreed that no method of treatment has yet been discovered by which the course of enteric fever can be shortened, but there is no absurdity in supposing this to be possible. We have good reason to believe that the disease sometimes spontaneously aborts at the end of the second week, and the number of cases in which this occurs may possibly be increased. Liebermeister is disposed to believe that by three or four doses of from eight to ten grains of calomel, given within twenty-four hours at an early period of the fever, he succeeded in certain cases in cutting short the disease, and this treatment is much followed in Germany. Antipyretics, antiseptics, and other remedies are more generally used there than in England, but here also we hear from time to time of new methods of specific treatment, which, as a rule, turn out on trial to be either useless or harmful.

At present the most rational and successful treatment of this, as of most other fevers, is to help the patient by rest, suitable food and good nursing; to watch carefully and intelligently, and to interfere when complications arise but not before.

Treatment of the pyrexia.—Of late years the most disputed question has been whether it is advantageous to keep down the temperature systematically by *cold baths*. A mass of evidence has been accumulated in support of this practice, which had fallen altogether into neglect, although it had been advocated and successfully practised by Currie, of Liverpool, nearly a century ago. The revival of the treatment was due first to Brand, of Stettin, in 1861, and secondly to Jürgensen, of Kiel, in 1866. Since that time it has been widely adopted both in Germany and in England; here no one has studied it more carefully than Dr Cayley, of the London Fever Hospital, who recorded his experience of it in the Croonian Lectures for 1880.

The fundamental proposition upon which the antipyretic method of treating enteric fever is based is that pyrexia is harmful, because the tissues, and particularly the heart, are injured by exposure to a high temperature, especially when it is continuous and protracted. Those who recommend this practice appeal not merely to the vague impressions derived directly from the observation of cases, but to a numerical analysis of the results which they have attained.*

Notwithstanding, however, the weight which the judgment of those who advocate systematic baths in enteric fever justly carries, many are unconvinced of its advantages. Statistics of results are much open to fallacies. Some epidemics are very slight, others very severe. A case early treated and carefully nursed and fed, will do well, when one not seen until the second week will die. The dependence of mortality upon the patient's age is so remarkable that we can only fairly compare those of about the same time of life. The two most formidable complications, hæmorrhage and perforation, may at once change a favourable into a hopeless prognosis. Lastly, there is no acute disease which varies so greatly in symptoms, severity, and reaction to treatment, as enteric fever.

At present, therefore, it seems desirable (in accordance with the experience of Dr Alex. Collie and many other experienced physicians) to employ means of reducing the temperature, not as a routine method of treating the

* See, for example, statistics of mortality in enteric fever, before and after the treatment by baths was introduced, at Berlin, at Hamburg, and in the German army, by Senator, Goltdammer and others, 'London Med. Record,' Dec., 1886, p. 557.

disease, but as a special method of treating the symptom of high fever. It is doubtful whether moderate pyrexia—say under 103° F.—is of itself injurious, and whether its suppression, if possible, is desirable. But there is no doubt that hyperpyrexia is a dangerous condition in itself and should be dealt with energetically. At what point we should interfere cannot be absolutely laid down. A temperature of 104° in a child may be left alone, when one of 103·5° in an adult must be dealt with. A considerable evening rise which subsided well towards morning may be only watched, while pyrexia which rose high last night and scarcely remitted this morning must this afternoon be checked as soon as it begins its ascent. Moreover, the presence of delirium and restlessness, or of a very rapid pulse, shows that *for that patient* the temperature is high and must be treated, although the same or even a higher temperature unaccompanied by grave disturbance of the nervous system and the circulation may be safely left to itself.

When we have decided on bringing down the temperature, there are several ways of accomplishing it. One is to place the patient at once in a cold bath of 60° or 65° F. The shock may sometimes serve as a useful stimulus; but it is almost always better to use a tepid bath of 90° to 85° F. and rapidly cool the water with lumps of ice. The temperature is best watched by means of a thermometer *in recto*, and it must be remembered that it will most likely fall considerably after removal from the bath. Another important precaution is to give brandy immediately after, or even before, the bath, so as to stimulate the heart and further the cutaneous circulation. Currie's original plan of cold affusion is best adapted to relieving headache and delirium with pyrexia by directing a douche upon the head. The practical difficulties of a bath in the case of adult patients, and the serious disadvantage of lifting and moving them, it may be many times in a few hours, speak strongly for applying cold to the surface as they lie in bed. For this purpose the "wet pack" has been often used with good success; it is usually soothing and sedative as well as antipyretic, but is less effectual than the bath, and sometimes is resented. Another plan is placing bladders of ice in the axilla and over the great vessels of the neck and thighs, or fixing a coil of Leiter's tubes in the same regions and feeding them from a receptacle of iced water placed over the patient's bed. But in many, perhaps in most cases, the easiest and safest is also the most efficient method, namely, sponging the surface with cold water, or rubbing the trunk and limbs with pieces of ice, as the patient lies on a blanket with a waterproof sheet under it. Even when there does not appear any call for active interference, sponging the face, arms, and legs, with cold water or spirit lotion, is grateful to the patient, and is often followed by tranquil sleep.

Many *antipyretic drugs* have been recently used in enteric and other fevers, sometimes in conjunction with, sometimes instead of, what may be called 'the cold-water treatment." And certain of these have been credited with antiseptic power in addition, by which the course of the disease apart from the pyrexia is modified. Of these, quinine in large doses was at one time popular, but, except under special circumstances, it cannot be depended on to bring down hyperpyrexia, and is of doubtful value for any other purpose.*

It also is believed in this country that salicyl compounds, invaluable as they are in rheumatic fever, are probably worse than useless in pyrexia from other causes.

* "Large doses of quinine are as useless and injurious as in typhus" was Murchison's verdict.

Kairin (a chinolin compound, the hydrochlorate of ox-ethyl-chinolin hydride) was one of the first artificial antipyretics introduced. It is efficient, but the effect is evanescent, and it depresses cardiac action.

Antipyrin (or antipyrrhin)—belonging to the same chemical series, dimethyl-oxychinicin—is more lasting in its effects, but it also has to be frequently repeated, and is dangerously depressing to the heart. Moreover, it is apt to produce vomiting and sometimes rigors, while collapse may follow full doses.

Thallin (the sulphate or tartrate of tetra-hydro-para-chinanisol) is probably as efficient, or more so, and safer. But it also sometimes produces rigors, and is not entirely free from danger.

Antifebrin (an anilin not a chinolin compound, phenyl-acetamide) is said to be more useful, more agreeable, and more safe than any. It acts more slowly than kairin or antipyrin, but its effects last longer. It is efficient in small doses; but, when repeated, three grains may prove depressing, and ten grains may cause alarming collapse.

One great drawback to all such drugs is the difficulty of knowing when to give them so as to *prevent* the rise of temperature, and another is their accumulative depressing action on the heart.

Apart from special treatment of the pyrexia, or of the particular symptoms to be mentioned immediately, it seems to be grateful to most patients to take what is called "a simple febrifuge," such as ten drops of dilute hydrochloric acid in infusion of orange, of calumba, or of serpentary. It is probably a mere placebo, but there is every reason to please as well as to cure our patients.

Treatment of other complications.—The most fatal of all accidents, *perforation*, can only be relieved by opium when it has once occurred. Treatment consists in preventing it, by checking peristalsis, and by scrupulous care in feeding after as well as during the attack. In one case, however, referred to above, it appeared likely that a patient recovered from this all but fatal accident under laudanum and brandy.

Hæmorrhage should always be regarded as a serious symptom, and arrested, if possible, by gallic acid, by acetate of lead, by ergot, and above all by laudanum, or subcutaneous injection of morphia and ergotine. Turpentine was used by Graves, and is sometimes efficacious, but the great indication is to stop peristalsis by opiates. According to statistics, the practice of systematic cold baths does not appear to favour intestinal hæmorrhage in enteric fever, but the reverse.

In cases which are not cut short by perforation or hæmorrhage, the cause of death is usually *failure of the circulation* with hypostatic congestion of the lungs. Beside the free use of stimulants, as described above, great benefit is derived from the common senega and ammonia mixture, or carbonate of ammonia alone in three- to five-grain doses, with its pungency removed by treacle, syrup of tolu, or liquorice, and, if needful, laudanum or paregoric added to prevent laxative effects. With this digitalis may be given, and sometimes its effect on the rapid, feeble, and irregular pulse is striking and most useful; but often it disappoints us, and on the whole is less to be depended on in these cases than when a similar condition of the pulse is produced by valvular disease of the heart.

Diarrhœa should be checked from the first. If there are not more than two loose motions in the twenty-four hours, if the patient is young, and the fever not severe, we may wait; but as soon as this is exceeded, the

starch and opium enema of the Pharmacopœia should be administered, and repeated after each movement of the bowels. When pain and restlessness are also present, Dover's powder is probably the best form of opiate to give internally, and chalk mixture, catechu, or other astringents are often prescribed with advantage; but in most cases it is better to leave the stomach as much as possible for food and alcohol.

When there is constipation instead of diarrhœa, and especially if there is much flatulence and discomfort, a soap-and-water enema may be administered. Murchison recommends a teaspoonful of castor-oil, and repeats it every three or four days if there is constipation throughout the fever. Knowing, however, that in all cases there is some ulceration of the ileum, and that the degree of diarrhœa is no certain guide to its severity, the writer ventures to think it better to err on the side of caution, and to abstain from meddling with the bowels until convalescence is established. He has frequently seen a patient pass through the disease favourably with constipation throughout; he has twice seen such cases die from perforation, and been thankful that even an enema had not been used; and again and again he has seen the bowels act naturally and comfortably after the temperature had fallen, without aid from drugs.

No qualified man would think of giving ordinary laxatives in a case of enteric fever, but we often meet with cases in which harm has been done by saline and other purgatives being taken for a supposed "bilious attack" before the true nature of the disease has been recognised.

Tympanites is not only an unfavourable symptom but is also injurious. It may be met by turpentine stupes or by enemata containing asafœtida or other carminatives. The distension is chiefly in the colon, and therefore drugs given by the mouth are not of much service.

Bedsores ought to be prevented by extreme cleanliness, by careful drying and dusting after each evacuation, and by daily friction with brandy from the first. When the skin is already red and threatens to break, it is better to apply lead lotion frequently. Careful shifting of the patient so as to vary the points of contact as much as possible is part of a skilful nurse's duties. A water-bed is an important preventive.

Treatment during convalescence.—Stimulants should be at once diminished on the subsidence of fever, and in many cases it is desirable to substitute an ounce or two of wine twice or thrice a day for brandy at frequent intervals. With young patients, however, after a favourable attack there is often no need for stimulants or drugs. No solid food should be given for a fortnight after fever and diarrhœa have ceased. The patient will bitterly complain of the restriction, but if the physician has once lost a patient from perforation during convalescence he will be inexorable ever after. Progress to health after this disease must be slow if it is to be sure. Leaving the bed, taking meat, going downstairs and out of doors—each stage of convalescence must be carefully considered. And there is no disease, not even rheumatic fever, in which it is so important for the patient to have a long period to recover his strength before returning to his ordinary duties.

Prophylaxis.—As we have seen, there is little danger of infection being incurred by doctors or nurses in charge of a case of enterica, either because the bacilli in the fæces are not yet become toxic, or because they rarely succeed in gaining entrance except when swallowed in liquids.

Nevertheless it is desirable for the patient's stools to be disinfected, so as, if possible, to prevent contagion being conveyed into drains, and, by want of sanitary precautions, into drinking-water.

For this purpose various methods have been tried. Crude sulphate of iron placed in the bed-pan is cheap and effective; carbolic acid has also been much used, and chloride of zinc and of lime. Dr Foote, of Yale College, U.S.A., has made some careful experiments on the sterilising effects of various antiseptics, and finds that 5 per cent. solution of carbolic acid and 2 per cent. of corrosive sublimate are ineffectual. Fresh chloride of lime he finds, on the whole, the most efficient. Corrosive sublimate with excess of hydrochloric acid is very active, but injures lead pipes.

FEBRICULA.—When continued fever was no longer regarded as one "disease" with endless varieties, and when the distinction between Typhus and Enteric was established, Relapsing Fever formed a third "species," and a fourth was called Simple Continued Fever, Ephemeral Fever, or Febricula.

This was described by Jenner in his well-known Lectures, and admitted by Murchison among the 'Continued Fevers of Great Britain.' But most physicians now believe that a disease without any morbid anatomy, without any known ætiology, and without any definite or characteristic course or symptoms, cannot be admitted into a useful nosology. This was the opinion of the late Dr Tweedie. In some admirable 'Lectures on Fever' delivered by Dr Peacock, of St Thomas's Hospital, he wrote as follows:—"The cases which have been classed under this head embrace a variety of different affections. Some are probably cases of imperfectly developed typhus, typhoid, or eruptive fever; others may be cases of relapsing fever, in which the relapse does not occur; and yet others may depend on common causes—exposure to cold or damp, noxious miasms, or on gastric, intestinal or hepatic disorder. I should rather regard the so-called ephemeral fevers as abortive attacks of one or other of these forms of disease, than as possessing a specific character of their own. You will constantly find that when a series of cases occurs in members of the same family, some are characteristic attacks of typhus or typhoid, while others present only slight febrile symptoms, to which we may apply the term Febricula or Ephemera. Thus, in the three cases of typhus which I have before referred to as examples of contagion and of the varieties in the form of the eruptions on the skin, the first two, those of the patient and the first nurse, were examples of characteristic and severe typhus; the third case, or that of the second nurse affected, was merely a slight febrile attack in which no eruption appeared on the skin. Again, among the typhoid cases is included one which presented the characteristic features of the disease during life, and proved fatal, and after death extensive intestinal disease was found. The brother of this boy was under treatment at the same time, and in his case the fever was only slight; and numerous similar examples might be quoted. We see facts precisely analogous during epidemics of the eruptive fevers, and especially of scarlatina."

This opinion will probably be shared by most scientific physicians. It may have been needful to include in the College of Physicians' Nomenclature (1884) "Simple Continued Fever," with the definition, "Continued Fever, having no obvious distinguishing character;" but if obvious characters for diagnosis fail, we must seek for those which are more latent.

It must, however, be admitted that remarkable cases of fever do occur, which we must at present leave unexplained and without a name.

(1) There are cases of pyrexia and even hyperpyrexia without other signs of fever. Some of them have been referred to already (*supra*, p. 41), which occur in women between the ages of fifteen and forty-five; but others remain. In 1886 there was a patient in Philip Ward, a healthy, decent mechanic, of about forty, who for several weeks suffered (or rather did not apparently suffer) from high temperature, with the normal diurnal variations exaggerated, and sometimes reaching 105·6° F. Yet he ate and slept well, and there was no local lesion to be found. At last the temperature gradually fell, and he went out well. The case was published by Dr Hale White.

(2) Children are apt to be affected by pyrexia, and sometimes by a sharp attack of fever, from causes which in adults would cause only insignificant disturbance. Slight gastric catarrh from eating indigestible food, diarrhœa from eating raw fruit, from chill, or exposure to heat or fatigue, will be enough to cause febricula, and the diminutive will often apply to the duration only, not to the height of the pyrexia. The "infantile remittent fever" of Evanson and Maunsell is probably almost always enterica without the rash.

In young women (who retain the pathology of children much later than youths of the other sex) similar short and sharp fever is not infrequent from "gastro-entérite" due to indigestion or chills, from retention, suppression or disorder of the menses, and particularly from a degree of sore-throat which falls very far short of quinsy.

Here may be mentioned the remarkable cases of gastro-enteritis with fever which are sometimes the result of eating bad meat or fish.

(3) Slight cases of enteric fever have often been called febricula. In the absence of the rash, enlargement of the spleen is probably the best diagnostic sign. See an interesting discussion reported in the 'Dublin Journal of Medical Science' for July, October, and November, 1879, by Drs Cameron, Grimshaw and others, and a valuable paper by Prof. Bäumler of Freiburg-in-Breisgau, on the question, "Can the mildest forms of enteric fever be distinguished from acute febrile but non-specific gastro-enteric catarrh?" (*ibid.* November, 1880). See also Jurgensen's paper in 'Volkmann's Sammlung,' tr. New Syd. Soc.

(4) There remain, however, certain epidemic and probably contagious febrile disorders which cannot as yet be brought under any recognised category; they are neither continued, eruptive, malarious, nor symptomatic. The best observed instance with which the writer is acquainted is detailed with great care and judgment by Dr Seaton in the Clinical Society's 'Transactions' for 1886 (vol. xix, p. 26). No less than 157 cases occurred during the four summer months (June to September) in a boys' orphanage. The attack began suddenly with rigor and severe headache, and the temperature rose rapidly to its maximum, which varied from 101° to 105° or even 106°. There was often severe vomiting, with no diarrhœa, but with scanty urine in which the chlorides were remarkably deficient. In most cases the fever ceased in two or three days, in others on the fifth or sixth, and labial herpes usually appeared at the same time. Earache with otorrhœa and pneumonia were the two complications noticed. Death occurred in seven cases. *Post-mortem* examination threw no light on the disease, but it appears to have been only made in a single case.

MEASLES*

"We bear diseases
Which have their true names only ta'en from beasts,
As the most ulcerous wolf and swinish measle."
WEBSTER.

History and Nomenclature—Ætiology—Incubation—Onset—Course and Eruption—Varieties—Complications—Sequelæ—Protection—Diagnosis—Prognosis—Treatment.

No mention of this common and well-marked disorder is found in the ancient medical writings; but, in the ninth century, the celebrated Arabian physician Rhazes† described Smallpox and Measles in a treatise which was translated into Syriac, Greek, and Latin, and is still extant. Measles was long regarded as a minor form of smallpox.

The name *Morbilli* (Ital. = a little plague, *i. e.* compared to Variola) seems to have been constantly used until the middle of the eighteenth century, when Sauvages invented the term *Rubeola* (the red rash, from *ruber*). This name was unfortunately adopted in Great Britain by Cullen and by Willan; and their authority led to its general acceptance. But within the last few years most English writers have reverted to the use of the term *morbilli*. The English word measles means spots, as in the phrase "measly pork."

Origin and propagation.—Measles is a strictly contagious, specific, febrile disorder, and has been propagated over temperate, warm and cold climates, so that it is now almost pandemic. Its introduction into Iceland, the Faröe Islands, and the Fiji Archipelago, took place in recent times. Like smallpox and syphilis, it was much more severe and fatal when thus imported into a virgin soil. Even in England local epidemics may be observed. Sydenham described the prevalence of measles in London in the years 1671 and 1674, and Haslam at Plymouth in 1741.

The contagion, though active and sure, is not persistent, and it is readily dissipated by ventilation. It is probably conveyed by the secretion from the affected mucous membranes, by the breath, and perhaps also by the skin after the rash has appeared.

The contagium has not been isolated, and it is unknown whether it is a microphyte. Messrs Braidwood and Vacher observed minute glistening particles in the mucus of measles which resembled those seen in vaccine lymph ('Path. Trans.,' 1878, vol. xxix, p. 421). Attempts to inoculate it by the blood have failed.

If measles occur among the lower animals, it has not yet been identified.

Measles is a disease of children. Most persons above puberty have already had it; but cases occur at any age, and unprotected adults take it

* *Synonyms.*—Morbilli—Rubeola.—*Fr.* Rougeole, *Germ.* Masern, *Ital.* Rosolia fersa, *Arabic* Hasbah.

† Abubekr Mohammed ar-Razi, born at Rai (whence his surname), a town of Irak Ajemi, in Persia, about 850 A.D.

very readily and as a rule severely. The only predisposing cause for measles that we know is Whooping-cough.

Incubation.—This has been carefully observed in cases which have occurred on board ship, and in the remarkable epidemic which invaded the Faröe Islands in 1846. Panum (afterwards the eminent professor of physiology at Copenhagen) found that a period of thirteen or fourteen days elapsed between exposure and the appearance of the rash, *i. e.* the incubation period was ten or eleven days.

Further exact observations have shown that although variations occur, they are less than in most other cases of contagion, so that the disease rarely manifests itself before the tenth or after the thirteenth day. When designedly inoculated by mucus from nostril to nostril, the period of incubation is shorter, as it is with inoculated variola—eight or nine days.

During this period the child continues apparently in perfect health.

Onset.—The patient is taken suddenly ill with anorexia, headache, and malaise; vomiting may set in, or diarrhœa, or epistaxis, or a rigor, or (in very young children) a convulsive seizure; and by the evening of the first day the temperature has reached 102°, or more. Yet the course of the fever during the next two or three days is uncertain. On the second or the third day the thermometer may fall to normal; and, as the appetite may at the same time return, the idea of an impending illness is often abandoned. In other cases, however, the temperature remains at about 102°, with only slight oscillations.

There are *catarrhal symptoms* from the first; the patient is troubled with coryza, sneezing, intolerance of light; fluid secretion pours from his eyes and nose, his face and eyelids are swollen, and his conjunctivæ are injected. He is sometimes hoarse, coughs, and sibilant or sonorous rhonchi may be audible on auscultation. By the second, or at latest by the third day, one finds on looking at the fauces that besides a general injection of the soft palate, there is an eruption of scattered points and spots over its mucous membrane. Ringer attaches some importance to the presence of thin, opaque, white patches on the gums, and the inside of the lips. In the pharynx one can hardly recognise anything more than a diffused redness, and the same has been observed within the larynx by the aid of the laryngoscope. Some writers have regarded all these appearances as indications of an exanthem of the mucous membrane, and have proposed to term it an "endanthem." This appearance may be of clinical value in enabling measles to be recognised among the dark races of mankind, in whom no cutaneous exanthem is visible. This slight angina is all but constant.

The tongue is coated with white fur, through which a few red papillæ may perhaps be seen projecting. The pulse is less rapid than in scarlet fever. The respirations are but little increased until the advent of bronchitis or pneumonia. The urine not infrequently shows a trace of albumen, or more than a trace, and this does not appear to add to the gravity of the case. The faucial affection and the early catarrhal symptoms in general continue into the eruptive stage and then subside. According to Professor Thomas, of Leipzig, indications of the approaching cutaneous rash may sometimes be seen on the face during the prodromal stage, in the form of minute puncta, around which the characteristic papules afterwards develop.

Course.—The *eruptive* stage which succeeds begins generally on the fourth, but sometimes on the third day of the illness, and from the fourteenth

to the seventeenth from infection. In exceptional cases it is postponed until, according to Trousseau, six, seven, or eight days of fever have elapsed; or even, according to Watson, until the tenth day. There is now a further rise of the temperature, which in thirty-six hours (as a rule, on the sixth day) reaches its acme, this being generally 104° or 105°. Afterwards it may either begin at once to decline, or may remain at or near the same point for a day or two. The fall, when it does occur, is rapid, and the normal temperature is reached often before the end of the week, or by the eighth or the ninth evening at the latest.

Prof. Thomas seems to have studied the thermometric changes in measles with more care than any previous observer; and his conclusions accord perfectly with those of the older physicians, who laid stress on the fact that the fever does not, as in smallpox, cease, nor even abate, upon the emergence of the eruption, but sometimes increases in intensity.

Exanthem.—The rash of measles first appears on the face; Ringer says that the earliest traces of it are seen on the forehead close to the scalp. It spreads over the face (not avoiding the parts about the mouth) and also over the whole trunk; on the limbs, especially the lower limbs, it generally comes out rather less freely, but it shows no decided predilection for the flexor surfaces, and it may be well marked upon the palms and the soles. In several of these minor points it differs from the eruption of scarlet fever The rash commonly takes three days for its complete development, but sometimes not more than a few hours; the later it is in beginning, the more quickly it diffuses itself over the body. Thomas says that before it has existed at any one spot for twenty-four hours it always begins to decline, so that when it comes out slowly it fades upon the face and neck, while it has still to make its appearance over the more distant parts; but according to Watson it may remain three days at least on the face before its subsidence commences. The old doctrine that a rapid retrocession of the rash was often followed by some dangerous complication is disputed by Thomas, in common with many other modern writers. On the other hand, he says that there is sometimes a brief recurrence of the exanthem when from any accidental cause there is an exacerbation of the fever.

The colour of this rash is a more or less deep rose or crimson, inclining to purple rather than to scarlet. It consists of spots of irregular form and of varied size. They are at first isolated, but afterwards coalesce into patches, the margins of which are sharply defined, and here and there present rounded notches, or (to use the current expression) are "crescentic" in outline. They are slightly raised; even the earliest papules can be distinctly felt with the finger. They have been supposed to be enlarged normal papillæ, or, again, to be the hyperæmic mouths of sebaceous follicles; but Gustav Simon, having excised a portion of the skin from a patient affected with measles, examined it histologically with negative results. When there is much sweating, a few vesicles of miliaria are sometimes to be seen. Before it subsides, the eruption acquires a yellowish tint, which is particularly well marked when the blood-vessels are temporarily emptied by the pressure of one's finger. This is no doubt the result of the diffusion of altered blood-pigment in small quantity into the substance of the cutis; for even in cases which are doing well, it is no uncommon thing for actual hæmorrhage to occur; so that, after the rash has faded, purple stains remain, which afterwards become brown and yellow, and do not finally disappear for two or three weeks.

Slight desquamation takes place, especially from the skin of the face. No large scales are detached, but only a fine mealy powder, which (as Trousseau remarks) is often best seen when one brushes the skin of the patient with one's coat-sleeve, or (according to Ringer) when the surface is stretched and viewed sideways. Desquamation begins on the sixth or seventh day of the fever, and continues for a week or ten days.

Convalescence is established about the end of the week, unless it is delayed by complications or sequelæ. The patient continues highly contagious during that period.

Varieties.—In many cases measles runs an irregular course, and, like those of other exanthemata, its aberrant forms are of two kinds: some *benign*, being shorter and more favourable than usual; others *malignant*, attended with very grave symptoms, and ending more or less rapidly in death.

In some benign cases of measles there is a rash, but no catarrh; in others it is the rash which is absent. Of *morbilli sine catarrho*, Thomas remarks that it is especially apt to occur in very young infants, and that it is almost unattended with fever. There is reason to believe that many cases of rubeola, or "German measles," were formerly supposed to be slight measles without catarrh, and perhaps this explains Sir Thomas Watson's remark that the incomplete form of the disease confers no immunity against recurrence. Dr Eustace Smith thinks it questionable whether *morbilli sine catarrho* is measles at all.

Of *morbilli sine morbillis*, Thomas observes that this form is in all likelihood diagnosed more often than it really occurs; but surely the only cases in which one would think of suspecting its presence would be if during an epidemic an unprotected person should suffer from catarrhal symptoms without any apparent cause. Some writers have asserted that, as in the case of latent scarlet fever, doubt may ultimately be removed by the occurrence of desquamation; but this is so slight, even when the affection of the skin is intense, that one would hardly expect to see it when there has been no eruption at all.

Of the malignant varieties of measles, there is one which is attended with hæmorrhages from the mucous surfaces, and with an intensely *purpuric* form of eruption. At the present day, however, this is very infrequent; it is observed chiefly in young and sickly children. Hence, when one finds the older writers laying great stress upon "black measles," one is almost inclined to suspect them of having mistaken cases of hæmorrhagic smallpox for this disease; and it is interesting to notice that Sydenham speaks of an unusually bad kind of measles as prevailing in London in 1670 and 1674, at a time when variola also was remarkably malignant and fatal. The hæmorrhages are said sometimes to begin before the ordinary morbillous rash comes out, sometimes afterwards, and then the exanthem quickly fades or turns of a livid purple colour. Petechiæ and vibices cover the skin, while blood oozes from the mucous membranes of the nose, kidneys, and intestines, as well as into the substance of the deeper tissues and internal organs. Death generally takes place within a very few days.

In other instances measles becomes fatal by the severity and prolongation of symptoms which are not in themselves unusual. The fever is from the first intense and persistent; the rash, although it may come out early, is of a livid colour, and often develops itself very imperfectly; instead of

the temperature falling about the eighth day, it remains high throughout a second week; the pulse is very rapid and feeble, the patient becomes delirious and drowsy, and passes into a "typhoid" condition with a dry brown tongue and sordes on the lips. Death is preceded by prostration and collapse.

One of the most important clinical uses of the thermometer in measles is to prove that cases of this kind deviate from the ordinary course of measles. According to Thomas, it is no less unfavourable for the temperature to be low when the eruption is coming out than for it to continue high at a time when it should be falling.

Complications.—It is a rule, to which there seems to be scarcely an exception, that in all but the most rapid hæmorrhagic cases of measles some complication or other is really the cause of death. Even when the symptoms are not sufficiently marked to lead to its recognition during life, a local lesion is sure to be discovered at the autopsy; and in cases that recover similar affections are very frequent, and often seriously modify the symptoms.

Broncho-pneumonia, with consolidation of scattered lobules throughout the lungs, is the most frequent complication, particularly in certain epidemics; indeed, it occurs so often that some writers have been disposed to include it in the regular course of the disease. But it is not really constant. The effects of this catarrhal inflammation of the lungs are the same as when a like affection arises from other causes. In some instances lobar pneumonia is found, and sometimes, perhaps, there is capillary bronchitis, but these are exceptional. More or less extensive pulmonary collapse often follows in young children.

A dangerous but happily rare complication is the formation of a layer of plastic exudation upon the fauces or within the larynx, a secondary membranous croup or *diphtheria*, according to the view which is taken of such morbid changes.

Again, in many cases, *diarrhœa* sets in so severely as to bring about a fatal issue, especially when the evacuations assume a dysenteric character. Little more than redness of the mucous membrane of the colon is found after death from this cause.

Epistaxis is not infrequent in measles, and occasionally serious. Among the less grave complications, so far as the life of the patient is concerned, must be mentioned certain affections of the eyes and ears. *Ophthalmia* is common, and often runs on for a long time after recovery. The author once saw a diphtheritic membrane form again and again on the conjunctiva. Sometimes iritis occurs, and sometimes destructive corneitis. In other cases the morbid action extends from the fauces along the Eustachian tube, causing *deafness* and pain in the ear. As a rule, it seems merely to lead to an accumulation of mucus in the tympanic cavity: but now and then suppuration takes place, and even necrosis of part of the temporal bone. Trousseau relates one instance of this, and another has come under the author's observation.

Sequelæ.—Almost any of the complications of measles may be so prolonged as to become a sequela; but there are some other affections which have a better right to that title, since they do not begin until after the patient has recovered from the primary disease. Among these is a form of gangrene, attacking the mouth (*cancrum oris*) or the female genitalia (*noma*).

According to Thomas it does not arise spontaneously, but is preceded by some slighter lesion of the same parts, such as a decayed tooth, or infantile leucorrhœa. It is generally, but not always, fatal.

In other cases measles is followed by *necrosis* of a portion of the upper or lower jaw, or by abscesses in the neck.

Again, children often remain for many months after recovery from this malady in a state of ill-health for which no obvious cause can be found, and during this time they are very apt to be seized with bronchitis, or sometimes by lobar pneumonia, especially in the cold seasons of the year.

The swollen bronchial lymph-glands may become caseous, and thus *tuberculosis* is not unfrequently set up. Indeed, this is one of the commonest and most fatal evil results of measles. An intercurrent attack of measles is said greatly to accelerate the downward course of a pre-existing phthisis.

On the other hand, certain cutaneous diseases, such as eczema, seem often to be favourably modified by the supervention of the exanthem, at least for a time; and, according to Rilliet and Barthez, the same thing happens in some cases of epilepsy, chorea, or incontinence of urine. In 1877 we had a case of paralysis probably due to peripheral neuritis which had followed an attack of measles.

Dr Goodhart finds that whooping-cough is often a sequel of measles, more often than its predisposing cause.

Protection.—Measles often fails in preventing a second attack. Even relapses are not unknown, and second or third invasions in a lifetime are not uncommon.

Diagnosis.—This depends on the late appearance of the eruption, the coryza, and the colour, distribution, and form of the rash. Measles has been mistaken for smallpox; but the symptoms are far less severe, the papules look and feel different, and the non-development of pustules decides the point. It is more often mistaken for scarlatina, but the eruption is later; it is purple, not scarlet, blotched, not punctate, and appears first and is most marked on the face. It is often confounded with Rubeola, or German measles, to be described in a following chapter.

Prognosis.—The mortality of measles varies widely in different epidemics. It has sometimes been so low as 2 to 3 per cent. of those who have been attacked, sometimes as high as 50 per cent. Malignant cases, killing within the first week by prostration, with or without a hæmorrhagic rash, are rare—much rarer than the corresponding malignant scarlatina; and petechiæ are often seen in ordinary benign cases. Trousseau relates that in 1845 and 1846 he lost from broncho-pneumonia twenty-two out of twenty-four children with measles who were under his care in the Necker Hospital. According to Thomas the few cases which occur during the first six months of life are generally mild; but the disease is more severe in infants during dentition than in older children. Convulsions, occurring after the rash has appeared, are of ill-omen.

Among adults measles is more severe than in children. It is most dangerous to women who are pregnant, or who have recently been confined, and to the very few old people who are susceptible of the disease.

Treatment.—The general plan of treatment is that suitable for febrile diseases in general, for we have no specific method of dealing with the

malady. The patient should be confined to the house, but only kept in bed if the temperature is high or the catarrh severe. He should be lightly clothed and allowed to drink as much water as he likes.

Poultices may be applied to the chest, but with little children a cotton-wool jacket is better, since it interferes less with breathing and does not get cold or slip off.

In ordinary cases of measles no drugs are required. The child should be kept in bed if the temperature is high and given plenty to drink—cold water, lemonade, raspberry vinegar, or any other cooling beverage. If the appetite continues, as it sometimes does, light solid meals may be allowed. When the cough is troublesome, black-currant jelly or sweetened barley water slowly swallowed relieves the irritation of the fauces and thereby the cough. Or a few drops of ipecacuanha wine with ten or fifteen of paregoric, sweetened with syrup of tolu, and given every two hours, promote secretion, and thus may relieve cough and procure sleep.

If the temperature is not above 104°, aconite, thallin and other antipyretics probably do more harm than good, and cold baths are undesirable; but sponging is of comfort and use. Bronchial complications may call for ammonia, and pneumonia or collapse, or great depression with persistent high temperature and rapid pulse must be met by the exhibition of brandy. Diarrhœa is not a good symptom, and should be checked by chalk or bismuth, by withdrawing beef-tea and substituting arrowroot, with milk and lime-water. Purgatives at any stage of the malady are undesirable. In severe dyspnœa from bronchitis, laryngitis, or pneumonia, Dr West records recovery after the application of leeches or venesection. Dr Eustace Smith recommends dry cupping.

During convalescence, the chief dangers are fresh bronchitis, whooping-cough, or the supervention of caseous disease of the bronchial lymph, and consequent phthisis. To guard against these the child should be well fed, and, if needful, the appetite helped by quinine. Anæmia should be combated by steel, and want of nutrition by cod-liver oil. The body should be warmly clothed in flannel and the child allowed to be much in the open air.

It must be remembered that measles is infectious from the very beginning of the disease, so that isolation should be enforced as soon as its presence is recognised.

SCARLATINA*

"This distemper is sometimes so slight as to require no remedies, and sometimes so violent as to admit of no relief."—HEBERDEN.

History and Nomenclature—Distribution—Contagion and transference—Surgical and puerperal scarlatina—Incubation—Onset and Course—The throat—The rash—Abortive and malignant varieties; scarlatina anginosa—Complications and sequelæ—Anatomy—Prognosis — Protection — Prophylaxis—Treatment.

SCARLET FEVER was recognised and discriminated as a distinct disease by Sydenham in 1675, and the separation from measles was completed by Withering a century later (1778). It had, indeed, been described as far back as 1556 by Ingrassias under the name of Rossalia, at Naples, and again by Döring, at Breslau, in 1627; but even Morton, who was a contemporary of Sydenham, maintained that it was only a variety of measles. The distinctive name *febris scarlatina* (Ital. *scarlatto* = *scarlet*) was given by Sydenham. It is remarkable that he did not mention sore-throat as one of its symptoms. Probably the bad cases of Sc. anginosa were then called *Cynanche maligna*, which was distinguished as late as Cullen and Heberden.

From the persistence and virulence of its contagion and from the severity of its effects, this disease is justly dreaded. Since the introduction of vaccination, it has taken the place of smallpox as the most common and fatal of all the specific fevers.

Its geographical distribution is now very wide, but like smallpox and measles, relapsing fever and typhus, it has been introduced from Europe into other parts of the globe within historical times. Thus it appears to have been unknown in the colonies of North America until the year 1737, and in South America for nearly a century later (1829). It was brought to Iceland in 1827, to Greenland in 1847, and about the same time the first cases were recorded in the Australian colonies.

Ætiology.—The channels and mode of entrance of the contagium are not always the same. The dry epidermic dust is probably most often taken in with the air in breathing, or with food and drink in swallowing. It is often conveyed by clothing or other fomites.

Attempts to reproduce scarlatina in the lower animals by transference of blood or epidermis have repeatedly failed; but investigation into a remarkable epidemic at Hendon in 1885, by Dr Power and Dr Klein, appears to render it probable that Scarlatina may not only be conveyed from one person to another by means of milk, as before ascertained, but that the disease may originate in cattle, and be conveyed from them by their milk to children.

The *contagium vivum* is chiefly present in the epidermis which is shed.

* *Syn.*—Scarlet Fever—Morbilli confluentes (Morton, 1700)—Febris rubra (Heberden).—*Fr.* Scarlatine, *Germ.* Scharlach, *Ital.* Febbre Scarlattina.

It is particulate, but although several forms of micrococcus and bacillus are present in the secretions of the fauces during the disease, and have been described by McKendrick in 1872, and by Pincus in 1883, none have been certainly identified as pathogenic or even as characteristic. Dr Klein has since, he believes, succeeded in reproducing the disease in cattle ('Proc. R. S.,' March 3rd, 1887), by inoculating them with micrococci obtained by cultivation from the blood of cases of human scarlatina, and also by feeding calves with the same microphytes suspended in milk.*

The contagion of scarlatina, though active and very persistent, is not easily diffusible. Thus, it rarely crosses a street, and epidemics do not spread so quickly as those of typhus, measles, variola, or diphtheria. But they linger long and die slowly out.

Although scarlet fever never arises but by contagion, yet, as in other cases, there are conditions which predispose to receiving and developing it.

First of these is the *age* of the recipient. It is rare in infants, most common in early childhood (Murchison found 64 per cent. of cases to occur between the ages of one and five), and rare after puberty; but it may occur even in advanced age. When measles and scarlatina are both rife in a town, it has often been remarked that very few unprotected persons escape the former and many the latter.

Secondly, puerperal women are peculiarly liable to take the contagion; and, with somewhat masked features, it forms a considerable proportion of the cases known as puerperal fever. Dr Braxton Hicks found that in eighty-nine cases of this terrible disorder no fewer than thirty-seven either showed a scarlatinal rash or had been subjected to infection from patients with scarlatina.

Thirdly, persons who are suffering from wounds are extremely prone to infection. The sore-throat and fever are frequently present without the rash, or the rash is slight and quickly over. But that the affection is genuine scarlatina is proved by its "breeding true" and by its protecting against future attacks. Moreover, although most of these cases are mild, like inoculated smallpox, occasionally a severe one occurs along with the rest and exhibits the sequelæ as well as the symptoms of the disease. Sir James Paget and Mr Howse, Dr Gee, Dr Eustace Smith, Mr E. C. Stirling, and Dr Goodhart have placed the true nature of this "surgical scarlatina" beyond doubt. It is remarkable that according to the observation of the last-named author, antiseptic precautions do not prevent the infection, so that the otherwise probable supposition that the contagion gains a direct entrance through the open wound appears to be untenable.

The *season* in which Scarlatina is usually more prevalent is in the autumn months from the middle of September to mid November. This was observed by Sydenham, who wrote: "Scarlatina febris licet nullo non tempore possit incidere, ut plurimum tamen exeunte æstivo se prodit" ('Obs. Med.,' sectio vi, cap. 1).

Incubation.—This period is short compared with that of enterica, typhus, measles, or smallpox. It most commonly lasts between two and six or seven days. Occasionally the interval between infection and the first symptoms may be still shorter, twenty-four hours, or perhaps even less. Among the cases with the shortest incubation are those of puerperal and surgical scarlatina just mentioned. Although rare, instances occur in which

* See also a paper by Drs Jamieson and Eddington, with figures of the bacillus which they selected as probably pathogenetic ('Brit. Med. Journ.,' June 11th, 1887).

more than a week has elapsed between contact with a case of scarlatina and appearance of its first symptoms, and still more rarely the incubation may be as long as that of smallpox or of measles.*

Course.—The onset of the disease is generally sudden. In children the first symptom is often vomiting or a convulsive seizure. In adults it is usually soreness of the throat, and there may be chilliness or even a rigor. The patient complains of headache, malaise, and prostration.

The face quickly becomes flushed, the pulse remarkably rapid, and the skin hot. For a child's pulse to be at 140 or 160 within a few hours is not uncommon, nor of unfavourable augury; and it may remain high for several days.

The *temperature* may rise to 104° or 105° in the course of the first day or it may attain the same point or a still higher one more slowly afterwards, while the rash is coming out. It then as a rule rests stationary, or nearly so, until the rash begins to fade. The extreme dryness of the surface is apt to give one an impression that it is hotter than is really the case; from the time of Addison we have spoken of the pungent heat of the skin in scarlet fever as comparable only with that of acute pneumonia.

Dr Gee describes the fever as frequently ending in a complete crisis; this occurred in two of his cases on the fourth day, in four on the fifth, and in three on the seventh. But its fall is more often gradual, taking from three to eight days for its completion. After the fever is over the temperature is often subnormal for a day or two.

The *urine* is scanty and high coloured as in other fevers, an abundant precipitate of lithates forms as it cools, and it often contains a little albumen even at this stage. Urea is in great amount, and the proportion of potash salts is increased.

The *tongue* is at first coated with a thick creamy layer, as in other febrile diseases. But before long enlarged fungiform papillæ are seen projecting as shining scarlet points, and after two or three days the white fur clears away from before backwards, leaving a smooth bright red surface; this, with the little prominences that are thickly scattered over it, has an appearance that has been compared with that of a strawberry. Sometimes, however, no such enlargement of the fungiform papillæ occurs. German writers speak of a miliary vesicular eruption as occasionally present, especially on the dorsal surface. The tongue does not regain its normal aspect until convalescence has set it.

On looking at the *fauces* one finds the arches of the palate, the uvula, and the tonsils more or less reddened or purple. Sometimes, but not always, they are swollen; and the redness may extend to the roof of the mouth and to the pharynx. Swelling of the mucous glands often gives a granular appearance to the affected parts; and their secretion may accumulate upon the surface so as to simulate the presence of ulcers. The tonsils may even project inward until, with the club-shaped uvula, they block up the passage; and after a few days one or both of them may suppurate. There is often swelling of the neighbouring cervical lymph-glands.

Exanthem.—The rash generally makes its appearance in from twelve to

* In one case, under my own observation, a child left his father's house, where three of the family had died from scarlatina, on November the 19th and continued well until December the 2nd, when he developed the disease, just a fortnight after the last exposure to contagion. It is, however, possible either that the infection was from a different and more recent source, or that it was derived from fomites which, notwithstanding every care, may have been carried away with him from the house.—P. H. P.-S.

thirty hours after the commencement of the disease. Sometimes it is later; but, as Dr Gee remarks, the proof of this is difficult, not only because the first signs of it are easily overlooked, but because it may recede for a time, and then come out again. In some of the more severe cases it is altogether ill developed, and writers say that it may then be delayed until the third or the fourth day. In the great majority of cases the rash begins to appear towards the end of the first day, and is fully out on the second.

As a rule, the eruption is first to be discovered upon the sides of the neck and the upper part of the chest; and it generally takes twelve or twenty-four hours, or even two or three days, to reach its full development But sometimes it comes out almost at once over a very large surface. The face often remains free; and, when present, the rash is in most cases limited to the forehead and temples, the cheeks showing only the ordinary flush of fever, while the parts round the mouth, nearly to the chin, remain pale. The upper arms are often covered with the eruption, and it is exceedingly well marked on the abdomen and on the inner side of the thighs.

The colour is usually a bright scarlet, so that Watson aptly compared it with that of a boiled lobster; but sometimes it is of a lighter pink, and sometimes purplish. It may appear to be uniformly diffused, but on careful examination one can generally see that it is made up of very minute red points, which are at first isolated from one another, and which, even when they have coalesced, often leave a little islet of healthy skin here and there. This minutely punctated appearance is even more distinct from the "blotchy" aspect of the rash of measles than the bright crimson of the one from the rose-tint of the other. It momentarily disappears on pressure except where there has been some degree of hæmorrhage or perhaps capillary stasis, in which case isolated red spots remain, with a more or less general yellow discolouration. As Watson long ago pointed out, on the forearms and the legs, as well as on the backs of the hands and of the feet, the rash of scarlet fever consists of larger and more prominent papules than elsewhere; the palms and the soles, however, show only a faint diffused blush through their thick epidermis. The skin of the affected parts is slightly turgid or even swollen; the eyelids and the cheeks look a little puffy. Trousseau remarks that tumefaction of the fingers often prevents the patient from closing his hand.

Löschner discovered exudation-cells in the rete Malpighii, and probably they were seen by Dr Fenwick also, who further observed that the basement membrane of the sweat-glands was thickened, and their channels were obstructed by an overgrowth of epithelium, or by extravasated blood. That the scarlet fever eruption bears no definite relation to these glands, nor to the hair-follicles, seems to follow from a case (cited by Thomas on the authority of Landenberger) in which it did not fail to develop itself over an immense cicatrix, the result of a burn which was said to have destroyed the whole thickness of the skin.

Certain modifications of the eruption of scarlet fever are sometimes observed. Instead of being punctiform, it may in rare cases consist of large, irregular, slightly raised maculæ, more or less like those of measles, or it may be markedly papular. Or, again, it may be associated with immense numbers of miliary vesicles, especially upon the neck and chest, when there has been much perspiration.

At a variable period after the subsidence of the rash—which has usually faded by the end of the first week, often on the fifth day and sometimes earlier

—the superficial layers of the cuticle begin to peel off, or, in technical language, to *desquamate*. This is sometimes observed within a few days, sometimes not until two or three weeks have elapsed. Its amount is not necessarily proportionate to the intensity of the cutaneous affection; it may be well marked where the rash was so slight as to have been overlooked, and so may be of service in clinching a doubtful diagnosis. In some cases it assumes a furfuraceous form; a mealy powder, or a fine scurf, becoming detached from the surface, especially from the face. But more generally it is "membranaceous;" distinct flakes come away, and may be some inches long. The first step towards the throwing off the epidermis at a particular spot is often the formation of a little opaque raised vesicle, very like those which are characteristic of eczema, but dry; this breaks at the summit, leaving a free edge in the shape of a ring, which gradually becomes larger and larger. The cuticle of the hands is now and then shed *en masse*, like a glove; and the same may be the case with the feet; in some cases not even the nails are left behind. So complete a desquamation as this necessarily takes several weeks for its completion.

Much more often the fact that the exanthem interferes for the time with the nutrition of the nails, is shown, not by their exfoliation, but by the formation of a transverse groove: to this Dr Wilks has especially drawn attention. It is seen upon several of the nails at equal distances from their roots, and of course it ultimately becomes lost at their distal extremities. Such grooves on the nails are not, indeed, peculiar to scarlet fever, being sometimes seen after other acute diseases; but even with this qualification, their presence sometimes throws valuable light upon the origin of sequelæ, the real nature of which might otherwise have remained undetermined. The hair comes off with the cuticle; but perhaps not more than after any other fever of equal severity.

Desquamation follows the same course as the rash, beginning on the chest, shoulders, and neck, then spreading over the arms and back, and then over the lower extremities. It is often delayed in the hands and feet until long after the rest of the skin is clear.

Aberrant forms.—A striking feature of scarlet fever, and one upon which all writers lay stress, is the great variability of its symptoms and of its course; so that, indeed, there is much difficulty in describing all the different forms it may assume. In this, as in some other characters, it resembles enteric fever.

In some cases the disease is *rudimentary* or *abortive*. Thus, during epidemics, it often happens that adults become more or less feverish, complain of a slight sore-throat with redness of the mucous membrane, and have a little pain and swelling of the cervical glands, but are at no time really ill. This is especially apt to be the case with those who have already had an attack in childhood; medical men say that it has again and again occurred to them, when attending patients suffering under scarlet fever. But the author saw an instance in which a father so affected gave to his children a disease of such severity that one of them died. Again, the chief symptom may be feverishness, so that the complaint passes for a mere febricula, there being little or no affection of the throat, and no redness of the skin beyond what might be attributed to the pressure of clothes or to some other accidental circumstance. Lastly, the sole indication may be an eruption, and this perhaps limited to a small part of the surface. The real nature of this affection is often first shown by the occurrence of

desquamation, or even by the unforeseen supervention of dropsy or of some other of the sequelæ of scarlet fever.

In a very different class of cases, the symptoms are incomplete because the end comes too quickly for their full development. In several recorded instances the patients have died in from eight to fifteen hours; the chief phenomena have been delirium or coma, convulsions (or occasionally tonic spasms with trismus), incessant vomiting and diarrhœa, with extreme rapidity of pulse and of breathing. Sometimes there has also been hyperpyrexia, in which case one would be inclined to regard this as determining the form of the disease. A temperature of 115° F. was observed by the late Dr Woodman in some patients. The only thing that could render a diagnosis possible would be the occurrence of other cases of scarlet fever in the same neighbourhood. The writer saw a little boy with Dr Andrews in 1883, who was taken ill the same morning with severe headache, vomiting, and prostration. When seen he was already comatose, but there was no exanthem. The temperature was 105·6°, the skin was pungently hot, and the pulse too rapid to be counted. Such a condition could be due only to smallpox or scarlatina, and the presence of good vaccination marks, together with the characters of the skin and pulse, justified the latter diagnosis. He died the same afternoon; and the nature of the case was confirmed by the child's nurse afterwards sickening of scarlet fever.

The cases just referred to constitute the most extreme examples of what in England is commonly called *malignant* scarlet fever. But there are other varieties which are almost as certainly though less rapidly fatal. Sometimes, with early severe constitutional symptoms of a "typhoid" character, the rash comes out late and imperfectly, and its hue is not bright scarlet, but rather a livid violet. In other cases the disease assumes a *hæmorrhagic* form; at least, almost all writers say so except Dr Gee, who is evidently inclined to think that the early hæmorrhagic roseola of smallpox may have been mistaken for it.

Sometimes, again, the eruption develops itself in the regular way; but the febrile disturbance, which from the first is high, runs on for two or three weeks instead of subsiding after a few days. The patient then falls into a typhoid state, with muttering delirium, a dry brown tongue, and sordes upon the lips. Many of these cases end fatally. In a large proportion of them the affection of the throat is unduly severe. Sometimes the fauces become gangrenous and slough away, leaving a horribly offensive cavity. The inflammation often spreads to the lining membrane of the nose, and an acrid fluid flows from the nostrils, which excoriates the upper lip. The nasal bones may even become necrotic. Still more frequent is extension to the ear along the Eustachian tube; the tympanum then fills with pus, which is discharged by perforation of the membrane. Here, again, there is often great destruction of the tissues: Sir Thomas Watson mentions a case in which, every time that the child swallowed, some of the liquid ran out at one of its ears; and it is no uncommon thing for fatal hæmorrhage to ensue from perforation of the carotid artery.

Faucial diphtheria is a very dangerous complication, and sometimes occurs in almost every case in a local epidemic. It is doubtful whether we should look on it as a local variety of scarlatina or as a combination of this malady with diphtheria.

In other cases, the chief local mischief is outside the throat, in the cervical lymph-glands and the adjacent structures. A large swelling may form

on each side of the neck near the angle of the jaw; or the whole space from the chin to the sternum may be occupied by a brawny shining mass, which has been termed a "collar." It does not appear that the salivary glands, whether parotid or submaxillary, take any part in this affection. The induration of the connective tissue may spread backwards between the pharynx and the spine, or downwards into the mediastinum. If suppuration occurs, the pus may be discharged by a series of fistulous openings, burrowing and undermining the skin in all directions; or a post-pharyngeal abscess may be formed, which, pressing on the larynx, may cause suffocation. Extensive sloughing often takes place, so that the muscles are laid bare, as in a clean dissection; and dangerous or fatal hæmorrhage may ensue, from perforation of one of the arteries or veins of the neck.

Complications and Sequelæ.—Some of the affections just described might be reckoned under complications of scarlet fever. We may, however, certainly give that name to one morbid condition which is comparatively rare, and is unrepresented in the normal course of the disease. This complication, which is by no means confined to cases of great severity, is *synovitis*, a painful swelling of some or all of the joints. It generally sets in while the skin is peeling, and affects sometimes the smaller, sometimes the larger articulations; it generally subsides quickly, and it is even more fugitive than the synovitis of ordinary rheumatism. In some cases, however, it settles into one particular joint, leading to chronic effusion, or even to suppuration. Probably most cases of synovitis following scarlatina are true rheumatism.

Dr Ashby, of Manchester, finds synovitis *in the course of the fever* to be very rare and unaccompanied by evidence of cardiac inflammation. Subsequent rheumatism is less rare in young adults than in children. Endocarditis (rheumatic or nephritic) is less common as a sequela than dilatation of the heart.*

A more frequent complication of scarlet fever is acute *nephritis*, accompanied with albuminuria. Whether any affection of the kidney is present in ordinary cases of scarlet fever, which end in recovery, appears to be still doubtful. Thomas remarks that the question could be settled only by the microscopical examination of the organs from a patient killed by an accident during the course of the disease. It is certain that nephritis has been found after death in some exceptional cases of scarlatinal dropsy in which the urine, although scanty, contained neither albumen nor casts during life. This fact obviously suggests the possibility that renal changes may be present in cases which do well, without any evidence being discoverable. It is also certain that the more assiduously one tests the urine throughout the whole course of the disease, the more numerous are the cases in which a trace of albumen is detected at one time or another. Thus some German writers look upon a renal catarrh as bearing to scarlet fever the same relation which bronchial catarrh bears to measles. But albumen and occasionally even tube-casts are found in the urine in other febrile diseases, so that their presence is not necessarily to be taken as part of the action of a specific poison upon the kidneys. Scarlatinal *dropsy* will best be discussed with the anasarca that results from other acute forms of tubal nephritis. Anasarca without albuminuria is occasionally met with after scarlet fever.

Serous inflammation.—Pleurisy and pericarditis are of rather frequent occurrence, usually in connection with the affection of the joints, and this confirms the view of its rheumatic character. This may also be attended

* 'Lancet,' May 22, 1886. Compare the report from Glasgow ('Clinical Trans.,' 1885–6).

with endocarditis, and so may doubtless be the starting-point of chronic changes in the valves of the heart; but whether this ever arises when the joints have remained free appears to be doubtful. In making *post-mortem* examinations of children who have recently had scarlet fever without synovitis, we have always found the valves healthy. Pleurisy and pericarditis, or pneumonia, may also accompany scarlatinal nephritis.

Chronic enlargement of the *tonsils* is very common in children who have passed through this exanthem.

Far more important sequelæ are various affections of the *ear*. Indeed, scarlatina is almost always the explanation of deafness acquired in early life. The most frequent local lesion is acute suppurative tympanitis produced by extension of the faucial inflammation along the Eustachian tube. It often leads not only to perforation of the membrane, but to necrosis of the petrosal bone, and subsequent fatal pyæmia. Necrosis of other bones is not very uncommon, and glandular abscesses sometimes follow during convalescence.

An important complication, sometimes occurring in the first week, sometimes not until convalescence, is *diarrhœa*. It occasionally leads to fatal exhaustion.

Anatomy.—Little that is distinctive is found in examining the body after death from scarlatina, beyond the appearances due to fever as such, and those produced by any complications which may have been present during life. No trace of the rash remains, although the presence of desquamating cuticle may give a hint of the nature of the disease in cases not seen during life. Even the inflammation of the fauces leaves very slight effects. Occasionally the heart is found in a state of acute dilatation, as after some cases of diphtheria. There are not infrequently signs of intestinal catarrh with slight swelling of Peyer's patches. The kidneys are large, and show either glomerular nephritis or ordinary tubal inflammation.

Prognosis.—The prognosis of scarlet fever can never be altogether devoid of anxiety, for in the mildest case some dangerous complication may arise. Sporadic cases are sometimes quite as severe as those which form part of an epidemic; and epidemics differ widely in their gravity. One prevailed for many months in a country town in Kent without there being a death, and without dropsy ever supervening, and Dr George Turner observed an epidemic of 120 cases at Portsmouth without a single death. Graves recorded the fact that whenever scarlet fever appeared in Dublin between 1805 and 1833 it was always mild, so that on one occasion eighty children were attacked in the same public institution, and all recovered; but during 1834 and 1835 the city was the seat of a very malignant and fatal epidemic. Thomas says that in Saxony a mortality of from 13 to 18 per cent. is very common, but that it not infrequently rises to 25, and sometimes even to 30 or 40 per cent. Scarlatina in adults is usually less severe than in children; in puerperal women, however, it is notoriously dangerous, though far from being always fatal.

Most of the inferences to be drawn from particular symptoms have already been indicated. But it may be added that, according to Trousseau, the occurrence of convulsions during the first or second day is always a sign of danger, whereas in other exanthemata it is not of evil omen. Another point which is made by this writer is that in scarlet fever, as in smallpox, the more intense the rash the more severe is the disease. Here, however, he differs from most other observers; and the statement perhaps only

means that those cases in which a bright red eruption covers the whole of the body, and lasts for an unusually long time, are generally grave ones, whereas when the eruption is slight and quickly over the fever also is moderate and of brief duration.

Protection.—This is less complete than in the case of typhus, enterica, smallpox, or perhaps measles. Second and even third attacks are not very uncommon. But they occur after a considerable interval; and relapses, though not unrecorded, are certainly very rare.

We have no means of *prophylaxis* but isolation. Belladonna was supposed to be indicated by the fact that poisoning by belladonna produces dryness of the throat and a roseolous eruption, just as in former times red curtains were hung round a patient's bed because of the colour of his skin. It has, however, been fully tried, and conclusively proved to be useless. Arsenic has been credited with a similar power, probably on insufficient grounds.

Treatment.—When the contagion has been once taken in, we must be content to treat scarlatina on the same general principles as those indicated in the chapters on Typhus and Enteric Fever.

In mild cases little is needed beyond careful isolation and free diluents. Tepid baths and sponging the surface is grateful in almost every case.

When the temperature is very high, 104° or upwards, we must check it by cold sponging, wet packs, rubbing with ice, or cold baths, as described above (p. 172).

The angina causes much pain and difficulty in swallowing. Sucking ice gives great relief; and, if the patient will allow it without too exhausting a struggle, it is useful to clear the throat with a large camel-hair brush or with cotton-wool firmly tied on to a penholder; or a spray medicated with thymol, carbolic acid, or some other disinfectant, may be employed. Older patients may use Condy's fluid or chlorate of potash as a gargle, or borax and honey as a linctus, with advantage. In severe cases nutrient enemata or suppositories may be tried; and if they fail, as they unfortunately often do, the patient must be fed by the nose; not with a catheter—a child's nasal passages are too small, and the process would be too long—but by inserting a glass funnel, of the kind used for filtration in laboratories, into the opening of one nostril, closing the other, and pouring milk and egg, beef-tea, or other fluid nourishment into the pharynx. An aural speculum answers the purpose well.

Brandy must be given if the pulse requires it. Quinine in doses of five grains three times a day for a child of five years old is recommended by Dr Eustace Smith. Chlorate of potash is also of undoubted benefit in many cases of scarlatina anginosa.

As soon as the eruption is fully out, the whole surface should be anointed with carbolic oil (1 in 30); and when the fever has subsided and desquamation has appeared, the hair should be cut short, and the whole body well washed with hot soap and water, followed by inunction. A warm bath with soap may then be given daily. The oil is not only pleasant to the patient, but prevents the desquamating scales from flying about.

Great care should be taken during the stage of peeling to keep the patient from draughts. The body should be clothed in flannel, and any chill carefully guarded against. The urine should be daily tested, and while it contains albumen the patient should not be allowed to leave his bed or to take meat.

If, in spite of precautions, nephritis and dropsy follow, the case is one of

acute Bright's disease, and must be treated by purges and other means, which will be detailed in the second volume.

Rheumatism as a sequela is amenable to salicyl compounds—another proof of its true nature.

During convalescence, tincture of steel is the most useful drug, and port wine the best form of stimulant, when it is required. Even in mild cases the child should keep his bed for three weeks and his room for a month at least from the beginning of his illness (Eustace Smith).

The rules for disinfection given on pp. 24, 25 must be thoroughly carried out. The patient must not associate with other children until the desquamation has ceased, except perhaps that on the soles of the feet, which sometimes lasts for weeks after the rest of the surface is clear.

The infection of scarlatina begins later and lasts longer than that of measles or of smallpox, probably for six weeks, or even more.

RUBEOLA*

" Facies non omnibus una,
Nec diversa tamen, qualem decet esse sororum."
OVID.

Recognition and nomenclature—Its characteristics—Discrepancies and probable explanation—Incubation—Onset and course—Diagnosis—Protection—Prognosis and treatment.

SCARLET FEVER had not long been universally recognised as distinct from measles, when German physicians in the latter half of the last century began to describe a third member of the same group of diseases, for which they adopted the term " Rubeola." Ever since, the relation of this Rubeola or Rötheln to Morbilli or Masern† has been a subject of controversy.

In this country, Dr Paterson, of Leith, described the supposed third exanthem in the 'Edinburgh Medical Journal' for 1840, and among English writers it has since been recognised by Murchison, Squire, Bristowe, Robert Liveing, Eustace Smith, and Goodhart.

The term " epidemic roseola," adopted by Squire and by Bristowe from Trousseau, would convey the wrong impression that the disease bears some relation to the other eruptions which have been known as species of roseola since the time of Willan. Moreover, Trousseau believed that his " roséole epidémique " did not correspond with Rötheln or Rubeola notha.

On the whole it seems best to use the word Rubeola, first invented by Sauvage as a synonym of morbilli, and afterwards appropriated to " German measles " by Copland. Measles in English, and *morbilli* in Latin, are satisfactory names, distinctive and unambiguous. It is a pity to lose a word which has already been applied to a disease in want of a name, and which, if not so used, must be abandoned altogether. Moreover, Rubeola is the accepted equivalent of Rötheln with German authors.

Rubeola, then, is a specific and infectious febrile disease, occurring in epidemics, arising from contagion and breeding true, accompanied by an exanthem, and protecting against itself.

Its most essential features are that, with catarrhal symptoms like those of measles, and a variable but distinct rash, it resembles scarlet fever in having a very short prodromal stage and in being attended with marked sore-throat.

A favourite idea with some of the older writers (Schönlein among others) was that it was a " hybrid " between these two diseases; Niemeyer wrote of the symptoms alternating in different cases, so that sometimes the rash of measles would be associated with the local lesion of scarlet fever, and sometimes the reverse would be the case. But this was purely fanciful.

* *Synonyms.*—Rubeola notha, Rubella, bastard measles (Babington); German measles.—*Fr.* Roséole epidémique, *Germ.* Rötheln.

† According to Seitz *Rötheln* is a local or dialectic name for ordinary measles (*Masern*) in many parts of Germany.

There is no doubt that two specific exanthems may exist together in the same patient, but they do not combine so as to produce a modified affection; still less can such an affection be epidemic, or protect against itself, while affording no immunity against either of the constituent diseases.

In the first edition of this work, Dr Fagge contrasted the account given by Paterson, and accepted by Aitken and other systematic writers, with that of German authors, and especially with Professor Thomas's article in Ziemssen's 'Cyclopædia,' based on two epidemics at Leipzig in the years 1868 and 1872. In the former account we read of marked disturbance at the invasion, high fever, severe catarrh, with œdema of the glottis, bronchitis, severe angina with dysphagia, and a raised blotchy eruption appearing on the third or fourth day. Death was not infrequent from suffocation or from convulsions.

The discrepancy of this account from those given by Thomas and other German writers must be admitted, but possibly Paterson's epidemic was not rubeola at all, but measles. When the writer was a student at Guy's Hospital the crew of a Peruvian vessel in the Thames were attacked by measles, and the patients were taken into our wards. The disease was exceedingly severe, and several died from it, in almost every case from bronchitis or pneumonia. The same unusual malignancy marked the introduction of measles into the Fiji Islands.

However this may be, there can be little doubt that the disease described by Thomas, Eichhorst, and other German writers, by Bristowe, Liveing, Cheadle, Eustace Smith, and Goodhart,* is one and the same, different both from measles and from scarlet fever.

There was an epidemic among the nurses at Guy's Hospital a few years ago which satisfied all who saw the patients of the reality of the disease. The cases differed considerably in severity and in the character of the rash. Sore-throat was always present, and catarrh was much less frequent, so that it was very distinct from measles; while the absence of the characteristic signs of scarlatina and of any severe cases prevented confusion in that direction. Some of the mildest cases, when the patients were about, and therefore the rash not obvious, might have been set down as "epidemic sore-throat."

Rubeola occurs in the British Isles and on the Continent, in Egypt, in India,† and in America.

Origin and incubation.—All writers agree that rubeola arises strictly by contagion from other cases. The most important predisposing cause is age; it is more frequent about puberty, is rare in young children, and not common in adults. Boys in a family are said to escape oftener than girls.

The incubation stage is generally somewhat shorter than that of measles. In an epidemic at Guy's Hospital in 1888 several of our students were attacked, and the shortest incubation period observed was eight days, while one seemed to be as long as seventeen days. Of 86 cases observed with respect to incubation, it lasted six to ten days in 32, eleven to fourteen days in 45, and two or three days longer in 9 only. Bristowe puts

* The reader may also refer to a succinct but full account of an epidemic of fifty cases by Dr Douglas, of Newbury ('Brit. Med. Journ.,' May 26, 1877), and to a short paper by Dr Ryle (*ibid.*, July 24, 1886).

† Surgeon-Major McLeod read a paper before the Epidemiological Society (Feb. 11, 1885) on an epidemic rose-rash which visited Calcutta in 1881, and which he rightly decided to be rubeola.

it at about a week, or rather less; Squire about ten days; Liveing ten to fourteen days; Douglas fourteen or fifteen days, never more, and rarely less; while Thomas gives a fortnight or even three weeks. Dr Clement Dukes, whose opportunities as medical officer of Rugby School were exceptional and exceptionally used, states that out of twenty-four carefully observed cases the incubation lasted twelve days in two, and from fourteen to twenty-two in the rest.

Dr Haig-Brown recorded an epidemic of rubeola at the Charterhouse School in the 'Brit. Med. Journ.' for April 16th, 1887. He found the infection is apparently less active than that of measles and less persistent than that of scarlet fever, for more inmates of a house or school escape during an epidemic of rubeola than during one of measles, and cases seldom occur after an interval of cessation. Nothing is exactly known of the nature or the mode of conveyance of the contagion.

Onset and course.—As a rule the first symptoms are not severe. There is usually little or no catarrh before the rash, and the fever is not high.

The *exanthem* appears early; often it is the first symptom noticed, and it is scarcely ever delayed beyond twenty-four hours after the temperature rises. Among 159 cases it was out on the first day in 119, and on the second in 39. It is more like the rash of measles than that of scarlatina, but it is of a brighter rose tint; it is patchy, but less mottled and more diffuse than measles, while it is less vivid red, less diffused, and less punctate than scarlet fever. Its distribution is earliest on the forehead and cheeks, and afterwards the trunk, particularly the back. It is not so markedly developed on the face as measles. The upper arms are affected, the nates and the thighs; but seldom the hands and feet. There is often troublesome itching. It spreads more rapidly and less regularly than the other two exanthems.

The eruption lasts three days or more, usually longer than measles, but not so long as scarlatina. Desquamation is present, but needs careful looking for, and never resembles that of scarlet fever.

With the rash there is, in probably every case, angina; the fauces are injected and swollen, but there is never sloughing, and the lymph-glands at the angles of the jaw are usually not enlarged. On the other hand, the glands which run along the hinder border of the sterno-mastoid muscle can as a rule be felt. There is lacrymation with some photophobia, and usually slight nasal catarrh, accompanying but not preceding the exanthem. In exceptional cases this is followed by bronchitis. But catarrhal symptoms may be absent, and they are rarely so marked as in even mild cases of measles.

Pyrexia on the second day is sometimes rather high, with quick pulse, restlessness, and occasionally delirium; 100°—103° are the usual temperatures, but 105° has been more than once recorded. It subsides with the rash on the fourth day, and the pulse falls with it.

Traces of albumen are often to be found in the urine, and Dr Liveing has in exceptional cases seen it persist and anasarca follow. The tongue is moderately furred, and does not resemble that of scarlatina. Dr Douglas informs the writer that, in an epidemic in 1883–84, he twice saw "rheumatoid" synovitis affecting the wrists and lasting three or four days.

The whole attack lasts four to six days, about as long as an ordinary one of measles.

Sequelæ appear not to exist. Dr Goodhart affirms that if after an attack

of German measles a child remains thin and feeble, or has a discharge from the ears, the disease was either scarlatina or measles.

Diagnosis.—The first cases of an epidemic may often be doubtful; but after seeing a few, the eye recognises the peculiar aspect of the rash. Between slight cases of scarlatina and rubeola the distinction is often difficult: the desquamation in the former case is probably the most helpful point to look to. The early appearance of the exanthem (usually along with the fever) is the best distinction from measles.

It is often difficult to distinguish rubeola from "ordinary rose-rash," whether set up by gastric disturbance or definite poisons, or caused by local irritation. Dr Dukes says that the roseola produced in some persons by handling the hairy larvæ of certain moths (the "woolly bears" which schoolboys collect) is sometimes exactly like rubeola. There is no doubt of the existence of a generally diffused bright red eruption, which, though not contagious, appears in groups of cases in hot weather. The name "roseola," or rose-rash, is suitable for this affection, which is quite distinct from true erythema multiforme (to be described under Diseases of the Skin) as well as from rubeola, from morbilli and from scarlatina. It is clear from the absence of catarrh and sore-throat, from the severe itching, and from the proneness to relapse, beside many other characters, that this affection, graphically described as Roséole by Trousseau ('Clinique Medicale,' tome i, p. 161) and as Erythème scarlatiniforme by Hardy ('Leçons sur les Mal. de la Peau,' 2me partie, p. 35), is not—as the late Dr Tilbury Fox ('Skin Diseases,' p. 93) and other writers have supposed—identical with the rubeola of German authors. Probably the papules described by Vogel and Borsieri as *ardentes et prurientes*, and named Essera Vogelii, were of the same nature. Essera is defined by Blanchard ('Lex. Med.,' ed. 1702) as *tubercula parva, ad rubrum vergentia, duruscula, cum insigno pruritu subito universum corpus occupantia.*

Protection.—Second attacks are rare, probably rarer than in the case of true measles; statements to the opposite effect no doubt refer to epidemic roseola, not to rubeola. It does not protect from measles or from scarlatina. Among sixty-three cases of rubeola seen by Dr Dukes, thirty-nine had previously had measles. Among thirty cases observed by Dr Cheadle, twenty-two had had measles, and ten of these cases had been under his own care. There seems no reason to suppose that measles predisposes to rubeola, but measles attacks, as a rule, younger, and rubeola older children. There are, however, several instances of an epidemic of rubeola following one of measles.

Prognosis.—Most authors agree in saying that in epidemics much more severe cases than the rest may occur, and that death may occasionally happen. If, however, we exclude the description by Paterson, these cases must be extremely rare. A favourable prognosis may therefore be given.

No special *treatment* is necessary. The patient need not in many cases be confined to bed after the nature of the complaint is clear, but he should be secluded from others for a fortnight if infection is to be prevented.

SMALLPOX*

"That day that the Friends on both sides met to conclude the Marriage, she fell sick of the Smallpox, which was in many ways a great trial upon him: first her Life was allmost in desperate hazard, and then the Disease (for the present) made her the most deform'd person that could be seene, for a great while after she recover'd; yett he was nothing troubled at it, but married her as soon as she was able to quitt her chamber, when the Priest and all that saw her were affrighted to looke on her. But God recompenc'd his justice and constancy by restoring her as well as before."—*Life of Col. Hutchinson.*

Name and history—Contagion and spread—Incubation—Onset—Early rashes—Specific eruption—Varieties of the Exanthem—Course and symptoms—Discrete, confluent, and modified Smallpox—Complications and sequelæ—Prognosis and treatment.

VACCINIA—*Inoculation—Introduction of vaccination—Course of the eruption—Its protective power—Its drawbacks—Its relation to Variola.*

THIS terrible disease—after the disappearance of the plague and before the introduction of vaccination, the most fatal of all epidemics—was unknown to the ancients. It was first described by the Arabian physician Rhazes about 900 A.D., under the name *Jadari*, translated into λοιμική, *i. e.* the pestilential eruption, by the Greeks, but was long regarded as a more severe kind of measles.

It is probable that some of the pestilences of the later Roman Empire and of mediæval Europe were really epidemic Variola. As the knowledge derived from the Arabian writers by Greek and Jewish physicians became diffused, the more severe and dangerous disorder was discriminated from measles, but even when, in the sixteenth century, smallpox was generally recognised, the two were often confounded together, and so late as 1660 Pepys speaks of measles as "the same disease" as the smallpox ('Diary,' January 11th, 1660–1).

Variola appeared in America soon after its discovery, and was terribly destructive among the natives. It is almost as much so in Central Africa now.

In China long before the Christian era Variola was well known, and is described in books still extant.

Origin.—Smallpox is in some respects the most typical of the class of exanthemata. Its infectious character has never been doubted, and its virus is as active as that of typhus and almost as persistent as that of scarlatina. It resides in the serum and pus of the exanthem, and can be preserved in the powdered crust of the skin. It has been proved by

* *Synonyms.*—Variola.—*Scottice* the Pocks, *Fr.* la petite Vérole, *Germ.* Blattern, Menschenpocken, *It.* Vajuolo, *Mod. Gr.* Εὐλογία, a euphemistic term.

The English word refers to the pockets or little pocks, pokes, or bags in which the matter is contained, and denotes the pustular form of the eruption. It also marks the distinction between this and the still more dreaded Great Pox, *i. e.* Syphilis. The low-Latin word *variola* is a diminutive formed from *varus* (with a short *a*, although this did not prevent a Ciceronian pun between it and *varius*), a pustule or pimple, used as a translation of the Greek term, ἴονθος. The word variola first occurs in this sense in the sixth century. In older medical literature the plural form *variolæ* is used: referring, like "smallpox" and "measles," to the individual little *vari* or "pushes" of the eruption.

filtration to be particulate; but no distinctive micrococci have been yet discovered, so that its bacterial character is an inference from analogy, and not a certainty. The disease may be spread without actual contact with a patient by inhaling dried-up secretion or by fomites which have been contaminated with the pus, and probably by the air at a distance. The blood does not appear to be contagious, nor the excretions.

There are no decided predisposing causes of smallpox. It attacks persons of every age and race, and is equally destructive in every climate. It has, however, been remarked that children and pregnant and puerperal women are particularly liable to take it severely.

Variola can be transmitted to many of the lower animals by inoculation with the pus, but is a much less fatal disease than in man, and does not spread. Many pathologists, however, believe that vaccinia in cows and one kind of the affection known as "grease" in horses is modified variola.

The incubation of smallpox is, as a rule, twelve days. On the thirteenth day from that on which the contagion entered the patient's body, he is seized with symptoms of fever. Most writers allow that the interval may sometimes be longer; Bristowe extends it in exceptional cases to sixteen days; but Marson affirms that he has never found the eruption fail to appear in fourteen days after infection. He admits, however, that the incubation is sometimes shortened by three or four days, and Curschmann alludes to one instance in which it did not last more than five days. Zülzer believes that in the hæmorrhagic form of variola it is constantly from six to eight days. When variola is transmitted by direct inoculation the period of incubation is only nine or ten days.

During this period the patient generally feels well; but occasionally he complains from the very first of a vague malaise, with gastric disturbance, headache, and giddiness, and towards the end there may be a little pharyngeal catarrh, with reddening of the uvula and tonsils.

Prodromal stage.—This usually sets in with a severe rigor or with a succession of slight chills. The temperature at once begins to rise, and may reach 102° to 104° within twenty-four hours, and 105° or even 107° by the second day (*fièvre d'invasion*). In some cases the patient becomes rapidly prostrate, and totters if he attempts to stand. Curschmann remarks that whereas a working man attacked with enteric fever will often come on foot to the hospital at a time when his temperature is already high, one who has smallpox must be carried. Such a patient, while he is out of bed, has cold limbs, a pale sunken countenance, and a small pulse; so that he is hardly to be recognised a few hours later, when he has become warm, and when his face is red and turgid. The pulse is much accelerated, varying from 100 to 120 in men, while in women it may reach 130 or 140, and in children 160. The breathing is quick, short, and laboured. The skin is generally dry, but sometimes moist or even sweating. Some medical men, among whom was the late Mr Stocker, believe that it already emits a peculiar odour; and it is said that the late Dr Guy Babington when taking in patients to the hospital could recognise any cases of variola by the smell. The breath is fœtid, and the tongue is often very foul. Anorexia and thirst are present. The patient almost always complains of pain across the forehead or over the whole head; and this is sometimes intense. Children are not infrequently attacked by epileptiform convulsions; or they may become

delirious or comatose. They often have diarrhœa; but in adults constipation is usual. In the more severe cases enlargement of the spleen may be detected.

So far there is little to distinguish the early period of variola from that of other fevers. A more characteristic symptom is *vomiting*, attended with violent retching and pain at the epigastrium, and sometimes so severe and persistent that a case has been sent up to hospital as one of ileus. Another symptom, still more characteristic, is *pain in the sacrum and loins*. This, however, is not constant; Curschmann says it occurred in rather more than half of his cases. It appears to be most frequent in severe cases. Women are apt to suppose that it merely indicates the approach of menstruation; or, if they are pregnant, that labour is about to set in. And, as a matter of fact, the catamenia do frequently appear during this stage; sometimes prematurely, but often in their regular course. In men the pain in the loins is likely to be mistaken for lumbago.

Another set of symptoms, which are of importance because they may cause the disease to be mistaken for measles, are those of common *catarrh*—sneezing, epistaxis, intolerance of light, lacrymation, sore-throat, and hoarseness. The tonsils and the palate may be seen to be reddened.

Lastly, the initial stage of smallpox is occasionally attended with one or other of two distinct forms of cutaneous rash, which differ in their characters and still more in their significance.

Early roseola.—One of them may be described as *roseola variolosa*, the name employed long ago by Rayer, and in 1853 by Eimer. Morton, writing in 1718, records it in one of his cases (No. xxxviii), and calls it an early rash resembling scarlatina. Until recently most observers regarded the presence of this remarkable rash as a proof that one of the other exanthemata was present in addition to variola. Reinhold, in 1840, seems to have originally propounded in express terms the doctrine that it is a preliminary symptom of smallpox alone. In this country Dr Wilks drew attention to it in the 'Guy's Hospital Reports' for 1857 and for 1861. Watson also mentions it in 1857. But Simon, of Hamburg, is the writer who has most fully described it, and has recorded the largest number of cases. It is more common in some epidemics than in others. As a rule it comes out on the second or the third day; but sometimes it immediately follows the initial rigor, and occasionally is the earliest sign that anything is amiss with the patient. It may last a day or two, but sometimes is over in a few hours.

There are several varieties of this early roseola. One is maculated, and is more or less like measles; it may come out all over the body, perhaps appearing first upon the face. Another is an evenly diffused red blush, resembling scarlet fever, but (according to Simon) darker and of a more purplish tint. This often has a characteristic distribution, which was pointed out by Hebra: it is limited to a triangle having for the base a horizontal line drawn across the abdomen about the level of the umbilicus between the two iliac spines, while the apex is formed by the contact of the two thighs. It may also be visible in the axillæ, and on the adjacent parts of the upper arms and of the chest. On the limbs it may affect the extensor surfaces of the elbows and of the knees, the backs of the hands and fingers as far as the first phalangeal joints, and the back of each foot in a line corresponding with the course of the extensor tendon of the great toe. Its distribution is so characteristic that it enabled the writer several years ago

to diagnose a case in which there was no other reason to suspect smallpox. Even if no other eruption should afterwards develop itself, one ought probably still to maintain the diagnosis of variola, for Simon has recorded an instance of this kind in which the disease aborted, notwithstanding that its real nature was proved by the patient's sister suffering from an attack of variola at the same time. Sometimes the seat of *roseola variolosa* is irregular. We have in our museum at Guy's Hospital models of a case in which it affected only the flexor surfaces of the forearms; and in women it may be limited to the skin about the nipples. Another variety resembles urticaria, while in many cases it assumes a petechial form, and leaves green or brownish stains after it has faded. A point of importance is that, even when it thus becomes hæmorrhagic, the prognosis need not be unfavourable. Hebra and Trousseau seem to have independently remarked that the parts affected by *roseola variolosa* afterwards remain free from the proper smallpox rash; and most subsequent observers have confirmed this statement.

Early purpura.—Very different is the second form of initial eruption, which in fact marks the most fatal of all the varieties of smallpox, the *Variolæ nigræ* of older writers, also known as *Purpura variolosa*, and *Malignant* or *Hæmorrhagic smallpox.*

After the usual early symptoms have been present during from eighteen to thirty-six hours there appears on the trunk and the limbs a diffused scarlet redness, which at first can be made to disappear by pressure with the finger. This soon becomes the seat of extensive effusions of blood, in spots and patches of all sizes and shapes. Large black rings now form round the eyes, and the conjunctivæ are ecchymosed: this, according to Marson, is often seen very early in the case, and is always a most dangerous symptom. The breath has a horrible fœtor, from the presence of a "diphtheritic" affection of the throat. Hæmorrhages may occur from any or all of the mucous surfaces. Bloody liquids are coughed up or vomited, or discharged from the uterus or the bladder; or there may be epistaxis, or a sanguineous flow from the eyes or the ears. The urine is extremely fœtid, and it is albuminous even when it contains no blood. The patient generally complains of severe pain at the præcordia, and suffers severely from vomiting. Yet, according to Curschmann, the temperature is seldom over 104° until just before the fatal termination. Both he and Marson remark that consciousness is generally retained almost to the very last; "few patients are so fortunate as to become quickly delirious or comatose." Anæsthesia or hyperæsthesia of certain parts of the surface, and paralytic affections of the limbs, are said to have been observed by Zülzer. Before death the whole body becomes black or of a leaden grey colour. According to Marson, the smallpox eruption is nearly always confluent in cases of this kind; but they often end too rapidly for this stage to be reached. Dr Fagge, however, recorded two or three instances in which, although the disease was prolonged for several days, not even papules could be discovered. The first of these cases occurred in the clinical ward of Guy's Hospital; the patient lived several days, and as no sign of a proper smallpox eruption could be detected, it was supposed not to be an example of *purpura variolosa;* but a few days afterwards the clinical clerk who watched the patient fell ill with a mild form of smallpox. It has often been remarked that strong muscular men are particularly apt to be attacked with hæmorrhagic smallpox; but it is also seen in drunkards, in women recently confined, and in those who are pregnant.

Slight and abortive cases.—Occasionally the symptoms during the pro-

dromal stage are very slightly marked, or even absent; the smallpox eruption then appears after a few hours' malaise, or is the first sign that the patient is ill. In such cases the disease itself is always very mild; so that Trousseau is not right in saying that the longer the exanthem is postponed the less serious is the attack.

Unlike a slight initial stage, one which is severe is no guide as to the ultimate progress of the case. In women and in children it often happens that the most alarming symptoms are present during the first day or two, notwithstanding that the rest of the disease is to be of a mild character. The most extreme instances of this are afforded by a form of smallpox which was long ago described by Sydenham as *Febris variolosa*, and by de Haen as *Variolæ sine variolis*. After a well-marked initial stage the disease aborts, and the patient is well in three, four, or six days at the latest.

The exanthem.—As a rule, the *third* day is that on which the characteristic eruption of variola appears; but in children it is often the second day. Sometimes nothing is to be seen until the fourth day, and then the prognosis is comparatively favourable. A still further postponement seems to occur only when there is some complication, as in a patient of Trousseau's who had choleraic symptoms.*

The proper variolous exanthem is first papular, then vesicular, and finally pustular. The papule is round and smooth; at first it may be scarcely redder than the rest of the skin; and as it is hard, like a small shot, it can often be felt better than it can be seen. But after twenty-four hours it is always decidedly reddened, besides having increased in size. It depends upon a definite change in the superficial and middle cells of the rete mucosum, which from the very commencement of the morbid process are swollen and opaque. Liquid exudation quickly takes place, so that by the end of two days the horny layer of the epidermis is raised to form a minute conical vesicle. By the fourth or fifth day of the eruption (seventh or eighth of the disease) the vesicle is generally as large as a split pea, hemispherical in form, and opaline in appearance. As a rule, the *pock*, as it is termed, has a central depression or umbilicus. The origin of this has been much discussed; it generally seems to depend upon the fact that the original papule was developed round the mouth of a hair-sac, or else round that of a sweat-gland, either of which structures may afterwards form a *retinaculum*, tying down the roof of the vesicle in the middle. Rindfleisch gives a drawing of a preparation in which a sudoriparous duct is plainly seen in this relation to the pock, and he says that he has many such specimens in his possession. However, it would seem that this explanation is not always applicable, since the pock does not necessarily bear a definite relation to any of the canals which traverse the cuticle. In all probability a similar function may be discharged by one of many other bands which cross the upper part of every vesicle in a direction more or less vertical, dividing it into a number of separate chambers. This *loculated* character of the pock attracted notice long before its nature was understood; it affords the reason why only a small part of the fluid is evacuated when a needle is introduced into the roof at a single spot. But the idea of the older observers was that the septa between the chambers were new formations, consisting of fibrin. Sir

* Among thirty-eight cases of Variola recorded by Dr Richard Morton in the City of London two hundred years ago (1670—94) the eruption appeared on the third day in twenty-six, on the fourth in seven, and on the second in five.

Thomas Watson, for instance, speaks of a "central whitish disc," consisting of "several little cells;" and this, he says, may by careful dissection be taken out entire with the "lymph" which it contains, even when the rest of the fluid in the pock has become yellow and purulent. Auspitz and Basch, however, showed several years ago that all the septa in question are in reality formed out of the original cells of the rete mucosum, small bundles of which cohere together, and become stretched out into filaments and bands, as the exudation accumulates around them. In this fluid leucocytes are present in small numbers from the very first; they go on increasing, and thus transparent serum gradually passes into opaque pus; the change is complete in about six or seven days from the first appearance of the papule,—that is, in the earliest part of the eruption, by the ninth or tenth day of the disease. The pustule, when at its height, is often hemispherical, the umbilicus having disappeared in consequence of the rupture of the retinaculum which formed it.

But while the roof and the cavity of the pock are thus being developed out of epidermic structures, a morbid process is at the same time going on more or less actively in its floor, which consists of the papillary layer of the cutis, with at least the lowest cylindrical stratum of the rete Malpighii. Whether a swollen state of the papillæ has any share in the formation of the original papule appears to be doubtful; but there is no doubt that its redness is due to their hyperæmia; and (according to Bärensprung) this extends down through the whole thickness of the skin. The exudation which fills the vesicle, and afterwards the pustule, is necessarily derived from these vascular tissues. But Curschmann says that, so far from the papillæ being always enlarged at this stage, they are often rather flattened by the pressure to which they are subjected. In some cases, however, they become the seat of an infiltration of leucocytes, which is so intense that it obliterates their blood-vessels, and, indeed, destroys their structure completely, converting them into a white or ash-grey substance. German histologists, in accordance with their usual terminology, describe this form of pock as *diphtheritic* (*vide supra*, p. 52). The contrast is the greater because the surrounding skin is of a bright red colour for a considerable distance, making what is termed the *halo* or *areola*. The infiltration of leucocytes into the floor of the diphtheritic pock may extend to a varying depth in the derma, or even through it into the looser tissue beneath. Rindfleisch gives a drawing from an injected preparation, in which the affected area had failed to receive any of the colouring matter.

Many of the pustules of smallpox undergo destruction almost as soon as they are fully formed. They break, or are ruptured, giving exit to a honey-like matter, which collects in drops upon the face or other exposed parts of the patient's skin, and saturates his shirt, his pillow, and his sheets. Others, however, remain uninjured, and ultimately dry up without discharging their contents. First a yellow-brown spot appears in the roof of the pock; this sinks in, so as somewhat to resemble the earlier umbilicus; gradually it enlarges and extends to the periphery. The process of desiccation, both in ruptured pustules and in those which are entire, begins about eight or nine days after their first appearance in a papular form. The crusts vary in colour from yellow to brown, or even black, as more or less blood is mixed with the pus of which they are formed. It is now that the extent to which the cutis vera has been involved in the inflammation affects the result. If it has escaped, the crusts fall off in four or five days (about the

fifteenth or sixteenth day of the disease), leaving purple-red stains, but little or no permanent cicatrisation. But if pus-cells have infiltrated the papillæ, or the whole thickness of the skin, these structures, to whatever depth they may have been affected, slough away and become detached, as shreds, adhering to the under surface of the crusts. Their separation takes longer, and may not begin until the eighteenth or twentieth day. Moreover, when it occurs, granulating surfaces are exposed which may take a considerable time to heal. The resulting cicatrices are for a time of a brown colour, but ultimately they become whiter than the skin around them. They may either be so faint as to be scarcely perceptible, or more or less deep and pitted. It often is not until many months after recovery from the disease that they become depressed below the surface to the full extent.

Distribution.—So much for the appearance, structure, and course of the individual pocks of variola; the locality and range of the eruption will now be considered. Like the rest of the exanthemata, it does not develop itself over the whole body at once; it appears first upon the face and scalp, especially on the forehead and about the eyes, the nose, and the upper lip. A few hours afterwards it is to be seen upon the trunk and the arms. Marson mentions the wrists as among the earliest parts affected, and says that on the legs and the feet it is generally two days later than elsewhere. He also lays stress on the fact that the papules come out "in threes and fives, forming crescents," or even a complete circle if two crescents happen to coalesce. During the first day or two fresh spots keep appearing, even on those parts which are already more or less thickly covered; but by the end of this time the eruption is complete, for even if a few more should afterwards show themselves they soon abort and die away. A curious circumstance is that smallpox specially affects any parts of the skin which may happen to have been recently irritated; the red patch from a mustard plaster applied during the incubation or shortly before will present many more pocks than the regions adjacent.

Certain *mucous membranes* take part in the eruption of smallpox, but not in quite the same way as the skin. There first appear raised spots, whitish or grey in colour, contrasting with the reddened state of the surface around them; but these quickly pass into excoriations. They are sometimes present in large numbers on the inside of the lips and on the cheeks; sometimes they rather affect the tonsils and the palate, which may then become greatly swollen and the seat of deep phlegmonous inflammation, ending in abscess. On the other hand, the tongue very seldom presents any traces of vesicles, but is sometimes enormously enlarged, protruding from the mouth, rendering the patient unable to close his jaws, and apparently helping to bring about a fatal issue. The air-passages are said to show more or less distinct pustules as low as the bifurcation of the trachea, or even (according to Wagner) down to bronchia of the second or third order; there may also be deep ulcers in the larynx, with perichondritis and necrosis of cartilages, and œdema of the aryepiglottidean folds. In the alimentary canal nothing resembling a pock can be seen below the upper part of the œsophagus, except perhaps in the rectum close to the anus, or at the entrance of the vagina. Marson speaks of twenty-six cases at the Smallpox Hospital, out of a total of over fifteen thousand, in which the conjunctiva showed a single pustule, which was generally situated midway between the cornea and the internal canthus. It did not in any way affect the sight or lead to any ill result.

Varieties in the eruptive stage.—Roughly speaking, the severity of variola depends upon the number of the pustules; and in the relation of the pustules to one another we have a criterion which, while it is itself based upon their number, affords a natural division of the most marked kind. Whenever they are so crowded that they run together the disease is said to be *confluent.* This may either be the case over the whole of the body, or only over certain parts of it, or even on but a single region: this is always the face, since the eruption is never more abundant than upon the face. On the other hand, if the pustules remain everywhere distinct from one another the smallpox is described as *discrete.* Between the two forms the state of the face alone decides; and there is seldom any difficulty in calling a case either confluent or discrete. Watson, however, speaks of some cases in which the pustules stand just thick enough to touch one another without coalescing, and would then call the disease *cohering;* and Trousseau alludes to instances in which a few isolated patches are found confluent, but which he would term discrete.

In ordinary discrete, as well as in confluent smallpox, the pustules run through all their stages, unless, indeed, the disease should happen to be cut short by the patient's death. But there is a third variety, consisting partly of discrete and partly of confluent cases, which is characterised by the premature subsidence or abortion of the eruption. For this, unfortunately, there is no satisfactory name. German writers* describe it as "*variolois,*" a word etymologically misleading as well as barbarous; since it is not "like variola," but is true, though modified variola, and capable of generating the severer forms by contagion. In this country it is generally termed *modified smallpox,* because it is comparatively seldom seen except in persons who have already passed through a former attack, or who have been vaccinated. But it is important to know that variola occasionally passes off in exactly the same manner in a patient who is altogether unprotected.

1. In *discrete smallpox* the eruption presents in their most typical forms the characters which have been described as belonging to the individual pustules. Its amount, of course, varies infinitely in different cases, from a few spots that can almost be counted on the fingers up to many thousands. At the time when they are acquiring their areola there is often a considerable degree of swelling of the surrounding parts, especially when the subcutaneous tissue is loose. Even three or four pustules upon the eyelids may cause them to be puffed out like bladders, so that on the ninth and tenth days the patient may be unable to open his eyes. Trousseau cites a case of Van Swieten's in which a single pustule on the prepuce produced phymosis and great difficulty in micturition. He remarks that the absence of a corresponding degree of tumefaction of the rest of the face renders the swelling of the eyelids more conspicuous in some cases of discrete smallpox than in the confluent variety of the disease. At about the same time the pustules first begin to be painful.

As regards the general symptoms of discrete smallpox, the first thing to

* Strictly speaking I think that this is not quite correct. It is true that German writers give *variola modificata seu mitigata* as a synonym for their "*variolois;*" but in practice they confine the use of the latter term to cases of a certain moderate degree of severity, and apply it to all such cases, whatever may be the course of the individual pustules. Thus Curschmann speaks of *variolois* as sometimes leading to destruction of the papilla and to the formation of deep cicatrices, "so that what distinguishes the case from one of *variola vera* is merely the very much smaller number of pustules." We should call such a case one of very mild discrete *unmodified* smallpox. On the other hand, I do not find German writers mentioning under *variolois* the examples of modified confluent smallpox which will presently be described.—C. H. F.

be noticed is that as soon as the eruption has fairly begun to develop itself, the fever which has existed during the initial stage subsides, and the temperature falls within thirty-six hours to normal, or even lower. Its decline is not always quite continuous, being perhaps interrupted by a slight evening exacerbation. At the same time the pain in the back and the sickness disappear, and the patient often feels perfectly well, with as good an appetite as when in health. This lull in the symptoms commonly lasts for three days; at the end of it he is, in all cases except those in which there are but very few pustules, again attacked with shivering and with febrile disturbance, which is known, as the *secondary fever*, or the *suppurative fever*, or the *fever of maturation*. The date at which this sets in seems to be differently stated by writers; Trousseau puts it at the seventh or eighth day of the disease, Watson at the eleventh. It is generally remittent in type, the daily variations amounting to one or two degrees Fahr. The height to which the thermometer now rises varies with the extent of the inflammation in the cutaneous tissues, and therefore roughly with the number of the pustules; even in severe cases it is seldom above 102° or 103°. The pulse is quickened, being at from 110 to 120. The patient at the same time complains of headache, and is restless and sleepless. He is not unfrequently delirious, especially during the first night or two.

It must not be supposed that discrete smallpox is always unattended with danger. Trousseau relates the case of a girl, aged twenty-one, who had passed through a remarkably mild attack, but who was one evening suddenly seized with cerebral symptoms and difficulty of breathing, and in an hour she was dead; and he remarks that when this form of the disease does prove fatal, death occurs at an earlier date than in the confluent form—namely, about the eighth or the ninth day. In ordinary cases of discrete smallpox the fever lasts about three weeks, or even less, and the crusts have fallen by the time that normal temperature is regained.

2. In *confluent smallpox* the eruption necessarily fails to some extent to present its typical characters, at least upon those parts of the body where an actual fusion of its elements has taken place. Even before any definite papules can be recognised, there is often a diffused redness of the face, which in itself could hardly be distinguished from the rash of measles. Watson mentions a case in which the appearances of urticaria, with its characteristic sensations, were at the outset so intermingled with the papules of variola that for twenty-four hours he doubted which of the two diseases was developing itself; and in other instances smallpox has been mistaken for erysipelas. Trousseau remarks that even where there are, in fact, multitudes of papules, the complete absence of intervals between them may render one almost unable to detect any unevenness of the surface by passing the hand over the patient's forehead or cheek. The papules are always smaller than in the discrete form; they coalesce so as to form the most irregular figures and patterns. At a very early period a milky fluid collects in their summits, giving rise to minute flat vesicles. The whole of the face has then a whitish-yellow colour, and its appearance is exactly like that of a mask of parchment, to which Morton, in his 'Pyretologia' (1718, Case xxxviii), long ago compared it. By this time, however, there is already a good deal of swelling, and it goes on increasing up to the ninth day of the disease, and until the features are so altered that it is impossible for the patient to be recognised by his nearest relations. The parts about the ears and the sides of the neck become enormously bloated; the eyelids cannot be opened. This

condition is very painful, and the scalp is often so tender that the pressure of the head upon the pillow can hardly be borne.

Somewhat later, about the eleventh or twelfth day, the hands and the feet become red, swollen, and painful. Trousseau, following certain of the old writers, attached a high prognostic value to this symptom; according to him its absence is almost always followed by death.

The *mucous membranes* generally suffer severely in confluent smallpox. The interior of the mouth not only presents the eruption already described, but the fauces may become covered with a diphtheritic membrane. Laryngitis often renders the voice very hoarse, and it sometimes causes a sudden fit of suffocation, which proves fatal in a few seconds before anything can be done to relieve it. Trousseau mentions three cases of this kind, in each of which death seems to have occurred unexpectedly on the eighth day of an illness that had previously run a normal course.

Another complication, perhaps due to extension of inflammation to the parotid gland, is *salivation*. Trousseau speaks of this as a characteristic feature of confluent smallpox. It begins, he says, about the fourth or fifth day, and goes on increasing until by the ninth or the tenth day one or two quarts may run from the patient's mouth within the twenty-four hours. Even during sleep there is a constant flow of saliva.

Again, ophthalmia is of frequent occurrence, which (unlike the harmless discrete pustules of the conjunctivæ) is often attended with sloughing of the cornea, and leads to permanent blindness.

From the very commencement of the eruptive period, the general symptoms of confluent smallpox run a course which differs from that of the discrete form of the disease. Instead of the temperature falling when the papules come out, it remains at 103° or 104°. There is often violent *delirium*, even during the daytime. It may be quite impossible to keep the patient in bed, except by tying him down with a folded sheet across the chest. If free, he must be most carefully watched, or he is very likely to throw himself out of a window or over the stairs, or to cut his throat with a razor. In those who have been intemperate, the nervous disturbance often assumes the form of delirium tremens.

Convulsions are a frequent and often a very grave complication.

Retching and vomiting often run on throughout this period of the disease; and *diarrhœa* is obstinate and troublesome. The urine contains albumen in a large number of cases.

When suppuration is established, there is a further rise of temperature. Indeed, it becomes higher than in the corresponding stage of discrete smallpox, although, from the patient having all along been feverish, the development of a fever of maturation is less conspicuous.

This secondary fever is the most dangerous period of variola. Few patients die before the eleventh day; the most fatal days are the twelfth, the thirteenth, and the fourteenth. Towards the last there is generally coma; sometimes hyperpyrexia is present, as in a case of which Wunderlich gives a chart, and in which, before its termination on the eleventh day, the thermometer registered 109°. In many instances the immediate cause of death is bronchitis, pneumonia, pleurisy, or pericarditis. Even under the most favourable circumstances the fever runs on for at least ten days longer. Not infrequently, during the third week, the patient falls into a typhoid condition, with sordes, a dry brown tongue, muttering delirium, and subsultus; and he is then very likely to die of exhaustion.

When recovery is to take place, whether from the discrete or the confluent form of the disease, the redness and the swelling of the face subside, as the pustules undergo conversion into crusts. The pain from which the patient has been suffering passes off, but only to be followed by the most intolerable itching. His fever also declines more or less rapidly, being now proportionate to the amount of inflammatory action still going on. He regains his appetite, and becomes once more able to sleep. He opens his eyes, and gradually his features begin to reassume their natural outlines. At the time when the crusts are falling off, or a little later, he generally loses almost all his hair, and if the deeper layers of the skin of the scalp have been involved in the morbid processes there is always reason to fear that permanent atrophy of the hair-sacs may result.

The confluent variety of smallpox usually lasts about four weeks before the fever subsides.

A minor variety of smallpox, which sometimes occurs in confluent cases, is characterised by the occurrence of hæmorrhage into the pustules. This must not be confounded with the "hæmorrhagic variola," which proves fatal at an early period before the proper exanthem has come out (*supra*, p. 202.) By way of distinction Curschmann calls it *variola hæmorrhagica pustulosa*. As a rule, those pustules which are seated upon the lower limbs are the first to show a purple colour. Bleeding presently takes place from the nose, kidneys, intestines, and other mucous surfaces. In women menstruation sets in, or abortion occurs if they are pregnant. The gums become spongy and bleed, as in scorbutus; the fauces show a kind of "diphtheritic" affection, which is attended with a terrible fœtor. The constitutional symptoms are in most respects severe; but although the pulse is very rapid the temperature during the eruptive stage is seldom above 102°, and it not rarely falls to 95°, or even lower, before the patient's death. Curschmann says that this variety of smallpox occurs chiefly in persons over forty years of age, and in such as were previously out of health. He describes it as being almost always fatal; but remarks that in patients who have, in their delirium, got out of bed and walked about during the early part of the eruptive stage, the pustules on the legs may become filled with blood, without the case being particularly serious. A few petechiæ on any part of the surface are not a dangerous symptom.

Another variety of the smallpox exanthem was described by Marson as *corymbose*. His account of it will be found in Reynolds's 'System of Medicine,' but no one appears to have confirmed his observations.

3. In *modified smallpox*—which, as we have seen, is not exclusively confined to protected persons—the course of the eruption is much less regular than in the ordinary forms of the disease, whether discrete or confluent. During the initial stage, however, no distinction can be drawn between it and them: no doubt there is a larger proportion of cases in which that stage is but little marked; but in some instances, at least, it lasts the usual time, and is attended with severe symptoms. But when the papules begin to appear, it is said that the characteristic irregularity is often shown by their being visible upon the trunk or elsewhere before there are any of them on the face; and it is also said that the period during which fresh ones develop themselves is variable, the number being sometimes complete in a few hours, while in other cases they keep coming out even when those which were first formed have already become pustules. And what appears to be still more indicative of this variety of smallpox is the course of the fever at

the commencement of the eruptive stage. The temperature falls, as in unmodified discrete variola, but with still greater rapidity, and without any interruption in its downward path, so that in less than twenty-four hours it is normal. Trousseau and others, however, speak of confluent smallpox running its normal course for ten or twelve days, and then suddenly subsiding, so as to show that it really belongs to the modified form of the disease.

It is thus evident that no definite period can be fixed at which the modified character of a given case of smallpox first becomes apparent. Sometimes the eruption dies away before it has passed out of the papular stage. Sometimes the papules undergo a partial conversion into vesicles, which then rapidly dry up. A special modification of this variety of the affection has long been known as the "horn-pox" (*variola verrucosa*); but writers by no means agree in their descriptions of it, for while Trousseau speaks of the vesicles as drying up and leaving in their places small, hard, corneous projections, which fall by a sort of desquamation between the tenth and the fifteenth days, Curschmann says that after the scabs have become detached, the solid bases of the pocks remain for a considerable length of time as warty elevations, especially on the face, to which they give an ugly appearance.

Again, in yet other cases of modified smallpox, the vesicles pass on into pustules. The change then appears to take place unusually rapidly, and the pustules themselves are often extremely small, and probably never reach so great a size as that to which they may attain in the unmodified forms of the disease.

Complications and results.—The complications of smallpox, besides those already mentioned, are few. Bronchitis is frequent, and hypostatic pneumonia not unknown; pleurisy, when it occurs, is apt to lead to empyema; and œdema of the larynx has occasionally proved fatal. Salivation is frequent and distressing. The febrile albuminuria rarely leads to subsequent nephritis.

The *sequelæ*, beside the pitting, are chiefly various forms of local suppuration, like those which follow enteric and scarlet fevers: abscesses in the skin and deeper organs, furunculi and ecthyma, local gangrene and phlegmonous erysipelas. In severe cases the cornea may ulcerate, and the eye be lost.

Convalescence is, however, usually rapid, and the health and strength are not impaired as after enteric fever.

The *protection* afforded by smallpox is probably the most complete known. Relapses never follow, and although second attacks undoubtedly occur, they are more rare than in the case of typhus, measles or scarlatina.

Prognosis.—The points upon which this depends have been already indicated. They are, first, the number and quality of *vaccination marks*. The statistics of the Smallpox Hospital for twenty years show that where four distinct and pitted scars were present the mortality was only one in two hundred; where three equally good were found, less than 1 per cent.; where three indifferent marks, more than 3 per cent. Two good marks kept the mortality down to 2·3 per cent., two poor ones only to 8 per cent. With one good mark, less than 4 per cent. of the patients died, and with one indifferent mark nearly 12 per cent. At the same hospital the mortality among unvaccinated patients generally was 37 per cent. (Marson).

The second indication for prognosis is the abundance and the character of the *exanthem*. Dr Marson's statistics give the mortality among 2654 of unvaccinated patients as 4 per cent. in cases of discrete variola; 8 per cent. in cases of semi-confluent, including coherent, variola; and 50 per cent. in cases of confluent variola. A purpuric eruption is of most serious omen—most so when it precedes the true exanthem; hæmorrhagic pustular smallpox is also extremely dangerous, but not so dangerous as hæmorrhage in the papular or vesicular stage.

The most favourable *age* is from eight or ten to twenty; even then about 25 per cent. of unprotected cases are fatal. More than half the unvaccinated patients above thirty years old die, and of those above sixty nearly 80 per cent. To children under five smallpox is as fatal as to adults between thirty and forty; about half of those who are attacked die. Marson observed that children who grind their teeth hardly ever recover.

Previous drinking habits, delirium, convulsions, and want of sleep are indicative of danger.

Pregnancy is usually a fatal complication; abortion usually takes place, and the mother dies soon after. Cases, however, are recorded in which both the mother and the child have survived.

Among sixty-seven cases recorded by Morton (before inoculation or vaccination was known), all but two being under thirty years of age, thirty-eight recovered and twenty-nine died—a mortality of 43 per cent. Of four cases complicated by abortion, only two were fatal.

Treatment.—There is no special means of curing smallpox apart from treatment of the fever and of the local complications. The most rigid isolation is of course necessary for the sake of others, and all who come in contact with the patient should be revaccinated. Free ventilation and the utmost cleanliness are the first essentials. Diarrhœa should be checked. Laudanum and morphia are extremely valuable as hypnotics. Ammonia and senega are needed for bronchitis, and stimulants during the suppurative stage, especially when boils or abscesses appear.

Constant watching is necessary, in order to prevent the patient escaping from the sick-room or doing himself an injury in his delirium. But Dr Collie recommends that a restless patient should be allowed to get out of bed, and even to sit by the fire, if he persists in the attempt.

In order to ease the irritation of the skin and prevent scratching, as well as to soothe the fever, warm or lukewarm baths of about 100° or about 85° Fahr. are recommended, followed by inunction with oil or vaseline. The face should be covered with a thick layer of lead or zinc ointment, and the pustules should be pricked as soon as formed, in order to diminish scarring.

Vaccination in unprotected persons exposed to contagion is imperative; and even if performed after the first symptoms of the disease have appeared, it probably has some effect in diminishing its severity.

But the true treatment for smallpox is prophylactic, and will next be considered.

VACCINIA. *Syn.* Cow-pox.—The terrible mortality from unmodified smallpox, and the scarcely less terrible results it left behind—the scarred and hideous features, the sight injured or lost, permanent deafness, and other disastrous results—can only be appreciated by those familiar with the general as well as the medical literature of the last century. Treatment of

the disease had been much improved by Sydenham, but its ravages were still almost unchecked until the introduction of the practice of *Inoculation*. This has been carried on in China from time immemorial, but it is not known how it was introduced into Turkey. An account of inoculation as a prophylactic for ordinary smallpox was first published in the 'Philosophical Transactions' for 1714, but it became popular seven years later by means of the letters and example of Lady Mary Wortley Montagu, the wife of the English ambassador at Constantinople. By inoculation with the lymph from a variolous vesicle the disease is reproduced with a shorter incubation period, with far less severe symptoms, and with a far smaller mortality than when contracted in the ordinary way.

Variola, however, thus produced was still variola, and as contagious as ever, so that the effect of inoculation was to make cases of smallpox more numerous, though generally milder. Thus, although the mortality was reckoned at only 3 *per mille*, the total deaths from smallpox were increased. The Smallpox and Inoculation Hospital was founded in 1746, and inoculation was practised there down to 1822, but after 1840 it was prohibited by Act of Parliament. The operation was performed in China by putting variolous crusts into the nostrils, but in Europe in exactly the same way as vaccination, except that the lymph was variolous instead of vaccine.

The practice of *vaccination*—the greatest achievement of medicine—was the fruit of the scientific temper and indomitable perseverance of the illustrious Edward Jenner. He observed that the milkmaids in the great dairy farms of Gloucestershire were subject to a particular eruption of the fingers, which was derived from similar pustules on the udder of cows. He ascertained the accuracy of a current belief that those who were so affected did not suffer from smallpox. After long investigation he was so satisfied of the fact that in 1798 he inoculated a boy with the matter from a cow's udder (*i. e.* with the cow-pox instead of with smallpox), and finding that this inoculation was as good a protection from the dreaded variola as the other, he made known his discovery.*

The practice of inoculation with this "vaccine lymph" rapidly spread. It was adopted by the best physicians abroad as well as in England, and the process of "vaccination," thus begun, was at last made compulsory in all civilised countries. Vaccination does not absolutely protect from smallpox: inoculation did not, and an attack of smallpox by ordinary contagion does not absolutely protect. But variola, like other exanthems, protects in the vast majority of cases against a second attack; inoculated variola is a mild and comparatively safe form of the disease, and likewise protects from further attacks; vaccination is a still milder and perfectly safe form of disease, and likewise protects from future smallpox.

The statistics given on page 209 show the truth of this assertion. It is also proved by the following example, quoted by Dr Eustace Smith. Dr Gayton had 1574 children under observation in the Smallpox Hospital at Homerton (1871–78). Of these 211 showed good vaccination marks, and only one of them died; 396 showed incomplete vaccination, and 39 of them died; 179 had been vaccinated, but showed no scar, and 46 of them died; while of 788, who unhappily had never been vaccinated at all, no less than 385 died.

* His 'Inquiry into the Causes and Effects of the Variolæ Vaccinæ, a disease discovered in some of the western counties of England, particularly Gloucestershire, and known by the name of the Cow-pox,' was published in 1798.

Similar evidence on a large scale is afforded by the compulsory vaccination carried out in the great Continental armies, by the results in our own army, in the military school at Chelsea, and by observations in Sweden, in Germany, in France, in the United States, and in India.

It is certain that vaccinated persons are to a small extent liable to smallpox, especially if the scars are few and slight, or if the time since vaccination has been very long. Efficient vaccination is necessary to obtain the full benefit of Jenner's discovery. A second vaccination is desirable about the time of puberty, and repeated vaccination is prudently submitted to by those who are going into contact with the disease, as doctors and nurses taking duty at a smallpox hospital.

With delicate persons the local inflammation attending vaccination may be severe, and cause some febrile action and several days' discomfort. With feeble infants the effects may be more serious. And it is now certain that vaccination from a syphilitic child may introduce the virus of syphilis as well as that of vaccinia, especially if blood as well as lymph be inoculated. This is, however, an occurrence so excessively rare that while every precaution is rightly taken against it by choosing healthy infants from whom to take vaccine lymph, the practical risk is infinitesimal.

A much more common but less certainly ascertained drawback to vaccination is that the slight febrile disturbance which attends it may call forth local or general disorders, especially eczema and catarrhal inflammations. Swelling of the axillary lymph-glands of the vaccinated arm and erythematous œdema of the arm itself are frequent, and sometimes, especially in adults, troublesome. Erysipelas or pyæmia is a possible result, but not more frequent than after any other slight puncture.

It has been suggested that these drawbacks, slight as they are, might be avoided by vaccination direct from the udder of a heifer affected with vaccinia instead of from a vaccinated child; and this plan has been extensively carried out. But its disadvantages are greater than its advantages. The operation is less certain of success, the febrile disturbance is usually more marked, and the maturation of the pustule and falling of the scab is considerably delayed. Hence "arm-to-arm" vaccination, from a healthy child to a healthy child, is probably the most efficient, least disturbing, and most practically eligible method.

Course of vaccinia.—When vaccine lymph has been introduced into the lymphatic spaces of the skin, an incubation period of two days follows. On the third a papule appears, which in two days more becomes a vesicle. Towards the end of the first week a large umbilicated vesicle forms, and attains its full development on the eighth day, when a congested halo surrounds it. The lymph then becomes turbid, its contents gradually dry up, the areola fades again, and by the end of the second week a scab has formed. This falls off about the end of the third week, and leaves a pitted depressed scar, at first darker but finally whiter than the surrounding skin. Meanwhile slight pyrexia has begun on the fourth or fifth day, increases at the beginning of the second week, and falls with the maturation of the pustule and the disappearance of the surrounding inflammation.

This process is sometimes slower, as when the vaccinated child is already in the incubation stage of another febrile disorder like measles; and it is sometimes quicker, or otherwise disturbed, as when the child is already affected by some inflammatory affection like eczema.

The course of a second vaccination is almost always rapid, and the

constitutional disturbance usually greater, owing perhaps to the patient's age.

Nature of vaccinia and pathology of vaccination.—Jenner himself believed that cow-pox was modified variola. This was long disputed, but experiments made by Mr Ceeley (1839) and by Mr Badcock (1840) went far to establish the fact. It is therefore supposed that variolous lymph inoculated on a cow's udder produces vaccinia; this inoculated in a human arm reproduces itself and protects from subsequent vaccination, as well as from subsequent smallpox. Yet it is remarkable that the careful experiments more recently carried out at Lyons by Chauveau throw fresh doubt on the question. He found that inoculation of vaccine lymph produces vaccinia in the cow, and that variolous inoculation produces variola. But the latter disease could not be thus propagated, and when both inoculations were made together the variola died out, while the vaccinia could be indefinitely continued. Cows only, not oxen, are subject to vaccinia, because the contagion is conveyed to them from human variola in the process of milking. The disease is only derived by cattle from human beings, and dies out when not thus reinforced. It may also affect horses and probably sheep, though the variola ovina is in certain respects different. The vaccinia of cows is probably the same as one of the diseases known as the "grease" in horses.

If vaccinia is true variola modified by "cultivation," it furnishes the first and hitherto the most successful example of the method of protection from specific diseases by previous inoculation with an "attenuated" virus.

For further details on the subject of vaccination, so interesting as an example of scientific experiment and reasoning applied to medicine, so important in its bearing on general pathology, and so deeply affecting the lives and happiness of the human race, the reader is referred to Dr Seaton's article in Reynolds' 'System,' and his 'Handbook of Vaccination' (1868), to Mr Simon's Reports to the Privy Council relating to the history and practice of vaccination, and to Mr Ernest Hart's excellent popular pamphlet.

VARICELLA*

"Each little pimple had a tear in it
To wail the fault its rising did commit."
DRYDEN.

History of the recognition of Chicken-pox—Its distinction from Smallpox Incubation and onset—Characters of the eruption—Symptoms and course Sequelæ—A disease of children—Diagnosis—Prognosis.

AT the end of the seventeenth century, soon after the final separation measles from smallpox, English writers mention a variety of the lat disease popularly called "chicken-pox." The same affection seems have been described in the sixteenth century by Vidus Vidius, and Ingrassias, under the designation of "Crystalli." Vogel (1764) is said have introduced the name of Varicella. In 1730 Fuller asserted th chicken-pox and smallpox were really distinct diseases; but Heberden, the first volume of the 'Medical Transactions of the College of Physician (1767), first indicated fully the differences between them. He also point out the chief reason which makes the recognition of chicken-pox importa namely, that those who had it might otherwise be deceived into a fa security, which might prevent them from keeping out of the way of t smallpox.

After the introduction of vaccination it became of great consequence distinguish the two diseases, since every case of varicella occurring in vaccinated person would otherwise have been regarded as an instance of t failure of the operation. But it is to be regretted that some of the earli advocates of Jenner's method, being anxious to show that the protecti afforded by it was absolute, referred every suspicious-looking eruption persons who had been vaccinated to chicken-pox. This, in its turn, led a reaction, since it necessarily involved the giving up of all the distincti characters of the affection, and the consequence has been that its claim be regarded as an independent disease has been disputed by several emine physicians, from Dr John Thomson, of Edinburgh (1820), to Hebra, Vienna. Unfortunately, too, the great German dermatologist helped throw the subject into confusion by employing the term varicella in entirely new sense, namely, for all very mild cases of smallpox. It certain that the varicella of other writers is unconnected with smallpox.

The proofs are (1) that it occurs in those who have been vaccinated, who have had variola, just as readily and with the same characters as those who are unprotected; and (2) that a person who has passed through remains as susceptible as before to the vaccine virus or to that of smallpo In the 'Lancet' for 1877 a case is recorded of an unvaccinated child w

* *Synonyms.*—Variola crystallina, spuria, volatica, Variolæ pusillæ (Heberden), Chicken-pox.—*Fr.* Varicelle, *Germ.* Windpocken, Wasserpocken. The word *varicella* evidently intended as a diminutive of variola. The vernacular name is probably a corr tion of *chickpease* (French *chiche*, Latin *cicer*), in allusion to the size of the vesicles.

was admitted into St Thomas's Hospital for chicken-pox, but who was placed on the floor containing the smallpox wards, because the diagnosis was at first uncertain. Two days afterwards vaccination was performed, which succeeded. Eight days later still the child fell ill with modified variola. According to Thomas, Czakert vaccinated with success a boy in whom the inoculation of the cow-pox had failed on three previous occasions, by introducing the lymph into the interior of the vesicles of a varicella with which he happened to be attacked. Indeed, if it were not that at the present day almost everyone is vaccinated in infancy, we should have in the cow-pox virus an almost infallible test, which we could apply at our own discretion, and which would soon clear up all doubts as to the nature of any individual case that might be difficult of diagnosis.

Whereas the fluid from even the mildest eruption of variola is capable of conveying the disorder by inoculation, almost all of those who have tried to inoculate varicella in the same way from its vesicles have failed. Early in the present century Bryce performed this experiment upon children who had never had either smallpox or cow-pox, to the number of thirteen, without any result. It is true that, in a long series of cases collected by Hesse in 1829, there was a small minority in which inoculation appeared to succeed; but according to Thomas there are reasons for thinking that some error crept into these observations.

The characters of the eruption of varicella, and the date at which it develops, are in themselves ample proof of its distinctness.

Incubation.—This seems to be of variable duration, so far as has been ascertained; but it has probably been calculated upon the precarious basis of the interval between the dates at which different children of the same family have been successively attacked. Dr George Gregory stated it at from four to six days; Heberden at eight or nine. Bristowe says that in some cases it lasts exactly a week, but perhaps more commonly a fortnight. According to Thomas, it may be from thirteen to seventeen days; according to Gee, about a fortnight; according to Liveing, variable, about thirteen days with eruption on the fourteenth. Eustace Smith gives seven to fourteen days; Trousseau, from fifteen to seventeen; Dukes, fourteen to nineteen; Eichhorst, thirteen to sixteen.

Onset.—This is usually simultaneous with the appearance of the eruption, so that there is no stage of invasion of the fever before the exanthem appears, as in typhus, enterica, scarlatina, measles, and smallpox.

Thomas took the temperatures of children who afterwards developed varicella (no doubt where the disease was already in the family), and always found them normal or not raised above half a degree (Fahr.), which in childhood is of common occurrence in health.

Even where there are slight prodromal symptoms, as headache or cough, with loss of appetite and feverishness, they only last a few hours before the characteristic rash appears, so that the period of incubation must, as in the case of Rubeola (*sc.* notha) be reckoned up to that of eruption.

In some instances, however, Thomas found that the child was feverish for a few hours before the disease made its appearance, and sometimes the fever lasted two or three days, and was accompanied with delirium or (as in a case which the writer saw some years ago) with convulsions.

The exanthem.—This begins as a series of small, slightly pointed red spots, which Trousseau compares with the rose-rash of enteric fever. Dr Gee says that they disappear when the skin of the part is stretched, this

being a proof that there is no exudation into the tissue of the cutis, but onl hyperæmia. In a few hours they pass into as many transparent ten vesicles, round or oval in form, and about as large as split peas. The sometimes have a red base; sometimes they are seated upon a perfect colourless surface, so that the patient looks exactly as if he had been sprinkl with drops of clear water. They are generally scattered quite irregularl but it is said that they may occasionally be somewhat clustered together, as herpes. A chief distinction between them and the vesicles of smallpox is the superficial position: they have no thickened floor, they do not consist of series of separate chambers, and when they are pricked they almost co pletely collapse. Thomas, however, says that when they first appear this not the case, and that they are at that time intersected by delicate sept Most of the vesicles of varicella are rounded, without the central depressi of smallpox; but in almost every case some may be found umbilicated, a occasionally most seem to be so. They appear in crops, which come out succession during the first, second, and third nights of the disorder. At leng they acquire a yellowish appearance, and the fluid in them turns slight opalescent, or may become puriform. After from twelve to twenty-four hou they begin to dry up and become flaccid; they are often ruptured, eith by the nails of the patient (for there is often considerable itching) or some other way; or they fall in first at their centres, so as to acquire spurious umbilicus. Ultimately they form thin brownish-yellow scabs, whi in a few days crumble away and leave reddish pigmented spots.

The eruption of varicella generally appears first on the upper part of t *back* or on the chest, rarely on the face. Formerly it was said to spa the face altogether; and although this is incorrect, it never comes o more thickly there than elsewhere, as is the case with variola. On the sca it is almost always present. Afterwards it spreads to the limbs.

The vesicles come out, not in a single crop, but in a succession of crop which may be prolonged over three or four days or even a whole week. Th one sees papules and vesicles and crusts side by side at the same time; in th it is very different from smallpox. The total number of vesicles is sometim not more than from ten to thirty, but according to Thomas it is general two hundred or two hundred and fifty; as many as eight hundred are sa to have been observed. Only a few come out on the first day—perhaps score. Then a hundred or more fresh ones are seen the following mornin The scabs which form as the vesicles dry up, fall off if left to themselves a week or ten days, and leave reddish marks which gradually disappea but sometimes a few small white cicatrices remain behind; and these m occasionally be depressed, and even pitted.

The *mucous membranes* take part in varicella. Thomas says that co plaints of pain in micturition have often led to his observing vesicles upo the labia of girls, but that on the prepuce in boys they are less commonly be found. On the palate they often persist for some time, having slight reddened bases; but on the lips, the tongue, and the cheeks they are to recognised only in the form of excoriations or small superficial ulcers.

Its varieties.—Some of the papules which, as we have seen, constitu the earliest stage of the eruption of chicken-pox, not infrequently abort a disappear, especially those which come out towards the end of the disea Thomas has recorded a case—the nature of which was established by t fact that the patient's sister had just before had varicella—in which eve single papule died away after thirty-six hours, so that no vesicles were forme

On the other hand, the vesicles sometimes increase in size until they deserve the name of bullæ; such an enlargement of them does not always begin until they have already become scabbed over, and they may go on spreading at the periphery of the crusts for a considerable time. Indeed, it would seem that in exceptional instances varicella lasts much longer than any other exanthem. Trousseau speaks of an epidemic in the Necker Hospital in which, during from fifteen to forty days, blebs like those of pemphigus kept appearing on different parts of the patients' bodies, leaving ulcerations which lasted for six weeks or two months. Mr Hutchinson ('Lect. on Clin. Surgery') saw a rash exactly like fading chicken-pox, which was said to have been out during a month, and which vanished spontaneously afterwards.

Course.—The general *symptoms* of varicella are very slight. In some patients the thermometer fails to rise above normal during the first twenty-four hours, while the vesicles are coming out. In others there is slight fever of two or three days' duration, but it often passes off in a single day. Moderate fever may last four days. It is particularly to be noticed that the temperature in chicken-pox does not fall, as it does in variola, when the eruption begins to develop itself. Trousseau, however, speaks of successive onsets of fever, occurring regularly at night, and accompanying the different crops of vesicles; and Thomas seems to have observed something of the same kind, although he speaks of such exacerbations rather as tending to interrupt the usual nocturnal rise and morning fall of temperature. The maximum temperature is about 102°. In some slight cases there is said to be complete absence of fever throughout the course of the eruption. The defervescence, when it occurs, is usually rapid.

All that need be said about other symptoms is that the patient may be a little restless or drowsy, but that he seldom complains of headache or wishes to be kept in bed. The tongue is clean, but there may be some thirst and loss of appetite. In a child suffering from chronic laryngitis, Dr Gee noticed decided increase of dyspnœa during an attack of varicella.

Sometimes, however, the symptoms are more marked, perhaps more in children above six or seven years old, or in adolescents.

In a case seen by the writer in 1886, the patient, nineteen years old, felt unwell one day, but went out to dinner and was then obliged to leave the table, and fainted. Next morning the rash appeared on her face and shoulders, and continued in successive crops. The highest temperature was 102° F., and it did not rise with the development of the eruption. There were good vaccination marks. The febrile disturbance lasted about three days. One scar only remained on the forehead close to the hair, where a scab had been repeatedly scratched off. There was an epidemic of chicken-pox among children in the village, one of which the writer saw.

Relapses and sequelæ.—Some writers speak of relapses of varicella; and Thomas, although he denies that the disease is ever followed at once by a second complete attack, admits that he has seen a few vesicles appear as late as a month from the commencement of the eruption.

The question bears on a remarkable cutaneous affection which Mr Hutchinson believes to arise out of chicken-pox, and which he therefore terms "varicella-prurigo." He has recorded notes of sixteen cases, all of which are said to have begun with the characters of varicella, most of them having in fact been diagnosed by medical men as examples either of that disease or of modified smallpox. But it is curious that it never affected several children in the same family; indeed, there is only one instance out

of the whole number in which more than one child suffered from the origina disease; and any doubts as to the correctness of Mr Hutchinson's views ar strengthened by the fact that he gives twelve other cases in which the sam skin disease was supposed to have been caused by vaccination. There is n doubt, however, of the validity of Mr Hutchinson's observation of gangren as a complication of varicella, though it is happily a very rare one. In stead of the vesicle running the usual course, it becomes tinged wit blood, and the scab is very large and black. The slight areola is mor marked and of a dusky hue, and under the crust is an ulcer. Thes multiple gangrenous ulcers may prove fatal in young children.

Predisposing causes.—Chicken-pox is almost confined to *children*. D Gee gives a table drawn up from the records of the Great Ormond Stree Hospital, according to which infants under six months of age were ofte attacked, while there was a much larger number of cases among childre between six and twelve months old than during any other period of equa length. Most patients are under six, and after ten years of age it ver rarely occurs. Most writers say that they have never seen it in grown-u patients, but Heberden relates one case in which a mother caught it fro her children, and another instance of it in an adult female was observed b Gregory. Among 584 patients recorded at Basle from 1875 to 1880 b Baader (quoted by Eichhorst), 382 were under five years old and 573 unde ten, 7 were between eleven and fifteen, and only 2 certain cases under twenty

Varicella is sometimes sporadic, sometimes epidemic. Thomas remark that in large towns epidemics are not separated by intervals of several year (as with measles and smallpox), but occur every year or every half-year—i Leipzig regularly a short time after the opening of the infant schools.

This disorder does not affect any particular season, although Heberde speaks of it as occurring chiefly at the end of summer, and Gee thinks it i most common in the fall of the year, in September and October. It is highl infectious. The contagion is probably communicable through the air, and i also capable of adhering to solid bodies. That it possesses comparativel little tenacity may perhaps be inferred from the fact that the extent of a epidemic is not generally large.

Varicella protects efficiently against itself, but not against vaccinia o variola. Second attacks of chicken-pox are almost unknown.

Diagnosis.—The diseases with which varicella has been confounded ar acne, pustular syphilide, and molluscum contagiosum, to distinguish whic needs only knowledge and care—and variola, especially variola in vaccinate persons, and here the distinction is sometimes difficult. The slightness o the early symptoms, the close sequence of the exanthem, the softnes of the papules, their appearance in successive crops, their distribution an rapid course will generally decide the question. If on the second or third day there is a vesicle, or a crust on the fifth, it cannot be variola (Heberden).

The *prognosis* is always good except in the rare cases of subsequen gangrene. There are no other complications and no proper sequelæ; bu occasionally children are some time in regaining strength, and both Gee an Eustace Smith have seen tuberculosis follow chicken-pox.

No *treatment* is needful, except to ease the troublesome itching. The vesicles should as far as possible be protected from scratching, to prevent deep cicatrices. Since the contagion of varicella is very active, isolation i desirable, particularly when young children are in the house.

THE PLAGUE*

"The pestilence that walketh in darkness."

History and geographical distribution—Incubation, onset, and symptoms—Diagnosis—Anatomy—Fatality—Treatment—Conditions of contagion and causes of the decline of the plague.

THE SWEATING SICKNESS.—*History—Symptoms and pathology.*

THE English word Plague was formerly applied indiscriminately to every fatal epidemic disease; and when the malady now to be described appeared in the fourteenth century, it was called the "Black Death." But for the last 200 years the only other epidemics comparable with those of former times have been separately named Typhus, Cholera, Yellow Fever, &c. Consequently we can now use the word "Plague" without ambiguity for what is more precisely termed the "bubonic" or "Oriental Plague."

The history of the true plague, identified by its mortality and its buboes, can be clearly traced back to an Egyptian epidemic under Justinian, about 550 A.D., and notices by medical writers show that it was recognised as early as the time of Trajan. It seems improbable that the "Plague of Athens" described by Thucydides was the Oriental Pestilence.†

Epidemics of the plague were frequent during the Middle Ages, when it was probably almost constantly endemic in Persia, Syria, and Egypt. The Black Death of 1348–49 began in China, spread to the Crimea, and thence by a ship's crew to Genoa, devastated the whole Continent, and put a stop to the war between England and France. Its ravages in Florence were recorded by Bocaccio. It probably reduced the population of England by one third, or even more, and led to important economic results. The same epidemic invaded Syria by way of Armenia, and thence spread over Egypt and the whole of Northern Africa, the hotbed of plague in the days of the Roman empire. There were several epidemics in the fifteenth and sixteenth centuries, and again in 1609, in the first year of Charles I (1625), and in 1635–47; but the most destructive and happily the last in England was the Great Plague of London in 1665, so graphically described by Defoe, although he was but two years old at the time, and could only know what he tells by hearsay. In 1665 no less than 68,596 deaths from the plague were reported in the bills of mortality.

There was a terrible epidemic in the south of France, known as the Plague of Marseilles, in 1720, when the scenes described by the great English novelist were renewed. The disease did not visit Western Europe

* *Synonyms.*—Pestis vel Pestilentia, λοιμός, Pestis inguinaria, Typhus bubonicus, Black death, Levantine or Oriental plague.—*Fr.* la peste, *Germ.* die Pest.

† There is no mention of buboes or of hæmorrhage, and the symptoms recorded are unlike those of the Oriental plague. Murchison believed the plague of Athens to have been typhus, Littré and Daremberg that it was smallpox, and a book has been published to prove that it was scarlatina. It may have been any of these diseases, or relapsing fever, or measles. Except fever and contagion, none of the recorded symptoms fit in with any known malady.

again, but epidemics occurred during the eighteenth century in Sicily and Poland (1743), Wallachia and Russia (1771).

In the early part of this century there were outbreaks of the plague in Malta, at Noja in Southern Italy, upon the Lower Danube, and in the Balkan Peninsula. Up to 1844 they were of frequent occurrence in Egypt, but that country has since been free from them. The disease also vanished from Smyrna and Syria, and hopes were entertained that it had become entirely extinct. But in 1858 it appeared again in Tripoli, on the south shore of the Mediterranean; and it is now known to have visited the highlands of Western Arabia in 1853. There is also reason to believe that it had never been absent for many years together from parts of India; in 1834 an epidemic which occurred in Rajpootana became known as the "Pali plague," and as recently as 1877 it prevailed at Kumaon, on the southern slopes of the Himalayas. Between 1863 and 1876 there were six or seven outbreaks in Western Asia, some in Persian Kurdistan, others upon the Lower Euphrates. In 1877 it showed itself at Resht, to the south-west of the Caspian Sea. From this place it probably made its way to the Lower Volga, where it prevailed, in the district between Astrakhan and Tsaritzin, from October, 1878, to February or March, 1879, and excited great alarm throughout Russia, and in Europe generally.*

This terrible disease, long unknown in Europe, has become rare in the Levant; but it still occurs epidemically at intervals in Yunnan (China), in Kumaon (Northern India), in Irak-Arabi and Syria, in Kurdistan and Northern Persia, in Arabia and in the single town of Benghari, in the province of Barca. It is believed to exist as an endemic disorder in certain parts of Northern India.

The plague does not appear in the tropics, and is stopped by the hot weather in subtropical regions. Nor, with few exceptions, does it survive the cold of winter in European climates. The most favourable temperature is from 60° to 85° Fahr. Dry air above or below these limits seems to destroy the contagion.

It has never extended to the New World, nor to Australia or South Africa, but is not unknown in China.

Course.—The general symptoms of the plague resemble those of typhus. Its *incubation* is believed to be from two to seven, usually from three to five, days. This period is followed by shivering and pains, great malaise, and depression. Intense pyrexia then sets in, the temperature rising to 106° or 107° on the first day; violent delirium is common, and the symptoms rapidly assume a typhoid form. Death not uncommonly occurs at a very early period, before there have been any symptoms characteristic of the malady; it may be preceded by the appearance of petechiæ and large vibices in the skin; there may also be hæmaturia, hæmoptysis, and vomit blackened by altered blood. But if life is prolonged to the second or third day there appear one or more *buboes*, attended with severe pain and tenderness. The seat of this affection may be either the groin (in which case glands belonging to the vertical rather than to the horizontal chain are affected), the armpit, or the neck. According to Liebermeister (in Ziemssen's 'Handbuch') a plague-bubo is sometimes so small as to be detected only on careful examination, but it may attain the size of a hen's egg, or more. Its formation is commonly attended with the subsidence of the delirium and of the fever,

* The English Government sent Surgeon-Major Colville and Dr J. F. Payne to investigate the disease on the spot, but it had already disappeared on their arrival at Astrakhan.

the skin becoming covered with a profuse sweat, and the pulse falling to 90 or 100. It may ultimately subside without discharging; but usually it breaks, or is opened by the surgeon, and gives exit to a mixture of blood and pus, and this may be followed by a tedious process of suppuration, which greatly prolongs the patient's illness.

Carbuncles may be associated with the buboes, but are much less frequent and of more fatal augury. They appear most often upon the lower limbs, on the buttocks, or on the neck; sometimes there are not less than a dozen carbuncles in the same case. Bullæ and pustules may also be seen.

Petechiæ and vibices are usually present over the surface of the body, and constituted the "plague-spots" and "tokens of the plague" in the popular accounts of the pestilence in England. They are more numerous than the petechial spots of typhus, and probably gave rise to the epithet which distinguished the pestilence of the fourteenth century as "the black death."

Pestis minor.—There is a much milder form of this disease, which often precedes or accompanies an epidemic of the plague, and has been called the masked, aborted, or lesser pestilence (*pestis larvalis s. minor, peste fruste*). There is no hæmorrhage in these cases. The buboes are few and chronic, and the pyrexia so moderate that patients usually go about. The illness lasts from a fortnight to a month. Indeed, so slight are some of these cases that they have been regarded as a separate malady; but there seems to be no doubt of their true nature, notwithstanding the absence of cases intermediate in severity. Whether this mild variety propagates itself or not is uncertain.

Diagnosis.—It will readily be understood that to distinguish the plague from other malignant fevers is not always easy, unless the character of a prevailing epidemic has already been determined. Dr Milroy, in Reynolds' 'System,' cites the remark of Heberden that, "on first breaking out, the disease has never been known to be the plague;" and he says that in Constantinople or in Cairo no physician ever ventures to give a name to an epidemic until a case occurs in which a bubo or a carbuncle is seen.* Moreover, as he goes on to point out, similar local affections are sometimes, though very rarely, present in other forms of pernicious fever, whether malarial or contagious. But it is certain that the diagnosis would not have been left open so long as it often has been, were it not for that reluctance to admit unpleasant truths which has always contributed so much to the spread of every infectious disease. According to Dr Milroy, a special difficulty has often arisen, during the prevalence of the plague, from the occurrence of glandular pains and swellings, or of carbuncles, in persons who remain well enough to follow their occupations, and who speedily get well under any simple treatment (pestis minor). Liebermeister remarks that these cases are often observed when an epidemic is declining; the general symptoms are so mild, and run so favourable a course, sometimes without any local lesion appearing, that they may be termed abortive.

Morbid anatomy.—*Post-mortem* examinations throw little or no light upon the pathology of the plague. The viscera are soft and blood-stained, and the spleen is more or less enlarged. The serous membranes are ecchymosed. Some of the internal lymph-glands are enlarged; perhaps a chain of swollen

* In a small treatise, *Pestis descriptio*, by Gratarolus, of Bergamo, published at Paris in 1561, twelve signs of the disease are given, most of which are indicative of any severe fever; but the twelfth is *omnium certissimum: si cum febre post aures aut sub alis aut circa inguina potissimum bubones sine manifesta aliqua causa, aut in aliis etiam partibus carbunculi subito oriantur.* Among the other symptoms *sanguinis sputum* is mentioned.

glands, extending up from the pelvis along the front of the spine, or a mas lying in the mediastinum; their tissue is of a bright red colour, or full o points of suppuration; and blood may be diffused and extravasated into th surrounding structures.* Briefly, the autopsy is one of typhus with th addition of swollen and suppurating lymph-glands.

Prognosis.—The fatality of the plague exceeds that of all other disease It usually destroys from 70 to 90 per cent. of those whom it attacks; ver seldom less than 60 per cent. Moreover, it often carries off half the popu lation of a town or of a district in which it prevails, and it may completel root out whole families, so that no survivor remains. The "Riley Graves, near Eyam, in Derbyshire, still tell of the tragedy that befell two familie in that place during the epidemic of 1666: one, that of the Talbots, con sisting of seven persons, was utterly eradicated within twenty-five days; th other, that of the Hancocks, lost seven out of eight members, the only on left being the mother. It is, however, probable that no small part of th excessive mortality from this disease was due to the fact that, instead o isolating each case at the earliest possible moment, the ancient practice wa to close up every infected house, preventing both ingress and egress, an confining the sick and the healthy together. Those who were attacked wer often left to take their chance of death or recovery. The most favourabl cases have been observed towards the decline of the epidemic.

The contagion.—That the plague is propagated by an infective viru derived from the bodies of the sick is now doubted by no one, although i former times there were "non-contagionists" with regard to this, as to ever other disease. Reasoning from analogy, we should suppose that the *conta gium* is one of the Schizomycetes, but none have yet been discovered in th blood or tissues.

The contagion is persistent as well as active, but it seems to be readil dissipated by the air, and rather to belong to the dwellings than to the bodie of the sick. For many of the clergy and physicians who remained firm to thei duties in the great plague of London escaped, and the same has been notice in more recent epidemics; while, on the other hand, those who live in th same house or room with persons stricken from the plague almost constantl fall victims to the same malady. Moreover, the virus clings to dwelling even after their inhabitants have died or have taken flight. This has led t the belief that the plague belongs to the group of miasmatic contagious dis eases (p. 12); that while the virus always takes its origin from patients, i may persist outside the human body, in the soil or elsewhere. Liebermeister indeed, advocates the opinion that its mode of diffusion is, as a rule, thu "indirect." The statement commonly made that it is inoculable might b supposed to settle the question; but the results of the experiments whic have been made on this point are far from conclusive.

The virus seems often to be conveyed by articles of clothing, wool silk, hair, paper, books, &c. In 1665 the local epidemic at Eyam, i Derbyshire, so well known by the heroism of the vicar, Mr Mompesson, began with the case of a tailor who had just received a box of clothes from London (150 miles off), and who was watching them hanging before the fir when he was taken ill; at that very time the disease in the metropolis wa at its worst. The bedding and the linen of those who have had the plague are particularly apt to be carriers of infection to other persons, sometimes

* The first account of an autopsy is given in a curious work by Dr Geo. Thomson called 'Loimotomia, or the Pest anatomised,' published in 1666.

after a considerable interval of time. When epidemics used to occur in Western Europe, the notion was that it reached Holland direct from Turkey in bales of cotton or silk, and that it was brought over to England from Holland in a similar way. Nevertheless the exportation of cotton from Alexandria to Liverpool and Marseilles during the prevalence of the plague in Egypt in the year 1835 was never prohibited, and yet no cases appeared in England or in France.

Predisposing causes.—Many collateral circumstances, however, greatly influence the spread of the disease. It is said to have been especially apt to prevail where the soil is alluvial and marshy—as, for example, along parts of the Mediterranean coast, and in the basins of great rivers. Warm and damp weather is favourable to it; thus in European Turkey it occurred chiefly in the spring and in the early summer, whereas in Egypt the winter was the chief season for it. In Nubia and in other countries with a hot dry climate it has hitherto been unknown.

Overcrowding, dirt, and poverty seem greatly to encourage its development. It affects especially the poor; and at Malta in 1813 it was noticed, according to Dr Milroy, to be far less common in the upper stories of the lofty houses of Valetta than in the basements. The higher parts of a district sometimes escape when the lower are infected with the plague; thus it is said that the citadel of Cairo and the village of Alem Dag, near Constantinople, used to remain free, although communications with the cities below were not interrupted. Lastly, it seems less apt to occur in persons over fifty years old than in those who are younger, and at least in English epidemics affected more adults than children.

Prophylaxis.—It is greatly to be hoped that the improvement in the hygienic conditions of the inhabitants of Western Europe, which has been going on during the last two centuries, has been the chief reason why the plague no longer appears among us, and that Hirsch is wrong in attributing this result mainly to the system of quarantine. For it is certain that at the present day, if the disease were again to travel towards our shores, there would be extreme difficulty in maintaining that system effectually. Liebermeister cites in detail the proceedings that were adopted to prevent the extension of the plague from Noja in Italy in 1815. The town was surrounded by two deep ditches, a triple military cordon was drawn round it, the soldiers had orders to shoot down anyone who attempted to pass, and no articles were allowed to be sent out except letters which had been first dipped in vinegar. The advantage to be derived from such measures is well illustrated by instances in which large bodies of persons isolated in the middle of an infected city have escaped. Thus Dr Aitken says that during an epidemic at Marseilles a large nunnery was "shut up," with the result that all the inmates remained free, although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other side for those who died of it. This writer, however, speaks of quarantine as a barbarism and as an unwarrantable nuisance; and it is well known that many of the best English authorities hold similar opinions. Indeed, the chief reason why our Government from time to time enforces quarantine regulations, in our possessions in the East and elsewhere, is that if we did not do so, other countries would at once refuse intercourse with our more exposed ports, on the mere chance of our becoming a source of danger to them.

If what has been said above of the persistence of the contagion in houses

is true, we can understand why the Great Fire of London, which destroye most of the City in the year succeeding the Plague, has been followe by complete exemption from the disease. And if the plague is trul miasmatic-contagious, the neglect of cleanliness, and particularly the d filement of the houses and soil which is characteristic of Eastern town and villages, may be an essential factor in its production.

THE SWEATING SICKNESS.—A few words may be added with referenc to another disease more obsolete than the Plague, which affected Englan in the sixteenth century.

The *sudor anglicus*, as it was called, was an infective febrile epidemic di order characterised by a rapid course, profuse perspiration, and limitatio to this island, or rather to its inhabitants, for it was said to single out ou countrymen wherever they might be abroad.* It first appeared in 148 and was supposed to have been introduced by the motley host which Henr VII led to Bosworth field; for the first recorded cases happened between h landing at Milford Haven, on August 7th, and the battle a fortnight afte wards. On the 28th it broke out in London, and at the same time Oxford, and spread over the whole country with a great mortality. ended as suddenly as it began in December of the same year. The secon epidemic was in 1507, the third in 1517, the fourth in 1528, and th alone spread to Hamburg and thence to all northern and central Europ devastating Scandinavia, Poland, Hungary, and Switzerland, but sparin France, Italy, and Spain, and most of Germany. The fifth and la epidemic was confined to England, Calais, and Flanders. This w described by Dr John Kaye (better known as Caius); it, like previou epidemics, is said not to have invaded Scotland or Ireland.

The onset of the "English sweat" was marked by rigors, headache, a pains in the back and limbs; after a short cold stage like that of ague the followed sudden and profuse diaphoresis with rapid pulse, thirst, and doubt raised temperature. No exanthem is noted, and there is no mentio of sudamina. The course of the malady was remarkably short; fatal cas ended in a few hours with deep sleep and signs of collapse; after twenty-fo hours the danger was nearly past. Relapses, however, sometimes occurre

Various guesses as to the ætiology of this curious disease were mad but it seems pretty certain, first, that it was not due to any of the suppos causes (the English constitution, dirt and squalor, moist seasons, Briti fogs, Continental heat, or too good living), and secondly, that it was (wh Caius denies) an infectious disorder, spreading by human intercourse.

Dr Payne, in an able article on the subject in the last edition of t 'Encycl. Britan.,' argues that this strange epidemic was not really an isolat phenomenon, but closely related to "Miliary fever" (*Schweissfriesel*, *sue miliaire* or Picardy sweat), an epidemic disease of short duration marked fever, sweating and sudamina, not very uncommon in France, Italy, a Germany during the last and the present century; and apparently endem in Northern France. If so, we have a close parallel to the endemic *pes minor* and the epidemic plague, as well as to endemic and epidemic chole and endemic and epidemic measles.

* Dr Payne remarks that this was either because they brought the disease with the or if settled in foreign parts they were merchants in communication with England.

CHOLERA*

"Hæc igitur subito clades nova pestilitasque
Aut in aquas cadit aut . . . . ."

LUCRETIUS.

History—Name—Onset and early symptoms—Stage of collapse—Stage of reaction: the urine at this stage—So-called "cholera-typhoid"—Cholerine—Complications—Post-mortem conditions—Pathology—Ætiology—Theory of contagion—The Comma Bacillus—Propagation of the disease—Diagnosis—Prognosis—Treatment.

IN the winter of 1817–18 there appeared in the camp of the Marquis of Hastings, then engaged in the Mahratta war on the banks of the Sind, a very fatal malady attended with vomiting and purging. Cholera, as it was called, appeared the same year at Jessore in Bengal, and there also was regarded as a new and unheard-of pestilence; but there is reason to believe that it prevailed in India from time to time during the eighteenth century, and indeed as far back as history goes. Mr Macnamara cites numerous notices of cholera epidemics both before and after the appearance of the English in India. During the next few years (to 1823) it spread slowly over a large part of Asia.

The first appearance of Cholera in Europe was in 1830, when it spread from what was apparently its endemic seat in Northern Persia to Russia, and thence to Germany. In October, 1831, it reached Sunderland, and London in the following January. After spreading over the whole of Europe, the new pestilence crossed the Atlantic and was very fatal in the cities of the United States and of Brazil. This epidemic was not over until 1837. Its first outbreak at Paris occurred in the midst of the carnival, and everywhere it came with such suddenness and was so rapidly fatal that it created as much terror as the plague in former times.

The second European epidemic was in 1847–48, when the mortality in London was very great. The well-known surgeon, Aston Key, was one of the victims.

The third, like the others, began in the East and slowly spread to England in 1853. It was one cause of mortality during the Crimean War, particularly in the French and Sardinian armies, and was very destructive in America. In London its effects, though severe, were remarkably confined to certain districts.

The fourth epidemic was in 1865–66. The last important invasion of Europe by Cholera was in Italy and the South of France and Spain (1884–86). At Naples and at Marseilles it was very severe.

Certain regions have hitherto escaped Cholera: Iceland, the Orkneys in Europe, Chili and Peru in the New World, the Cape Colony in Africa, and the whole of the Australian continent.

* *Synonyms.*—Epidemic, Asiatic or Indian, malignant or blue cholera; Cholera pestifera, Cholera morbus, Cholerica passio. In India the word *morxi*, used by the Portuguese in the sixteenth century, probably denoted cholera. The French in the Deccan called it "*mort de chien.*"

Name.—The term "cholera" does not seem to occur in the Hippocra writings, but it is used by Aretæus and by Alexander of Tralles. T description by the former writer (De causis et signis acutorum morboru lib. ii, cap. v) refers to severe diarrhœa with vomiting; the evacuations said to be first fæcal, then mucous (φλεγματώδεα, pituitosa), and las bilious (χολώδεα); pains, cramps, scanty urine, feeble and frequent pu are given as the symptoms which accompany this disease; none of th characteristic of Asiatic Cholera, as distinct from severe diarrhœa.

The word χολέρα is usually and probably rightly derived from χο and means bilious diarrhœa. So Celsus understood it: *bilis supra infra erumpit, primum aquæ similis, deinde ut in ea recens caro lota esse videat interdum alba, nonnunquam nigra vel varia. Ergo eo nomine morbum h* χολέραν *Græci nominarunt* (lib. iv, cap. xi).*

The name applied to the new disease was extremely inappropriate, Indian cholera is marked by an absence of bile in the matters vomit or discharged from the bowels. For a time the epithet "Asiatic," applied to the new malady by way of distinction from so-called "Engl Cholera." But of late it has become usual to speak of the epidemic order alone as *cholera*, and to classify as diarrhœa or gastro-intestinal cata sporadic cases which are now and then seen, although their symptoms more or less "choleraic" in character.

Course.—After exposure to the exciting cause of the disease, there i short period of *incubation*, which is believed to be generally two or th days, but sometimes not more than twelve or twenty-four hours. Goodeve cites an instance, recorded by Dr Barry, in which a detachment sepoys, on their march from one place free from cholera to another, pas through a village where it was raging; one of the sepoys was attacked af forty hours, and fresh cases appeared subsequently.

The invasion of cholera may be either gradual or sudden.

When the disease sets in gradually, the earliest symptom is genera diarrhœa, which is often called "premonitory," attended with gripi pain and with a sense of exhaustion. In some cases malaise, depressi of spirits, headache, vertigo, noises in the ears, oppression at the epig trium, are present during this period. At the London Hospital in 186 nurse was doing her work as usual when she was attacked with "singing her ears," and nausea; so that she lay down hoping that her sympto would pass off, but they were quickly succeeded by those of chole Another patient there complained not only of noise in the head, but being pained by the sound of his own voice; and a third kept shifting h head from the top to the bottom of his bed every few minutes, trying escape from the ever-pursuing noise. The countenance of a patient duri the premonitory stage is often pallid, anxious, and sorrowful. Annesl cites a case in which the approach of cholera was suspected mainly fr the aspect of the patient nine hours before the characteristic sympto appeared. The duration of the premonitory symptoms varies from a f hours to two or three days.

In many instances this stage is altogether absent, and then the disea

* Alexander Trallianus gives another etymology: *Intestina vero* χολάδες *veteres app labant ut etiam Homerus testatur. . . . Hujus gratia etiam affectum* χολέραν *nuncupar* (lib. vii, cap. xvii). Hesychius's Lexicon (quoted by Liddell and Scott) gives a seco explanation of the word as an application of χολέρα, "the gutter of a roof down wh the rain is discharged:" but this is nonsense.

with startling suddenness. It very often—according to than half the cases—begins in the early morning, perhaps nt up from sleep.

mptom of the developed disease is violent *purging*; the con- els are rapidly swept out in a fluid form, and the discharges ost colourless, like whey, or like water in which rice has at they are commonly spoken of as "rice-water stools." On id deposits a loose whitish-grey material, which consists of containing numerous leucocytes, and immense numbers of ng various bacteria. At one time it was thought that ial cells were present, but this is now known not to be the er fluid discharges during life, although what is found after death is full of them. The specific gravity of the 006 to 1013; it has a neutral or slightly alkaline reaction, ains chloride of sodium, with a very small quantity of ofuse is the flow that Dr Goodeve speaks of the patient as pan of a night-stool in two or three hours, and sometimes pints or even quarts. He remarks that when all that is d in the same vessel, the bile contained in what was first it a yellowish colour. Sometimes the tint is pinkish from f blood. There is often no pain whatever in the bowels, s complain of more or less griping.

erval, or occasionally at the very commencement of the ets in; the fluid rejected from the stomach (unless mixed pale and watery, being in fact identical with the rice-water even have a still lower specific gravity of 1002 to 1005, a large admixture of water taken in during the attack. It ut of the mouth suddenly and with great force.

y symptom is severe *cramp* in the muscles of the feet, in the s, and sometimes in the thighs, hands, chest, or abdomen. es on at intervals and lasts for a few minutes at a time. It nising character, causing the patient to shriek out and to e bed on which he is lying. Lebert says that cramps were a third of the cases observed in the epidemic at Zürich

collapse.—These symptoms are followed by the develop- kable condition, known as "cholera collapse," the "algide nonly appears within six or seven hours of the commence- ging, and often earlier still. Indeed, in some exceptional actually dies collapsed before there has been either vomiting the rice-water fluid is found accumulated in the bowels after ential sign of this state is a failure of the circulation, begin- iphery, but afterwards affecting parts less distant from the e at the wrist becomes more and more feeble and thread-like, ther imperceptible; even the brachial artery may no longer on auscultation, according to Lebert, the second sound of e inaudible, while the first sound is still heard. The surface mes cold, livid, and shrunken. The hands feel like ice, and s though they had been long soaked in water. The features ue, the eyeballs are deeply buried in their sockets, the nose ongue feels cold, even the breath may be entirely devoid of kin is often covered with a profuse sweat.

According to Goodeve a thermometer placed in the mouth indicates fr 79° to 88°. In the axilla, however, the temperature is seldom below 93° 94°, as was shown by careful observations made at the London Hospital 1866 by Mr F. M. Mackenzie. And in the rectum, or in the vagina, t temperature rises through the period of collapse, reaching 100°, or 102°, even 104·4°. Mr Simon, in discussing these facts in his 'Ninth Repo infers that the choleraic affection of the bowels is a "heat-making" proce But there seems to be no reason for doubting that the heat is generated the muscles and in the glandular organs, just as it is under ordinary c cumstances. Mr Mackenzie observed that a severe attack of cramp rai the axillary temperature as much as 2° F. The only question seems to whether the elevated temperature in the interior of the body is due to mere accumulation of heat from deficient loss, or whether there is positive shifting upwards of the point to which heat regulation is set, in true pyrexia. It is a strong argument against the latter view th during reaction, as we shall presently see, when the peripheral circulati becomes restored, the internal temperature falls and is actually below norm Moreover, the collapse of cholera is essentially different from the init rigors of acute specific diseases. One distinction on which Hutchinson l insisted ('Lond. Hosp. Rep.,' vol. iii), is that in collapse the pupils are natural size, whereas in rigor they are widely dilated.

The breathing during collapse is increased in frequency, being at t rate of 24, 30, or even 40 in the minute; and there is often a sensation dyspnœa. The heart's action, however, is but little accelerated, remaini at about 100. There is great muscular weakness. The voice becon feeble and whispering, or may be so completely extinguished that when attempt is made to speak nothing but a movement of the lips follo Lebert thinks that this is due to dryness of the vocal cords, as well as relaxation of their muscles. In some cases the patient has been known sit up, or even walk across the room, very shortly before his death, but a rule, he becomes unable to rise from the recumbent position. There usually great restlessness, the limbs being abruptly tossed and jerked abo

The mental state is generally, from the first, one of indifference rath than of anxiety; and in some cases it is only when the cramps cause t sufferer to cry out that he seems to be conscious of his condition. But can easily be roused to understand what is said to him, and may answ quite rationally. Coma sets in only towards the very last, if at all.

It is to be noted that when collapse has developed itself in a mark form, the purging ceases altogether, or becomes greatly diminished. Probab what is now passed had been poured out into the bowel at an earlier perio Goodeve describes it as often containing gelatinous mucus-like masses. T stomach, however, still remains very irritable. There is the most inten thirst; the patient craves for water, and drinks it with eagerness, only vomit immediately afterwards. Another distressing symptom is a sense burning heat at the epigastrium, often accompanied by great tenderne and sometimes by hiccup. The abdomen is commonly retracted.

Lastly, one of the most marked phenomena of collapse is *suppression urine*, which seems often to be present from the earliest period of the d ease. There is no doubt that the arrest of the renal secretion in cholera a result of the defective flow of blood through the kidneys. Herma and Cohnheim have independently shown that precisely the same thi occurs in animals when the renal arteries are compressed or ligatured.

—Cholera collapse often leads directly to a fatal termination, akes place between twelve and twenty-four hours after the of the attack, but sometimes earlier, and sometimes during

tions that before death the eyes may become dry and the opaque. The internal temperature has often been found has become extinct; and spasmodic twitchings and quiverings have sometimes been observed within the first half-hour ne limbs may actually move; in a case at the London Hospital ow became raised about three inches above the level of the ich it was lying.

Not infrequently, even extreme collapse is recovered from. *ction* usually begins to develop itself at the end of twenty- ht hours. Goodeve remarks that the subsidence of restless- tion is often a favourable sign. The patient dozes quietly ation; then a flickering pulse at the wrist is detected, which es more distinct, the superficial veins on the back of the fill, the surface is felt to be less cold, the features look less colour begins to return. As a rule, the improvement occurs by step. But the older Indian physicians spoke of cases " standing at his door on Wednesday, was in perfect collapse

o Mr F. M. Mackenzie the temperature during reaction, when fairly recovered, is usually about 97° in the rectum or the ° in the axilla ('London Hosp. Reports,' vol. iii). But in he London Hospital in 1866 it was observed that certain face conveyed to the hand a sensation of burning pungent e of which, however, was not determined by the thermo- r point noticed at the same time was that the pulse often even to 45, in patients who were doing perfectly well. other hand, speaks of the pulse as remaining at about 100. es full and bounding, and may be dicrotic. The patient's ar. The cheeks usually present sharply defined patches of the conjunctivæ are deeply injected, the eyes half closed turned upwards, the expression heavy and vacant.

of reaction is by no means free from danger. Sometimes a purging, vomiting, and exhaustion set in again, and end er cases the reaction is said to be *imperfect*. The pulse up to a certain point, remains weak, and the surface of the to be colder than natural. The bowels are still relaxed ions are watery, although more or less coloured with bile. etite and the patient is depressed and drowsy. Sometimes nent symptom is irritability of stomach attended with thirst nsations along the œsophagus and at the epigastrium. entions sleeplessness, lasting for two or three days or more, t discomfort to the patient.

anthem.—In some cases slight pyrexia occurs at this stage, d with the development of a bright crimson or scarlet *rash*, nly spoken of as a roseola, though it may rather have the rticaria. Sometimes it nearly resembles the eruption of Mackenzie found in 1866 that it was always accompanied perature, both external and internal. In one instance the

former was 101·6°; the latter 102·4°. The backs of the hands and the forearms are its favourite seats, but it may also cover the trunk, and even be seen on the face. In the museum of Guy's Hospital we have excellent models of this choleraic roseola made during the epidemic of 1854. It seldom comes out until a week or ten days after the commencement of the attack, and in one case recorded by Wilks it did not appear until the seventeenth day. It lasts two or three days, and may be followed by desquamation. It is more often observed in young patients than in old, and is particularly well marked in children. As a rule, the cases in which it occurs end in recovery.

Urine.—In the reaction of cholera the most important condition is that of the kidneys. We have seen that during collapse there is generally complete suppression of urine; if any is secreted it is apt to be, even at that period, albuminous. When reaction sets in, the suppression often continues for several hours, or even for two, three, or four days. According to Buhl, if it lasts up to the sixth day the case is hopeless. Sometimes, however, when the renal function has been re-established, the patient fails to void the urine in the bladder. The hypogastric region must therefore always be carefully examined, and, if necessary, a catheter must be passed. The instrument should be new and lubricated with carbolic oil, for cystitis and pyelitis have often been observed in cases fatal at an advanced stage. In a woman who died in Guy's Hospital during the epidemic of 1866 there was suppurative nephritis, which appeared to have arisen by extension from the bladder.

The urine which is first secreted during the reaction stage of cholera is described by Goodeve as high coloured, acid, and possessed of a strong animal smell. As to its specific gravity there are some discrepancies in the recorded observations. At the London Hospital in 1866 it was found to range from 1005 to 1017, the average being 1006. Wyss, however ('Arch. d. Heilk.,' 1868), found that the density of the first urine passed was from 1012 to 1033, the average being 1020. It was only at a later period, when the quantity was more abundant, that it fell to 1010, or even lower. The amount of urea and salts is much diminished, but albumen is generally present, and sometimes blood, and there are usually hyaline casts in immense numbers. Lebert says that the occurrence of albuminuria at this stage is constant, but his statement does not accord with the experience of English observers. At the London Hospital in 1866 albumen was found "in about half the cases examined." As a rule, the urine continues to be coagulable during from two to seven days. Until recently the renal affection attended with albuminuria in cholera was commonly regarded as parenchymatous nephritis, and as comparable with the acute renal affection that follows scarlet fever or diphtheria. But Bartels, in 'Ziemssen's Handbuch,' regards it as the result of the ischæmia which exists during the stage of collapse, and this view has since been upheld by Cohnheim.

In cases fatal before reaction has set in, the kidneys are found small, tough, and of a dark brown or slightly livid colour. At a later stage they are much enlarged—Dr Moxon speaks of them as weighing up to fifteen or sixteen ounces—soft, greyish white, or yellowish. The epithelium very early becomes cloudy, opaque, or fatty; it has lately been suggested that this change in it is analogous to the "coagulation-necrosis" which Litten has found to occur in the renal epithelium of rabbits after arrest of the arterial blood-current through the kidneys. The Malpighian tufts and their capsules

ent no morbid appearances in cases of cholera. Sometimes
ıd in the kidneys, being doubtless results of the ischæmia of
lapse. The renal affection of cholera appears never to form
nt of chronic Bright's disease. This fact accords with the
s theoretically to be distinguished from the various forms of
t is to be observed that dropsy and other renal symptoms
ıy the renal affection of diphtheria, the inflammatory nature
sputable.

id.—The reaction stage of cholera often presents a grave
ıich is known as "cholera-typhoid." This sets in about five
r the commencement of the attack. It is attended with
n, headache, giddiness, and stupor. The face is at first
rwards becomes pale. The temperature does not rise more
The tongue becomes dry. The pulse, which is sometimes
w, becomes weak and small. There is often a remarkable
nuscles, so that the patient strongly resists any attempt to
to separate his eyelids, or to straighten his elbow. Convul-
observed. Death by coma usually occurs in about forty-
sometimes the typhoid state continues for several days, and
in recovery.

rerichs, most writers regard this complication of cholera as
Urea has, in fact, been found in the blood in cases of
by several observers. Buhl estimated the quantity of it in
2 per cent. In some instances, too, there has been observed
l neck an efflorescence of urea in a crystalline form, excreted
nds. But Goodeve has observed that in some cases a con-
ttended with typhoid symptoms, occurs independently of
least while urine free from albumen is being secreted.
Vagner drew attention to the same fact, and attributed the
inflammation of the intestines or other internal organs.

.—There are certain minor forms of cholera, cases of which
uncommon, but of which the frequency in relation to that
isease cannot be positively stated, because they are often
eir course without being brought under medical observation.
ommonly called "choleraic diarrhœa." It is identical with
y diarrhœa" already described, except that it subsides after
not pass on into cholera. It often begins suddenly, after a
patient has eaten unwholesome food. There are three or
cuations of yellow fæcal matter daily, with some pain, and
ght cramps. A more severe form is sometimes termed by
s "cholerine;" it is attended with vomiting, with cramps,
disappearance of bile from the stools, and even with some
s of the limbs. Lebert says that he has seen it followed
yphoid symptoms.

and sequelæ.—The convalescence from cholera may be
certain complications, which require brief mention. One of
ion of the lower segment of the cornea, generally of each
less results from the part having been exposed and irritated
of collapse. It now, four or five days after reaction,
covered with a layer of opaque lymph; and ulceration
in it. Sometimes perforation takes place, but generally—
uld recover, which seldom happens—the eye is preserved.

In other cases, during the second or the third week the parotid gland swell or suppurate. This interferes with swallowing food, often with fatal result. Sometimes bronchitis, pneumonia, or pleurisy sets in; o diphtheritic inflammation of the fauces, or the bladder, or of the sexu organs, or gangrene of the scrotum or penis, or even of the nose. Bedsore too, are often a source of danger at this period of the disease.

Morbid anatomy.—This throws but little light on the pathology of cholera The bowels commonly contain more or less of the rice-water fluid; thi microscopically is found to contain an immense quantity of columnar epith lium, not only loose cells, but coherent masses of considerable size, formin complete casts of the villi. If there is no liquid, the cast-off epithelium forms a soft creamy pulp. At one time the opinion was held that sheddin of the intestinal epithelium was the fundamental lesion of cholera; but it now known to be of *post-mortem* occurrence. For not only are the cel absent from the evacuations passed during life, but (as Cohnheim observe denudation of the mucous membrane, if it took place before death, could n but be followed by severe inflammatory changes. All that is found is slightly injected, swollen, and œdematous condition of the intestinal coat with perhaps some ecchymosis of the valvulæ conniventes. The lymph follicles in the mucous membrane are also in many cases enlarged, an Lebert says that there may be slight ulceration of Peyer's glands. In case that occurred in Guy's Hospital in 1854 the lining of the rectum showe a patch which was œdematous and sloughing, with suppuration in the su mucous tissue. The peritoneal coat may be reddened and sticky, and 1866 Dr Moxon found in one case, in which the collapse had been unusual protracted, that a tenacious viscid material like spider's web lay between th coils of intestine, and could be drawn out into long filaments by separatin one coil from another. Other points on which he insists are the want fæcal odour and of the blackening produced by sulphuretted hydrogen, an the absence of gas in the intestine, so that the coils lie in a flabby compact ma in front of the spine. The mesenteric glands are sometimes slightly swollen

The blood which is found in the heart and in the great vessels is remarkab viscid and tarry; and whenever venæsection has been practised during lif the same peculiarities have been seen. This state of the blood, however, is n peculiar to cholera; for it was well marked in a patient who died in Guy's Ho pital in 1871 of acute ulceration of the intestine. It is no doubt a consequen of the drain of fluid from the body. Another result, which has been note by more than one observer, is the disappearance of dropsical effusion in th course of a few hours when the patient happens to be attacked by cholera.

In all probability the change in the blood is the cause of the formatio during life of ecchymoses, which at the autopsy are often found scattere over the outer surface of the heart, upon the pulmonary pleura, in the p mater, and elsewhere. All the tissues are peculiarly dry. Cohnheim r marks that the serous cavities in the dead body of a cholera patient have soapy feel, which is not likely to be forgotten by anyone who has ev put his hand in them; and the spleen, the lungs, the liver, and the oth organs are tough and leathery. The peculiarly shrunken appearance of th lungs is attributed by Dr Moxon, no doubt correctly, to the dry, empty sta of the bronchi, which allows the elasticity of the organs to drive the air o of them more completely than usual after the chest is opened. On th other hand, when death has occurred during reaction, the smaller tubes a

f pus, and parts of the lungs may be œdematous, or even
cho-pneumonia.

ne whole course and natural history of an epidemic of
into close relation with the specific contagious fevers of
hus, smallpox, and scarlet fever are types; and we shall see
s now ascertained to be infectious, though not in the direct
which is observed in those maladies; its mode of spreading
of enteric fever, and it may be not improperly regarded
alady as a "miasmatic-contagious" disease (p. 13). More-
han probable that the *contagium vivum* has already been
form of a vibrio, to be presently described.

ia, which we have hitherto found to be a constant attendant
of a swarm of microphytes, is either absent or strangely
it appears in a slight, inconstant, and equivocal form.
ng prevents this virus from reaching the thermotaxic
ous system; or possibly some thermolytic process prevents
g the temperature from being manifest. Or it may be that
nicrobes is chiefly or entirely local, and that the intestinal
lf the cause of the thickened blood, the low temperature,
nptoms.

nsider cholera as a local inflammation, like erysipelas or
from the absence of pyrexia, the characters of the rice-water
e those of any known inflammatory exudation, in any part
we cannot possibly suppose it to be of that nature. An
ever, originally made by Moreau, suggested the clue to
e. Physiologists had previously learnt from Thiry how to
of the intestine, so as to obtain from it an unmixed
in small quantity. Next Moreau found that when an
testine has all the nerves in its mesentery cut through, it
thin a few hours at latest, with an abundant secretion of a
wish fluid, which is of very low specific gravity, which
s mucous flocculi, but in which there is only a very small
en, while the chief salts in it are those of soda.* There
ytic over-secretion of the succus entericus and, according
sesses the property of converting starch into sugar. But
at the rice-water liquid of cholera likewise contains a fer-
property, beside corresponding closely with succus ente-
respects. The inference, therefore, seems justifiable that
olera is nothing else than the secretion of the intestinal
ly increased in amount. Whether its formation should be
n of paralysis of the splanchnic nerves is left by Cohnheim

In proof that the muscular coats of the bowel are not
in cholera he cites the fact that *post-mortem* invaginations
which occurred after or shortly before death. But he in-
alled "cholera sicca," in which rice-water liquid is found in
er death, though none had been voided during life, owes its
n early exhaustion of the excitability of the intestinal walls;

* ts have since been confirmed and extended. See the Reports of the
l by the British Association, published in the 'Transactions' for 1874
o papers by Dr M. Hay in the 'Journal of Anatomy and Physiology'
vol. xvii, p. 441), and by Dr Brunton and the present writer in the
November, 1884, *et seq.*

and he refers to the same cause the frequent cessation of vomiting a purging as collapse becomes developed.

The above view with regard to the nature of the rice-water liqui strongly supports the doctrine held on other grounds by many observe that the primary action of the cholera virus is upon the solar plexus rath than upon the bowel itself. Cohnheim, indeed, agrees with those who thi that the phenomena of collapse are adequately explained by ascribing the to the drain of fluid from the body, and to the viscid inspissated conditi of the blood which we have seen to result from it. This opinion was oppos several years ago by Dr George Johnson, one of whose arguments was th no relation could be traced between the degree of severity of the collapse in given case and the amount of fluid discharged from the stomach and fro the intestine. A stronger argument has since been advanced by M Sedgwick, namely, that cholera collapse closely resembles that which follo cases of perforation of the stomach, of intestinal obstruction, and of oth abdominal diseases in which no purging occurs. At Guy's Hospital, durin one of the London epidemics, a man actually died of internal strangulatio of the bowel who was supposed during life to be suffering from the so-calle "cholera sicca." It must therefore be supposed that collapse is the result some disturbance of the abdominal sympathetic. This disturbance, how ever, is by no means identical with that which causes syncope. Chole patients have no sensation of faintness; and even when they have no pul at the wrist they may be able to sit up or to walk about without sudde failure of the heart's action. On the other hand, the collapse of cholera essentially different from rigor. The suppression of urine which occu in collapse is a part of the general state, since it is observed likewi in cases of acute intestinal obstruction, of perforating ulcer of the stomach and of arsenical poisoning. Thus few pathologists agree with Dr Johnso in endeavouring to trace all the phenomena of cholera collapse to obstru tion of the flow of blood through the pulmonary capillaries. An although the high authority of Parkes can be cited in proof of the fa that the lungs weigh much less than usual in cases of cholera, this may b sufficiently accounted for by the diminished volume of the blood generally since the lungs contain so little solid material that their weight, so long a they retain their spongy character, must be mainly that of the fluid in thei vessels or in their interstices. During the epidemic of 1866 we entirel failed to observe that the branches of the pulmonary artery and the righ side of the heart were distended or gorged with blood when death ha occurred even at an early stage. It is true that cholera is attended with lividity which is wanting when collapse is due to other causes. But th altered constitution of the blood may be fairly supposed to interfere with it due aëration, and the amount of carbonic acid exhaled from the lungs ha been shown to be much reduced.

Absorption is retarded, although not entirely annulled. Magendie i said to have found that when camphor was injected into the rectum, fiv minutes elapsed before its odour could be detected in the breath, instead o only one minute, as in health; and Lebert states that atropine fails t dilate the pupils when administered by the mouth, though not when injecte into the blood. It is, however, a very curious circumstance that in women who are suckling children the secretion of milk continues during collapse even to an extent which is troublesome. The menstrual flow also goes on unchecked; it may actually set in, before reaction is established, if the prope

ound for its appearance. A sanguineous muco-purulent he vagina also frequently appears, independently of the n the disease seizes upon a pregnant woman, the fœtus die; if the period of gestation is early, abortion takes e mother often dies undelivered. In this case it appears rform the operation of Cæsarean section, even immediately The mental and bodily vigour are often maintained through- an attack of cholera in such a way as to show that the mains active in the brain and in the muscles.

ch have been already stated as to the course of the external emperature respectively during the reactive stage, support er cholera should be reckoned as a febrile disease. The be what its name implies, and analogous to the hot swollen l in a part when its vessels are allowed to fill with blood, kept empty, as in experiments on the ears of rabbits.

may now be regarded as certain that the diffusion of a over Europe is the result of human intercourse.*

3 the first epidemic entered Russia by Astrachan, and l in a north-westerly direction, it was ascribed to some pheric or telluric agent. But its progress was far too slow such a view to be even hypothetically tenable. Prussia holera in 1831, in October of that year it passed from derland, and entered London in January, 1832. Shortly vaded France *viâ* Calais, and it also spread across the a and the United States. In 1833 it appeared in Portugal, erly direction through South Europe, and became for the 837. One can hardly doubt that its diffusion from India ner than to the East depended upon the circumstance on from town to town and from country to country e free in the one direction than in the other. The which had begun in India in 1840, extended to Europe like the first, reaching London direct from Hamburg in ing to prevail in England during the following summer. a occurred among us in 1866, differed from the others in rope from Alexandria; it had been carried from India to e to Egypt, partly by coast-trading vessels, but also by grims to and from Mecca. A detailed account of this Netten Radcliffe, appeared in a supplement to Mr Simon's ivy Council' for 1874.

pean epidemic started in June, 1884, at the port of Toulon, a month later at the port of Genoa. The only cases seen n sailors from the Mediterranean at Cardiff and Bristol.

st remarkable circumstances with regard to cholera is that read to almost every part of the world, and has sometimes idely different thermometric and other conditions, it seems stablishing itself permanently in no country except India, a particular region.

ever, cholera seldom passes directly from a sick person to

* interesting accounts of the spread of the several historical epidemics amara in his 'Treatise on Asiatic Cholera,' 1870 and 1876. Also Dr the Cholera in India from 1862 to 1881,' published in 1885.

those who nurse or visit him. A few instances have indeed been record which look as though direct infection did sometimes occur. Thus, in paper in the 'Edinburgh Medical Journal' for 1838, the late Sir Jam Simpson related how certain mendicants brought cholera with them Bathgate on May 27th, 1832, and how one of the nurses who attend upon them was attacked and died on the 30th. He also cites the case of woman who, having caught the disease at some ironworks where it rag and being afraid of being sent to the hospital, went on May 12th to a cotta where her mother lived, four or five miles off: on the 14th the mother f ill, and died in a few hours. Dr John Snow relates that a man who had be working at Chelsea died at Streatham of a bowel complaint which last only a day or two: at that time no other deaths from cholera had be registered within two or three miles of Streatham, but the man's moth who attended him, was taken ill on the very next day and quickly s cumbed to the disease. But instances of this kind are very rare, and, a rule, persons who come into contact with patients are scarcely, if at a more likely than others to fall victims to cholera.

The explanation is believed to be that the contagion of cholera escap from the body in the rice-water evacuations, but that it is not then in active state, and becomes so only after an interval of from two to four five days; in other words, the evacuations are supposed to be infective on at a certain stage in their decomposition, and not when they are fres The evidence in support of this view consists mainly of the results of exp riments made by Thiersch in 1854, and repeated by Burdon Sanderson 1866. These observers took liquids passed by patients during life, or tak from the intestines after death, and left them exposed to the air in gl vessels. Every day pieces of filter-paper were carefully soaked, one wi each liquid, and dried. The paper was then given to mice, a square in to every animal. The consequence was that a considerable number of t mice died with an extremely low temperature and with disturbance of t functions of the intestinal canal, their excrement being soft and altered colour. Of the mice employed by Sanderson, 11 per cent. suffered wh the rice-water liquid had been one day old, 36 per cent. when it had be two days old, 100 per cent. when three days old, 71 per cent. when fo days old, and 40 per cent. when five days old; by the sixth day it becam innocuous again. The morbid appearances found in the mice after dea appeared to be consistent with the view that they were affected with chole and Mr Simon afterwards expressed the opinion that the value of t experiments in explanation of the facts of human infection was conclusiv No equally satisfactory results have hitherto been obtained with other kin of animals; but some instances have been recorded of dogs being attacke by a fatal disease resembling cholera after having devoured matters vomite by cholera patients, and in 1853 Dr Lindsay set up a similar disorder i dogs by confining them in a small room, the floor of which was strewed wit the excreta of such patients.

It may be thought that if a living organism, possessing specific propertie is present in however immature a form in rice-water discharges, it ough to have been long ago discovered. But the alvine evacuations, even i health, contain various microphytes in large numbers, and it is therefore b no means surprising that the search for special cholera microzymes was lon unsuccessful.* Dr William Budd, indeed, published figures of what he sup

* In the 'Journal of Microscopical Science' for 1881 Dr Cunningham states th

cholera fungus as far back as 1849; and many observers similar discoveries, but their statements all lacked evidence.

bacillus.—When the epidemic of cholera which prevailed in in 1884–85 first made its appearance in Egypt, the German t thither Dr Robert Koch, who had lately discovered the ercle. As the result of his examination of the disease at believed that he had ascertained its contagium vivum to be aped organism, which he called a bacillus. He was after- ombay, where cholera was prevalent, and there announced hyte characteristic of cholera was of a curved shape, and this character "the comma bacillus of cholera"—an un- for it is in form not a bacillus but a vibrio. Subsequent only confirmed Professor Koch in this belief, which is competent observers.

ever, opposed to the opinions of the majority of Indian t with much adverse criticism. The late Dr Timothy Lewis, f the *Filaria sanguinis*, stated that the so-called bacillus is met with in the mouth of healthy persons, and it was ases of diarrhœa by Prior and Finkler in Germany. The ment sent Dr Klein and Dr Heneage Gibbs to India to subject, and their report was adverse to Dr Koch's theory. yal Society and Medical Research Association united to send mbridge, to investigate cholera then prevalent in Spain, and year the same Association deputed Dr Sherrington (who l Dr Roy in Spain) to clear up doubtful points by researches cholera was still partially epidemic in the summer of 1886. l also been made by French physicians at Toulon.

t of these difficult and prolonged investigations it must be Koch's conclusions were premature. This was the verdict n Sanderson and also by a Commission appointed by the ne and report on the evidence.*

the criteria for determining the causal relation of a micro- ntagious disease stated on p. 13, we ask the following *Is the disease itself definite and capable of accurate diagnosis?* the marked and striking character of the symptoms and a given in the preceding pages, there is no doubt that slight ur during an epidemic might at other times be put down as rrhœa; that the diagnosis between so-called cholerine or cholera (both of them bad names), and true Asiatic malignant difficult or perhaps impossible; and that isolated cases occur ch, in the judgment of cautious and experienced men, are from genuine cholera and yet do not spread the disease. *ortem* appearances are not always uniform or decisive. Dr nd that a large number of the supposed cases of cholera in y diarrhœa, or enteric fever, and Dr Roy had observed the Even granting, what is no doubt true, that if sufficient care ecting well-marked cases, no appreciable risk of mistaken

ng either to *Cercomonas* or to *Trichomonas*, are found in excreta of om cholera in unusual numbers and in a peculiarly active state, but he r to some extent under other conditions of disease, and even in health. be to be very abundant in certain cases of cholera; bacteria, he says, e part even of the normal fæces, at least in India.

given in full in the 'Quart. Journ. of Micr. Sci.,' vol. xxvi, p. 303.

diagnosis is present in the case of men, the difficulty becomes very mu greater when we seek to determine whether the clinical features or t morbid anatomy observed in mice and other animals entitle us to regard disease induced in them to be the true cholera of man.

(2) *Is the microphyte clearly distinguishable from others?* This we m probably allow as the result of many patient and independent investigatio The organism is not properly called a bacillus, it is a vibrio, or rather fragment of a vibrio, and it is indistinguishable in size, shape, or reaction staining agents from Lewis's microphyte. But when cultivated its coloni show distinctive characters, so that mistakes may be avoided by adequa experience and pains.

(3) *Does the microphyte only occur in cases of cholera?* The answer this question in involved in the preceding. Very similar or morphological identical comma bacteria occur in the alimentary canal both in health a disease, but the genuine organism, tested by its growth and development well as its form and size, has not yet been proved to exist except in t intestines or the dejecta of cholera patients.

(4) *Is its occurrence in cases of the disease constant?* This is not y proved, but in the great majority of cases there are numerous "comm bacilli" to be found in the intestinal contents, and occasionally their abun ance approaches what Koch called a "pure cultivation" in the small inte tines. But they do not occur as the spirillum of Relapsing Fever, and (as shall afterwards see) the bacillus of Anthrax, in the blood or tissues. T commas are found in the dejecta and in the rice-water contents of t bowels, and also in the detached epithelium found after death loose in t ileum, but they have never been discovered deeper than the epithelium, at furthest the *mucosa* (corium), of the intestinal walls.

(5) *When separated by cultivation from other organisms, does a new bro of the microphyte reproduce the original disease when introduced into the circul tion of an animal?* This, the last and crucial proof of the causal relati of the plants to the disease, is wanting in the case of cholera. Koch's i oculations are far from convincing, and the attempts of others to reprodu cholera by introduction of the commas into the stomach or intestine blood of various animals have not succeeded, even when the acid digesti of the stomach has been evaded, or when the influence of the bile has be excluded by ligature of the bile-duct. Whether man is the only anim capable of contracting cholera, whether the right animal has not been fou (for the earlier experiments of Thiersch and Sanderson above-mentione are inconclusive), or whether the contagium of cholera is another microphy than the comma—these are questions not yet decided, but at present t evidence that the true contagium has been discovered is defective.*

Nevertheless, the comma vibrio is so constantly present in cases Asiatic cholera that, as Dr Klein admits, its discovery in the fæces duri life or in the intestines after death is a proof of the nature of the disease.

Modes of transference of the contagium.—Passing on now to consider how t contagious principle of cholera gains access to the human body, we find t best ascertained fact to be its frequent entrance by means of drinking-wat

* Klein observed some very minute rod-shaped bacteria to be more constant in the int tines than the comma vibrios, and thinks they may be more closely related to the dise Emmerich found straight bacilli in the blood of cholera cases.

It may be that bacteria in the intestines cause diarrhœa with catarrh of the muco and that in this nidus the true contagium of cholera finds its suitable conditions for d velopment.

hn Snow deserves special commemoration, not only as having
o uphold this view with regard to the ætiology of the disease,
aving devoted infinite labour and pains to establish it. He
nces occurring as far back as 1849, in which local outbreaks
ore or less conclusively to the contamination of surface-wells
e such occurred in Thomas Street, Horslydown, another in
, Wandsworth, a third at Salford, a fourth at Ilford. The fol-
was very striking. A gentleman who lived at Bath was the
houses at Locksbrook, near that town. Cholera, which did not
, appeared at Locksbrook and became very fatal. The people
the water of the well attached to their houses, drainage
ools having entered it. The owner went to the place, said he
thing wrong with the water, was asked to taste it, and drank
This was on a Wednesday; he returned home, was taken ill
nd died on the Saturday.

not until 1854 that the evidence of the communication of
king-water became irrefragable. Then occurred the celebrated
d around Broad Street, Golden Square, which is said to have
n days more than five hundred persons living within a radius
This was traced by Dr Snow to the water of a surface-well,
tuated at the corner between Cambridge Street and Broad
t sixty-eight of the first eighty-three deaths occurred in persons
lly known to have drunk the water in question; and it may
at many took it without being aware that they did so, mixed
ublic-houses. A lady at Hampstead, who was in the habit of
day for the Broad Street water, was attacked by cholera and
ece, too, being on a visit to her, drank some of the water,
ards to her own house at Islington, and died there. On the
rcely any of the inmates of the workhouse in Poland Street,
s a separate well, were affected; and the disease also spared
yed at a Brewery in Broad Street, close by.

as on a still larger scale were made, in 1854, in the south of
district containing a population of at least 300,000 persons,
ith water partly by the Lambeth Water Company, conveyed
es at Ditton, partly by the Southwark and Vauxhall Company,
as from the same river at Battersea. What rendered these
eculiarly conclusive was the fact that over a large area the
two companies ran side by side, each feeding some houses and
rding to the arbitrary preference of owners or occupiers at
when there had been an active competition for custom. Dr
detail, throughout the area in question, from street to street
to house, and he found that, during the last four weeks of
ut of three hundred and thirty-four fatal cases of cholera,
hundred and eighty-six in houses supplied by the Southwark
Company, twenty-two among persons who obtained water
ping a pail into the Thames, but only fourteen in houses
ly from the Lambeth Company. It is to be noted, however,
r of houses supplied by the Southwark and Vauxhall Company
n that of the houses supplied by the Lambeth Company, in
f forty to twenty-six. As the epidemic continued, the differ-
lera death-rate between the customers of the two companies
hough it still remained very striking. Bethlehem Hospital,

the Queen's Bench Prison, and other institutions on the south side of
river which had deep wells, scarcely suffered at all from the disease.

In 1865 and in 1866 further evidence was collected bearing in the sa
direction. In 1866 the parts of the metropolis mainly affected were
eastern districts, and Mr Netten Radcliffe, who investigated the matter
the Privy Council, found that there was a great preponderance of ca
among persons whose water, supplied by the East London Water Compa
had passed through the reservoirs at Old Ford, whereas comparatively
cases occurred among those who received the water of the same Compa
pumped directly from the filtering beds at Lea Bridge into the mains. T
difference was far from being as conspicuous as in South London dur
1854, but one must bear in mind that among the poorer classes very m
eat and drink and pass their days at a distance from the houses in wh
they are said to live. Moreover, at an early period of the epide
a notice was issued, warning people not to drink any water which
not previously been boiled, and it is worthy of notice that from the v
week in which this notice appeared the epidemic began to decline. A pe
of particular interest is that, assuming the reservoirs at Old Ford to h
been in some way concerned in causing the disease, one can account for
entrance of the cholera poison into their water; for part of it was someti
drawn from two reservoirs which communicated freely with the river Le
soakage, and this part of the river was, in fact, a canal with locks,
received so large a quantity of sewage, that it was little better than a cesp
Now, shortly before the epidemic in East London began, a man and his w
living in Priory Street, Bromley, near the banks of the Lea, had died
cholera, and their evacuations had entered the river about 600 yards be
one of the open reservoirs.

In the previous year, 1865, there had occurred in an Essex villa
Theydon Bois, a most remarkable local outbreak of cholera, which illustra
in a striking manner how insidious its propagation may be. Between J
and November the disease seems to have been several times introduced i
Southampton by persons who arrived from the Mediterranean ports wh
it was then prevailing. Whether it was carried from this town dur
August or September to Weymouth or Portland or Dorchester, or whethe
reached either of these places in some other way, was never ascertained.
so much is certain; that a gentleman and his wife, visiting these places (but
Southampton) in September, contracted diarrhœa at one or other of them,
this, in the case of the lady, developed into fatal cholera after her return
her home at Theydon Bois on September 25th. On the 30th, while
was still collapsed, one of her daughters was attacked and died in a few ho
The same night a serving-lad in the house was seized and barely escaped w
his life. Altogether, in a fortnight, eleven persons within a narrow ci
father, mother, grandmother, two daughters, son, doctor, foot-boy, m
labourer, and countrywoman, fell ill, and only three of them recove
Now, the drinking-water of the house all came from a well beneath
scullery, and into that well there was habitual soakage from the water-cl

Soil-water theory.—The "soil-water" or "ground-water theory" of Pet
kofer is that cholera never prevails epidemically where the soil is imperme
to water, or where the level of the soil-water is not liable to fluctuations.
brings forward several instances in which districts seated upon hard r
escaped, while adjacent towns built upon alluvium suffered. Some appa
exceptions to the rule he accounts for, more or less satisfactorily. T

ll known, shows no immunity, but, in visiting it, he found
ally lies on a slope of red earth, containing more than
s. So, again, Malta, where the disease has prevailed
of solid rock; but this, a sandstone, is so soft and so
er that the Government Comptroller told Professor Petten-
like a sponge, saturated with all kinds of filth. On the
s has, during each of the European epidemics, remained
rom cholera, when both Paris and Marseilles were ravaged
wenty thousand persons are said to have flocked thither
or safety. But the greater part of Lyons lies on a river
on the ground-water theory one would expect its inha-
rely attacked.*

Pettenkofer, the condition of soil under which cholera is
il is that which occurs when the level of the ground-water
l after being high. This might perhaps cause noxious
hed into wells which would at other times be free from
more likely cause for the development of an epidemic
nstances is the desiccation of contagious microzymes in the
e soil, and their subsequent diffusion in the air.†

conveyance.—Whether the virus of cholera is ever conveyed
btful; but apart from Pettenkofer's theory, the following
Dr Simpson, of York, and cited by Dr Snow, seems to
n may in certain cases take place apart from drinking-
ltural labourer was attacked at Monkton Moor on December
me when the disease was not known to be prevailing within

n which Pettenkofer gave is that in this town, unlike most others,
-water is diminished by that of the rivers which flow through it, the
nd the Saone being in fact so porous that their streams may almost be
bterraneously beneath Lyons. In other words, the supposition is that
either because there is too much surface-water, or because its height
any variation. But, ingenious as these explanations are, one cannot help
might not be possible, by similar reasoning, to explain away the very
ettenkofer relies.

owever, considers that a complicated theory is needed to explain his
that when human intercourse conveys cholera from one place to another,
ff by a patient, or carried by an individual not himself susceptible, is,
gion, but what may be termed a "cholera-germ," which is capable of
ion when it meets with a certain "material substratum." An analogy,
will make his notion readily intelligible. It is, he says, conceivable
gus were absent from certain countries, the inhabitants might prepare
s, or apples, or malt, and drink them without any liability to intoxica-
e were to come from a country where the yeast-fungus was found, and
with him upon his clothes or in any other way, the hitherto harmless
ly produce an "epidemic of drunkenness." Yet the cause of the
be the yeast-fungus, but the alcohol of the fermentation set up by it.
nes that the "cholera-germ," x, acting on the soil under certain con-
"cholera poison," z.

staunch supporter of Pettenkofer's observations, has more recently
rms a "diblastic theory," according to which the union of x and y is
e within the human body. He imagines that the soil gives off certain
ust be present in the body of everyone who is to afford favourable con-
pment of another set of microzymes derived from a pre-existing case
n fact, to reduce the "soil"-element in the ætiology of cholera to the
ing" cause. Nägeli appeals to cultivation experiments as affording
which the growth of one kind of fungus in a medium induces changes
ord pabulum to another kind of fungus. Or, as he points out, it is
agious microzymes of cholera which enter the body are commonly too
rcome the resistance of the living structures, unless the latter has first
e previous operation of soil-microzymes.

thirty miles. His wife and some other persons who visited him were sei on the following day. Presently it was found that his sister had died cholera a fortnight previously at Leeds, and that her wearing apparel h been sent to Monkton in a box which had been opened by him the even before he fell ill. During the illness his mother, who lived in a healt village five miles off, came to attend him, washed his linen, and after t days set off to return home, but was attacked on the road and had to conveyed to her cottage. Not only did she die, but her husband and daughter likewise.

In 1866 Parkes attributed an outbreak of cholera in Southampton mai to an atmospheric effluvium disengaged from sewage, which was constan being pumped up by a steam engine, and sent churning down an o channel for some eight or nine feet.

Altitude and climate.—There are still to be mentioned certain conditi which seem undoubtedly to influence the diffusion of cholera, however direct their operation may be. Dr Farr laid down a "law of altitude," least for London, showing that the incidence of the disease upon the popu tion varied inversely as the height above the river Thames. This, howe is but an expression of the fact that on an average persons living at a level are affected by the causes of cholera in larger numbers than pers living at a high level. Sometimes the case is reversed; thus Lebert s that throughout the epidemic at Zürich in 1855 the upper parts of the to suffered more than the rest. The circumstance that in Europe cholera never been known to prevail at an elevation of more than 600 or 800 mè above the sea may be set down to the absence of the conditions required its development; in other quarters of the world it has raged at altitudes 2000 to 2500 mètres. In temperate climates the summer and the autu are the seasons most favourable to it. It often dies out as winter approach to reappear in the following year; but in Russia, in 1830, it withst extreme degrees of cold.* Cholera appears never to have invaded Austr the Cape Colony, or the South Seas.

Diagnosis.—To recognise cholera in its well-marked forms is sel difficult. Almost the only morbid state which can be mistaken for it is produced by the poisonous administration of arsenic. Lebert relates Louis actually committed this blunder in regard to the Duke of Chois who killed himself with arsenic the day after his arrest for the murde his duchess. At that time, in the summer of 1847, the disease was prevailing in Paris. There is therefore good reason for bearing in mind a crafty person might take advantage of an epidemic to give poison with little risk of being suspected.

To distinguish the slighter effects of the virus of cholera from ordi diarrhœa is often quite impossible. Under the name *cholera nostras*, or *ch Europœa*, writers describe an affection of which the symptoms are ident with those of the specific disease (called by them *cholera Asiatica*), but wh differs from it in being scarcely ever fatal and in generally occur sporadically. A typical instance occurred at Guy's Hospital in 1865,

* The remarkable distribution of cholera in its visitation of England 1848–6 spread, ingravescence, and decline in the several towns it attacked, are described, and bearing on its ætiology as then appreciated impartially discussed by the late Dr Baly Dr (now Sir William) Gull in their 'Reports on Epidemic Cholera' to the Royal Co of Physicians, 1854.

corded by Wilks in the 'Medical Times and Gazette' for acksmith's hammerer, aged thirty-three, was at work as on May 25th, when he was suddenly seized with profuse ging. He was admitted collapsed, with cold breath; he had ks and in the legs; the evacuations and the matters vomited ce of rice-water, consisting of an alkaline liquid, with flocculi e recovered in about twenty-four hours. Dr Wilks added he saw one or two such cases, but seldom so early in the o be observed however, that at the very time when this l, an epidemic of cholera was approaching England; and, e as it may seem that an isolated case should spring up in eeks before the commencement of the local epidemics at at Theydon Bois, the possibility of such an occurrence l. At any rate, no case of so-called "English cholera" llapse, rice-water stools, and cramps) seems to have been y's Hospital since 1870; and it is certain that when the se in 1831 Sir Thomas Watson, the elder Dr Babington, hysicians of experience, declared that they had never "met mplaint before." However, it is undesirable to call any xcept that which comes as an epidemic from Asia, and ht never to call attacks of diarrhœa, in infants or even aic" merely because the symptoms are severe.

his is less favourable in children and in old people suffering in adults or in middle-aged persons. The mortality, which ulation generally averages about 50 per cent. or a little ng the very young and the aged to 70 or 80 per cent., ng adults it may fall to about 40 per cent.

n 1866, of 130 cases at the Meath Hospital 71 were ir Patrick Dunn's Hospital, 85; and of 197 at the Mater pital, 106.

f 21,519 patients in Italy, 11,563 died. In the following 338,685 cases of cholera were officially reported in Spain, 620 were fatal.

more than usually dangerous when it affects drunkards or eady sick or weakly. In an individual case the degree of greater prognostic importance than the amount of purging. mic diseases, the earlier cases are far more often fatal than when the pestilence is subsiding. At Calcutta, in 1850, lity was 47 per cent., but among the earliest cases it was among the latest only 25.

afforded by an attack of cholera against a subsequent one he slightest. Cases are not infrequent of persons who have epidemics, and some have died from the second attack. e parts of the East Indies where cholera is endemic, one ffords no immunity from a second, but seems scarcely to ty. All that can be said is that cholera does not predispose as erysipelas undoubtedly does.

he *prophylactic measures* which should be adopted when a ned or actually attacked with an epidemic of cholera were ed by Mr Simon and his colleagues in 1866, and will be

found detailed in his ninth 'Report.' They are generally such as might anticipated from what is known of the ætiology of the disease. Mr Sim attached great importance to the avoidance of all such food as is apt set up diarrhœa—half-fermented beer or wine, meat or game no longer fr or not completely cooked, stale fish or shell-fish, vegetables or fruit l gathered or badly kept, and the like. He deemed it unwise to take pu tive medicines, except of the mildest kind; and, in accordance with alm every other observer, he insisted on the necessity at such seasons of ch ing looseness of the bowels, however painless and trivial. Dr Bristowe al maintains that the diarrhœa which so commonly prevails when choler epidemic is neither more nor less likely to pass on into that disease, whet it is left alone or encouraged by purgatives; nor will he allow that, if i really premonitory of cholera, it can be arrested by any medicine whate Dr George Johnson's theory, that the purging of cholera is an effor nature to get rid of the *materies morbi*, and should therefore not be chec but even encouraged by administering mild laxatives like castor oil, does appear to be accepted by any other physician.

Most writers recommend that the initial diarrhœa should be treated v opium in considerable doses, and with astringents, such as acetate of l nitrate of silver, catechu, or chalk. In India Dr Goodeve says that i usual to give from one to five grains of calomel with the first two-g doses of opium, and to check the diarrhœa as much as possible.

When *collapse* has developed itself the administration of opium astringents is believed to be useless, or even worse than useless. state of the circulation is now such that absorption is nearly arrested; drugs introduced into the stomach in successive doses may, if not vomi accumulate there so as to produce ill effects at a later stage, should reac happily occur. For the same reason alcohol must be used with g caution, if at all. Dr Goodeve says that its admissibility depends upo effect on the pulse. If a dose of weak brandy-and-water causes the puls revive ever so little, there is no harm in continuing to give the stimulan small quantity. In any case ether or ammonia may be used, prop diluted, unless sickness is produced. But Dr Goodeve finishes his rem upon this point by saying that many cases in which there is extreme coll come round by themselves, as well as, if not better than, with stimuli Ice-cold water may be freely allowed to patients with cholera, not indee such large quantities as their morbid thirst might lead them to swal but by tablespoonfuls or wineglassfuls at a time. Lebert would also effervescing draughts made with carbonate of soda and with lemon ju and at the London Hospital in 1866 a "saline lemonade" was emplo with apparent advantage.

There seems to be no objection to administering a dose of morp subcutaneously if the cramps are very severe. Or an occasional whi chloroform may be given by inhalation. It may also give relief to rub p chloroform or turpentine into the painful parts. The burning sensa at the epigastrium may be alleviated by applying a mustard plaster.

The limbs should be kept wrapped in warm flannels, and hot bot should be placed in the bed, but not in contact with the patient's s At the London Hospital, in 1866, baths at a temperature of from 98 104° were employed in about one hundred and thirty of the worst ca and with most marked effects. For a few seconds after immersion th was commonly difficulty of breathing, and sometimes an unpleasant se

But in less than a minute a favourable action generally ; the pulse returned, or (if it had been perceptible before) d quicker; the patient grew less distressed, ceased to moan pain, and sometimes fell into a quiet slumber. In many ppeared to be the direct consequence of the bath ; but in ymptoms returned unaltered.

ce at the London Hospital seems also to have been not un- practice of injecting a warm saline solution into a vein, e in an apparently hopeless condition from cholera collapse; ses so treated by Mr Little there were four recoveries. , however, consider this treatment incapable of producing ood result. The immediate effect of an injection is admitted be often marvellous; a person speechless, and almost dead, sciousness, sit up, and talk; and the pulse may become ll. But in most cases the improvement has been only symptoms soon return and end fatally even though the be repeated. Still, as Sir Thomas Watson observes, even y amendment may sometimes be of great importance, mple, a will to be executed. The fluid used by Mr Little grains of chloride of sodium, six grains of chloride of potas- s of phosphate of soda, twenty grains of carbonate of soda, s of pure alcohol, to twenty ounces of distilled water. Eight oduced at a time, the temperature being about 110°, and thirty minutes were occupied in the operation. In three on was repeated, and one of them ended successfully.

n begins, the management of the case of cholera continues care and caution. A little beef-tea or chicken broth may n small quantities at intervals, or some light farinaceous el or arrowroot. Should vomiting continue, it may often dose of opium, or by a blister applied to the epigastrium. it is necessary, for a time, to have recourse to nutrient ake up for the loss of salts from the blood, carbonate of e of sodium should be given in moderate quantities. If mes dry and brown, a powder containing two grains of m cretâ, half a grain of ipecacuanha and two and a half s powder was found useful at the London Hospital. When urine continues during reaction, the patient should be drink freely; he may take a drachm of liquor ammoniæ e or ten drops of tincture of digitalis, every two or three ard plasters or cupping-glasses may be applied to the loins.

ERYSIPELAS*

Ἐρυσίπελας ἔξωθεν μεν εἴσω τρίπεσθαι οὐκ ἀγαθόν, ἔσωθεν ἔξω ἀγαθόν.
HIPPOCRATES.

Definition of the disease—Its contagion—A specific febrile exanthem—Microb
Relation of the phlegmonous form to facial idiopathic erysipelas—Incuba
and onset—The eruption—Its histology—The symptoms and course—Recur
erysipelas—Complications—Sequelæ—Diagnosis—Prognosis—Treatment

HITHERTO all the diseases discussed have agreed in the essen features of the group called Specific Fevers, and each has been easily defi and separated from any other malady. But now we come to less ea limited categories.

Erysipelas and Diphtheria have both been the subjects of much con versy as to the extent to which the name should apply, and as to "specific" character of many cases. The difficulty arises from the pro nence of the local lesion and its likeness to non-specific inflammation.

Erysipelas is frequently mentioned by Hippocrates as a form of ac inflammation of the skin, and the term has never since been lost in med literature. There have, however, been great differences of opinion a what affections should be included under this name, and particularly to the relation of certain local inflammations to the specific fever.

Some physicians, especially in France, have been anxious to separat "medical erysipelas" of the face and head from the cases seen in surg practice, of erysipelas attacking the limbs and the body after injuri and in England this distinction received Sir Thomas Watson's suppo Another question, at one time much discussed, was the relation betw erysipelas and erythema. The latter term is applied to various forms superficial dermatitis, which will be described in the last section of t work; but there can be no doubt that it has sometimes been applied light cases of erysipelas.

Many English surgeons regard as "erysipelatous" almost every form spreading inflammation of connective tissue, even including diffuse suppu tion of the post-peritoneal structures after operations on the rectum. T view is strongly objected to by Volkmann and other German writ They will not even allow that we are right in describing as "phlegmon erysipelas" cases of suppuration or gangrenous inflammation, attended w intense redness of the skin, but also affecting the subcutaneous and int muscular textures.

Lastly, the epithet "erysipelatous" has been applied by some obstet physicians to the septic peritonitis of puerperal women.

Pathology.—All these questions must ultimately be settled by ætiologi considerations. Erysipelas is a contagious disease. Of its propagation

* *Synonyms.*—Febris erysipelatosa, Ignis sacer, Rosa, the Rose, St Anthony's fire— Erysipèle.—*Germ.* Rothlauf. *Ἐρυσίπελας* is a classical Greek term,

l examples were recorded many years ago by Dr Wells, of spital, the celebrated author of the 'Essay on Dew.' For a nental observers disputed the possibility of such an occur nann cites more than a dozen cases in proof, and it is now tted. Perhaps the most striking series of recorded cases fore the Paris Academy in 1864 by Dr Blin. One of the Lariboisière Hospital had under his care two patients rysipelas, when he was himself seized with it. A medical ise visited him and fell ill after returning to that place, case of the disease then existed. That gentleman's servant nd also a relative who came to see him, and who lived in od. The latter gave erysipelas to his wife, and three other family who were repeatedly in contact with them ess suffered in their turn. From this family the disease sters of mercy, and they carried it to their home and gave nan who attended them. Lastly it passed from him to his

not only a contagious, but also a *specific* disease, in the always dependent upon the entrance into the body of a om without. It is a great mistake to suppose that the æmia, necessarily becomes prevalent wherever surgical owded together under unfavourable conditions. In the the Austro-Prussian war of 1866, and in the Franco- 1870 repeated examples presented themselves of hospitals possible to keep healthy, and yet erysipelas failed to appear. nd, there have been many instances in which it has pre- lly, sometimes in a single ward or in several wards of some metimes in various institutions of a city or town. Whether as an epidemic over the inhabitants of an entire district, ue, and the exanthemata, is more doubtful. Between 1841 said to have been the case in various parts of the United Volkmann and Hirsch are of opinion that the disease was rom erysipelas and allied rather to diphtheria.

l the poison of erysipelas often clings to particular wards, icular beds, with extreme obstinacy. Mr Savory, in the n.' for 1873, remarks that at St Bartholomew's, during a which occurred there, the disease almost always, in passing to another, attacked the nearest patient who had an open he late Mr de Morgan, in 'Holmes' System of Surgery,' hority of Dr Goodfellow, a most extraordinary instance in in regular order throughout a ward of thirteen beds to ient in turn, going down one side of the ward and then up The following case was observed by Mr de Morgan ing been found at the Middlesex Hospital that patients djacent beds with a window between them were particu- ttacked, the suspicion arose that this might be due to the ustbin in the area below. It was cleaned out and there cases. But two years later the disease reappeared, where- nd that the dustbin had again become foul, although no ium from it could be detected. The adoption of the same ore rendered the beds again healthy. In this instance it e doubted whether the impure air actually conveyed a

poison to the patients, or whether it merely predisposed them to attacked. But no such doubt seems to be admissible in regard to anot set of cases, recorded by Dr König, of Rostock, in the 'Arch. d. H kunde' for 1870. In the hospital of that town a small epidemic erysipelas was clearly traced to infection from the cushion of the operat table. This cushion was deeply discoloured with blood, and from the it was removed no fresh case occurred. The cushion was now soaked water, and a brownish solution was obtained which was inoculated u two rabbits, with the result that one of them had an affection which clo resembled erysipelas. The contagion is undoubtedly conveyed not o by direct contact with a patient but also by fomites, probably by the and there is reason to believe by the dead body.

Dr J. Orth, of Bonn, recorded in the 'Arch. für experim. Path.' 1873, a series of experiments by which he has demonstrated the possibi of infecting rabbits by the subcutaneous injection of fluid taken from erysipelatous bulla in man; he also transmitted the disease from rabbit rabbit by inoculating with liquid from inflamed and œdematous parts of skin and even with blood from animals already infected. That erysip can be conveyed in a similar manner from one human being to another se to be established by an old observation made by Doepp, who vaccinated n children with lymph from a child who on the following day fell ill w erysipelas; all were attacked by the latter disease.

Orth found motionless micrococci in the infecting fluid with which made his experiments on rabbits, and he succeeded in producing erysip by inoculating animals with micrococci that had undergone artificial cu vation. The presence of such organisms in erysipelas had, indeed, b previously demonstrated by Lukomsky and by Hüter, not only in tiss affected with the disease and in the œdematous fluid which saturates th but even—in small numbers—in the blood. Subsequent investigations Fehleisen and Koch in 1883 have confirmed the constant presence *Micrococcus erysipelatosus*. Lastly, a cancerous tumour has been injected w a pure cultivation of this microbe, and erysipelas followed. It often is fou as a streptococcus in the thin serum of a bulla and pus of an abscess—and the lymph channels; but is absent or sparingly present in the blood. It is cessively minute, smaller than the granules observed in the lymph of vacci

Thus all the tests enumerated in p. 13 seem to be satisfied, and may conclude that erysipelas is a "specific disease."

Phlegmonous erysipelas.—We must now revert to the question of relations between the various forms of disease that have been included un the name of erysipelas. As regards the "spreading inflammation" f mentioned, Volkmann himself admits that acute diffused suppuration, sev diphtheria, or even puerperal fever, when introduced into a hospital free fr erysipelas, often forms the starting-point of an outbreak. He cites a case Pujos, in which a man who had a severe gunshot wound of the foot die erysipelas, or, as he says, "pseudo-erysipelas," which in several places passed on to gangrene. A brother, who attended him, was attacked w erysipelas of the face, and so was a nurse; and several other persons bec affected with phlegmonous inflammations. Volkmann adds that those attend upon cases of puerperal fever often become the subjects of facial sipelas. Such occurrences are the more striking because it is admitted all hands that contagiousness is a far less conspicuous feature of erysip than it is of many specific maladies. But, after all, it is quite possible

without giving up the view that erysipelas is a specific be that there are certain cases of puerperal fever, or of puration, or of diffused phlegmonous inflammation, which to the poison of erysipelas, and that these cases alone are ucing it in their turn. Or it may be that just as in culti- nts there is great difficulty in keeping fluids free from nination with foreign bacteria, so the discharges from dis- living patients afford a favourable nidus for the growth of which may have no relation to the disease from which these ing. In phlegmonous erysipelas it is quite possible that *es aureus* may be mingled with *Streptococcus erysipelatosus*.

y it was thought that erysipelas of the face and head, as differed altogether from the surgical affection in one very t, namely, in appearing upon the unbroken skin, inde- wound or abrasion. Trousseau, however, pointed out that om some slight breach of surface, as from a suppurating gle of the eye, which the patient may have scratched, or czematous eruption on the nose, or from a fissure at the uth, or from a herpetic affection of the fauces, or even from o a decayed tooth. This view has since been adopted both d by Zülzer. A most careful record of observations for the g its correctness was made by König. Among twenty-nine as of the face or scalp which occurred in the inmates of a ain, fifteen were traceable to previous injury of the affected mainder no such starting-point could be found, but in all of as the seat of the disease, and in many of them it was so en the patient was first admitted that no complete examina- de.

ion, of course, is that the contagious microbe of the disease spot which afterwards becomes the seat of erysipelatous in- hat infection of the blood is secondary. The complete want he distribution of the cutaneous affection may be mentioned ong argument in favour of this view.

Watson mentions cases in which the incubation of erysipelas a week, and Murchison states that in his experience it was ee or four days. In Fehleisen's experimental inoculations ubation was from fifteen to sixty hours.

ason to believe that during this time local changes are , although there is no obvious inflammation of the skin. go been noticed by Frank and by Chomel that the develop- las of the face is often preceded by pain, tenderness, and cervical glands; and the late Mr Busk, after close observa- number of cases, was convinced that this was an invariable haps inflammation is already going on, but the lymphatics udation as fast as it appears. The possibility of such a he inflammatory process is expressly admitted by Cohnheim. ion cases, too, seem to show that the infective microzymes ust at the same time be multiplying themselves locally, for bered that the lymph which conveyed erysipelas was taken a child in whom the disease did not appear until the follow- here any difficulty in imagining that the blood may even at

this early period be contaminated both with microzymes and with inflamm
tory products, whether by direct absorption through the veins or by tra
mission through the lymph-glands and onwards through the thoracic du
And thus it is quite easy, without supposing a primary infection of t
blood, to account for the production of constitutional symptoms, which se
to be premonitory of the local affection but are really not so. Watson s
that before the outbreak of erysipelas the patient often feels ill, shive
feeble, languid, and often drowsy; that his pulse is very frequent; that
may suffer from nausea, vomiting, and even diarrhœa. It must be add
however, that he speaks of sore-throat "as an early, and almost a consta
accompaniment of the complaint." Volkmann declares that in the ma
cases in which he has watched the development of erysipelas in patien
already in hospital, whose temperatures had been systematically obser
for some time previously, he has never seen any prodromal symptoms wh
ever. In his opinion, whenever they seem to be present, the fact really
that the local affection has already begun; but in some deeply seated pa
so that it remains undiscovered. Now, Trousseau has maintained th
erysipelas of the face often starts from the mucous membrane of the pal
and fauces, and reaches the skin by passing outwards through the nostr
It is obvious that such an affection might well account for the early swelli
of lymph-glands already alluded to.

A case in point came under the author's observation in 1882. A m
who had had jaundice for some time was admitted under my care on June 24
His temperature was then 105°. He said that his febrile symptoms had beg
with a slight rigor on the evening of the 22nd. There was no evidence
pylephlebitis or other local affection of the liver to account for the pyrex
On the 26th the end of the nose was found to be red, and a blush
erysipelas soon spread over the face. It was then remembered that when
first came into the hospital he complained of sore-throat, and that on t
25th the clinical clerk had noticed the submaxillary glands to be swollen.

Onset and exanthem.—The onset of erysipelas is usually sudden, and it
often marked by rigors, but in persons already suffering from a febrile illn
the increase of pyrexia may be comparatively trifling. The late Dr Woo
man (in his translation of Wunderlich's work) remarks that even in adu
epileptiform convulsions are not uncommon. The temperature may rise
twelve hours to 104°, and usually reaches its *fastigium*, which may be at 10
or even 106°, within the first two or three days.

In some cases severe febrile symptoms may set in and last two or thr
days before the rash appears. This "prodromal" period between that
incubation and the appearance of the exanthem is exactly like those
scarlatina, measles, and smallpox.

Usually, however, as soon as the fever sets in, or within a few hou
some part of the skin, usually near the angle of the eye or the ala of t
nose, begins to burn and tingle and becomes red and swollen. The redne
rapidly deepens into a crimson blush, which fades under pressure, but retur
as soon as the pressure is removed. Its extent increases until in a day
two it may cover the whole of the face. Where it is spreading, it has
sharply-defined, raised border, beyond which, however, projecting process
advancing into the subcutaneous tissue may be felt with the finger. On th
other hand, where it is stationary, its edge fades off gradually into th
healthy skin beyond. There is extreme swelling, especially where the sk
is loose, as upon the eyelids; these become converted into rounded pu

is impossible to separate them, so as to get a view of the atures are so altered that the patient cannot be recognised. se and shining, though it may be made to pit by keeping e on it. There are often a few scattered vesicles, or blebs, a large size; Volkmann confirms a statement originally that minute vesicles can always be seen with a lens.

four days the disease may go on spreading, until, if it began the head, it may cover the whole surface down to the root olkmann, however, remarks that the chin always remains The conditions which determine its advance in one line ier have lately been carefully studied by Pfleger, whose y Zülzer with approval. It would seem that this depends arrangement of the subcutaneous connective-tissue bundles; interlace, so as to form rhomboidal meshes, but these are or oblique, whereas upon the chin their direction is vertical. tains that wherever the skin is tied down to the deeper of erysipelas is retarded or arrested, as for example, along ilium and Poupart's ligament. Erysipelas of the face and nds far upon the chest; but when the disease begins upon n a limb, it may spread until it has covered the whole body. ancing, however, it subsides in the parts first attacked while o elsewhere; hence it is never a universal or even a sym- n. Beyond an affected area small islets of redness may not een, but these are always connected with it subcutaneously. es that erysipelas never gives rise to two or more patches at ne another: it may, indeed, happen that in a case of double stumps are affected; or that in a case of erysipelas of the breaks out a few days later round a pimple on the leg; ould be regarded as multiple attacks of the disease.*

cium.—During an epidemic of erysipelas in a hospital, it is ing for cases of sore-throat to occur, which are evidently re, but in which the skin remains unaffected. An account ne disease has been given by Cornil, based upon a study of He describes a shining, purple-red, œdematous swelling of etimes accompanied with the formation of bullæ. The e no part in it. The lymph-glands below the jaws and in h enlarged. There is considerable pain in swallowing, and use flow of saliva.

The minute anatomy of erysipelas was first studied by more recently by Volkmann and Steudener. In the dead is so little marked, from the redness and swelling having y disappeared, that Volkmann evidently was surprised at er strata of the cutis, as well as the subcutaneous tissue, enormous numbers of granular leucocytes. He gives a icroscopical appearances, in which the cells are seen packed ide. In the more superficial strata, they are, however, paringly. The structure of the bullæ was investigated in by Dr Haight, of New York. He found them to be divided

f the case of a child in whom, after a wound of the temple, redness first subsequently upon the chest, the right arm, and the left wrist in turn; t the morbid process might not have been continuous over the back of —C. H. F.

into loculi by irregular septa which are made up of cells of the rete, dra out into long, spindle-shaped and branching processes. The fluid of the bu of course contains numerous leucocytes, and is often converted into p The micrococci of the contagion are found in the fluid that fills the vesi or blebs, and in greater numbers in the lymph-spaces of the affec cutis.

Symptoms.—While the local process is thus running its course, the pyre continues, the temperature rising and falling irregularly, or remaining nearly the same level. According to Dr Reynolds it is frequently lower the evening than in the morning. The pulse is quick, and it is gener soft and feeble; it may be dicrotic or intermittent. There is usually m or less delirium at night, and sometimes violent maniacal excitement. T patient often complains of headache, sleeplessness, and irritability of si and hearing. He has no appetite, and is thirsty; the tongue is thic coated; he has nausea and may vomit repeatedly; there is often diarrh with extremely fœtid stools. The urine is scanty and commonly albumino and may even contain casts and blood.

The duration of erysipelas is variously stated by different writers; it in fact, very uncertain. Billroth says it seldom reaches fourteen da Volkmann puts it at about six or eight. When the disease wanders o the body and limbs it may run on for weeks, or even months. The f defervescence is usually sudden, the temperature falling to the normal p in a few hours.

The subsidence of the exanthem is also rapid, and then the s becomes pale and flaccid, and shrivels; at the same time other parts of surface may be at the height or the beginning of the morbid proc Volkmann and Steudener have investigated the histology of this stag the process; they find that in the subcutaneous tissue the leucocytes appear with extraordinary rapidity, breaking down in a few hours int granular *débris*; in the superficial layers of the cutis they remain visibl little longer, but within a day or two all signs of tissue changes vanish. the meantime the vesicles or bullæ have dried up into yellowish crusts. T cuticle subsequently desquamates, and is detached either in flakes or s branny powder. When the scalp has been the seat of the disease the h falls out for a time, but is soon reproduced.

But it does not always happen, even when a case of erysipelas e in recovery, that the local affection subsides thus favourably. Delic parts, such as the eyelids, the prepuce, or the labia sometimes slough, consequence of the tension to which they have been subjected. In ot cases, when the swelling has gone down, abscesses form here and th beneath the skin, and need lancing. Suppuration of the swollen lym glands is a very exceptional occurrence.

Recurrence.—Erysipelas seems to have no tendency to protect agai its own recurrence in the same individual, or rather, as Fehleisen found, protection is only for a very short period. In surgical wards it used to no uncommon thing for a patient to have two or even three success attacks while a large wound was healing. Women sometimes have the dise once or oftener every year. In such cases it is often directly excited cold, as by exposure to an east wind; but Zülzer and other writers say t it almost always has a starting-point in some local affection of the face the nasal mucous membrane, the ear, or the lachrymal passages. In cou of time this "recurrent" or "habitual" erysipelas leads to a persist

nduration of the nose, ears, or eyelids, which greatly deforms

le effect of a single attack of the disease is that its sub-
etimes followed by the disappearance of long-standing
ions. This seems to have been first noticed by Cazenave
ic eczema or lupus. More recently it has been found that
s growths of considerable size may vanish in a similar way.
copies of photographs taken from a woman under the care
no had several tumours on the face, varying in size from a
igeon's egg: a portion of one of them was excised, where-
ttacked with erysipelas, and this led to the absoption of
n two other patients enormous tumours of the cervical
nt marked decrease of size under similar circumstances;
lapse, however, set in, which in one case proved fatal, while
se, as recovery took place, the growth rapidly regained its
ons. This patient had been intentionally exposed to ery-
ion, in the hope that it might act beneficially upon her dis-
our from the former patient, who died, was examined histo-
lfleisch, who found that almost the whole of it had undergone
on, so that only in certain portions could the structure of a
na still be recognised.

.—When there have been severe cerebral symptoms it has
osed that inflammation has extended from the scalp or face
es of the brain. Examination after death, however, has very
his suspicion. Volkmann says that he has seen the disease
rom the orbit through the sphenoidal fissure, the orbital fat
llen and infiltrated with pus, as had been indicated during
otrusion of the eyeball.

ommon for the disease, when it affects the fauces, to extend
larynx, and so to destroy life, unless tracheotomy succeeds
danger. The folds at the entrance of the larynx are then
œdematous, or infiltrated with pus. This was the immediate
n the case of John Stuart Mill, who died at Avignon of
.

es the immediate cause of death is pneumonia or pleurisy.
Hospital Reports' for 1861, Dr Wilks recorded two cases in
of the abdomen appeared to have set up fatal peritonitis.

mplication of which two instances have been observed in
cher ('Arch, Génér.,' 1864) is ulcer of the duodenum;
nterest, on account of the occurrence of a similar affec-
ult of burns and scalds. In a third example, reported by
h. Génér.,' 1865), there were ulcers in the lower part of the
Bayer has related in the 'Arch. d. Heilkunde' for 1870, a
vere hæmorrhage from the bowels preceded death; the only
the autopsy was intense congestion of the ileum.

questioned whether pyæmia is frequently associated with ery-
me patient. Zülzer says that this is not the case, except in
s form attended with diffuse suppuration of the connective
observations at Guy's Hospital seem to bear out this state-
nn, however, declares that metastatic abscesses were present
d to one half of all his fatal cases; but he adds that every
nts also had a severe wound, and so there may have been an

embolic and infectious process going on independently of the spec contagion.

Sequelæ.—Scars only follow the phlegmonous form of erysipelas, l when repeated attacks affect the same part, a chronic hypertrophy of t skin with œdema durum of the subcutaneous tissues and lymphatic engor ment sometimes follows: and thus the condition known as elephas of t legs and the scrotum is often the result of recurrent erysipelas.

Various affections of the eyes have also been observed to follow an atta of erysipelas—opacity of the cornea and even optic neuritis.*

It is very rare for the febrile albuminuria which is so very frequen symptom of this malady to persist, so that Bright's disease is scarcely ev to be traced to this origin.

Diagnosis.—The recognition of erysipelas is easy, if we leave out consideration the theoretical doubts as to the relation which it bears, the one hand, to phlegmonous dermatitis and diffused suppuration, and, the other hand, to certain forms of erythema. Zülzer speaks of the œdem tous form of anthrax as being sometimes mistaken for it. There is anoth disease, which is often mistaken for erysipelas, and that is herpes zoster the forehead and face.†

Prognosis.—This is generally favourable for the cases of erysipelas th come under the care of physicians, except in old people and in infant in the latter it not infrequently appears at the umbilicus, and proves rapid fatal. Even in surgical practice recovery so generally takes place that it difficult, as Dr Wilks has remarked, to understand how it came to pass th the common form of certificate submitted to a magistrate in cases of slig wounds used to be that there was no danger, "unless erysipelas shou ensue." It may be, as he suggests, that what really was pyæmia was oft set down to erysipelas. There can be no doubt, however, that erysipe itself was once far more fatal, at least in certain institutions, than it is present; thus Volkmann says that in the old Hôtel Dieu at Paris it wou often happen that the majority of surgical cases attacked by it ended death. But probably much of the difference lies in the fact that patien suffering from erysipelas are now well supplied with beef-tea and wine a brandy, instead of being bled and leeched and kept upon low diet.

Even in adults a fatal termination is apt to occur when the patient h chronic disease of the kidneys, or has been intemperate, or is otherw broken down in health. Before death the temperature usually rises to great height, and sometimes it goes on rising for a short time afterwards.

Treatment.—In this country we believe that the tincture of steel has marked influence in checking erysipelas. Mr de Morgan from his experien at the Middlesex Hospital, spoke most positively of its efficacy in reducing t

* See on this point, however, an abstract of numerous observations made in Rus which appeared in the 'London Medical Record' for November, 1888, p. 473.

† Only a few weeks ago I found lying in the hospital a youth whose face was cove with flour, through which a diffused redness was visible, while the eyelids were enormou swollen. The first glance, however, showed that the affection scarcely, if at all, passed middle line of the forehead; and according to the account which the patient himself ga it had begun with an eruption of vesicles. I therefore made a confident diagnosis that case was one of zoster, and dropped atropine into the eye, on account of the danger iritis. At my next visit, the redness and swelling had disappeared without desquamatio and the vesicles had dried up into characteristic dark brown eschars.—C. H. F.

isease, so that it subsides in from two to four days, instead or ten days. He gave at least a drachm or a drachm and sometimes as much as an ounce and a half or two ounces. it is more usual to give quinine.

comes under observation at the very commencement of the is believed by many to be useful. Dr Ringer believes that red at this period may cut short the attack. Given after the appeared it usually brings down the temperature but has nd possibly may do harm.

y years ago Mr Higginbottom introduced the practice of of silver round the circumference of a patch of erysipelas, its spread might in this way be arrested. This was termed ethod, and there are still some who have faith in it, nn, who prefers the silver salt to the tincture of iodine, een suggested with a similar object. The part must be first with soap and water, or with a solution of soda or potash, so at from its surface. It may then be brushed over with a trate in from eight to ten parts of distilled water for a inches round the reddened area, on all sides of it. Some- ceases to spread, and on the following morning defervescence in charts given by Volkmann. The application of collodion ed surface is said to be useful as a palliative. The traditional ur over the affected skin; and this is certainly a valuable g the local smarting and irritation, probably by excluding st strongly recommends the local application of carbolic acid tine (1 to 15) painted on the affected skin every hour. rt of phenol to 20 of olive oil) has been used with the same ng protection from the air and antiseptic action at the same

delirium accompanies erysipelas of the head, an ice-bag is relief, and to be altogether harmless. If the swelling is so ne seems likely to occur, a series of minute incisions affords eans of preventing it.

n necessary to employ cold baths in the treatment of ery- pyrexia, although reaching a great height, usually subsides in itself a source of danger. But if a high temperature is ore than a week, one must adopt measures to reduce it. a chart of a case in which a cold bath was given on the ninth neter then indicated 105·8°; in the course of the following ure was repeated thirteen times and the patient recovered. collapse seems to be impending, it may be well to give tur- recommended by Copland. At least it is difficult not to which he has recorded the details in his 'Dictionary,' and n who was already comatose, with a black tongue, and with ld not be counted, was apparently saved by this drug, doses of three drachms in an electuary of castor-oil and

DIPHTHERIA *

" Inde ubi per fauces pectus complerat et ipsum
Morbida vis in cor mœstum confluxerit ægris
Omnia tum vero vitaï claustra lababant
Spiritus ore foras tetrum volebat odorem."
LUCRETIUS.

History of the recognition of the disease—Its definition—Its anatomy and p
logy—Relation to croup and to "diphtheritic inflammation"—Mi
—Ætiology—Contagion—Course and symptoms—Varieties—Complica
and Sequelæ—Albuminuria—Paralysis—Prognosis—Treatment.

ABOUT the year 1855 attention was drawn in England to the preva
of an epidemic disease, resembling scarlet fever in being generally atte
with an affection of the fauces, but differing from it in the charact
that affection, and in the absence of a rash. Many accomplished physi
including Addison, declared that they had never seen this disease be
and although a search into medical literature brought to light se
instances of its occurrence, both sporadically and in an epidemic form
the fact remained that, at least within the present century, it had
previously spread over any considerable part of the country at the
time. In France it had been well known for several years, and had
carefully studied in 1818 by Bretonneau, of Tours, who had given i
name of *diphthérite* (διφθέρα = leather or membrane). In fact, the epi
of 1855–57 was introduced from France, and was known as the "Bou
sore-throat."

It is impossible to distinguish, in the accounts of the ancients, bet
the angina of scarlatina and that of diphtheria, or even of syphilis.
undoubted epidemics of diphtheria occurred in Spain in the sevent
century, and were described in 1614 by Mercatus, physician to the
Philip II and III, and in 1670 by Heredia, physician to Philip IV
spread to the Spanish provinces in Italy, and was epidemic at Napl
1618, when it was described by Cortesius (1625), Aetius Cletus (1
and the celebrated Danish anatomist Bartholinus (1646). It pro
appeared in Edinburgh in 1733, according to Fothergill, from whose tr
on 'The Putrid Sore-throat attended with Ulcers' the above referenc
taken. In 1746 there was an epidemic at Bromley, in 1747 o
Greenwich, and in 1749 one in Cornwall, which was described i
'Philosophical Transactions' by Dr Starr. Its reappearance as an epi
in South Wales in 1849 was recorded by Mr J. D. Brown.

There is reason to believe that the *cynanche maligna* of the older w

* *Synonyms.*—Ulcera Syriaca? (Aretæus, c. 70 A.D.), Ulcera pestifera in to
(Paulus Aegineta, c. 680 A.D.), Epidemica gutturis lues, Angina puerorum epi
(Bartholinus, 1646), Putrid sore-throat attended with ulcers (Fothergill, 1748).—*Fr.*
gorge gangréneux (Chomel), Angine couenneuse (Louis), Diphthérie (Bretonneau)—
Bösartige Rachenbräune—*Ital.* Morbus strangulatorius (Cletus, 1636)—*Sp.* Angina m
(Heredia, 1673), Garrodillo.

y cases of scarlatina anginosa, but also some which would as diphtherial.

this disorder has constantly prevailed with more or less ng up here and there in different districts of England, as countries. Numerous investigations have been made as to ode of propagation, but in regard to many points doubts particularly about its relations to certain other diseases. which is now almost universally applied to it is Diphtheria. n, has indeed proposed to term it "Cynanche contagiosa;" ms to have no chance of being generally adopted, although dly would avoid many sources of error and confusion, by anyone who may read Senator's paper in the second n Clinical Lectures, published by the New Sydenham

pathology.—As the term diphtheria implies, the essential sore-throat is the presence of a membranous substance, nore or less extensively over the tonsils, the uvula, or other nneau, and his followers, great stress was laid upon the stance could be detached from the mucous membrane be- the latter was then found to be only reddened and t the utmost slightly excoriated. They pointed out that layer, being of an ash-grey colour, often simulated very rance produced by sloughing of the parts it covered; and, disease had long been known under the name of gangrenous nant sore-throat. But they declared that such appearances and that no considerable loss of substance occurred, still e sloughing. Finding, moreover, that in many cases the down into the larynx, they formed the opinion that the as "membranous croup" was only a form of diphtheria. vriters the subject has been developed in a very different now, in the first volume of his well-known 'Archiv' (pub- distinguished between a "croupous" form of inflammation "diphtheritic" one. In the former, he said, the exudation e surface of the mucous membrane; but in the latter its the superficial layer of that membrane, which generally hing as the result of its presence. These definitions, being equent writers without a due comprehension of what pro- ow's real meaning, have led to great confusion, which is to be cleared up. A good description of the characters of flammation," in the sense attributed to it by the great erlin school, is given by Rindfleisch in his 'Pathological makes it consist of an infiltration of newly formed cells ithelial connective tissue of the mucous membrane (the llowing an idea originally suggested by Buhl, he teaches nce of this infiltration compresses the blood-vessels, and so lation through the affected parts, brings their nutrition to a eprives them of life. As examples of such an affection, he nflammation of the urinary bladder that is set up by decom- nant urine, the more severe forms of dysentery, and the mation of the uterus and vagina which may occur imme- turition; and he points out that a similar condition may be

met with upon other free surfaces: upon the skin, as in the more destru tive kinds of variolous eruption, which lead to permanent pitting; a upon wounds, as in hospital gangrene. It must be admitted that all the forms of inflammation have characters in common, and deserve to be know by a special name (cf. p. 52).

But, if this view is to be taken of the distinguishing features of "dip theritic" inflammation, one sees at once that the disease "diphtheria" fin no place in it. Accordingly, Rindfleisch describes the latter under croupo inflammation, and by the name of "pharyngeal croup." He gives a very fu account, which the writer can confirm by his own observations, of the hist logical characters of the pellicles which are found upon the surface of t mucous membranes in what we call diphtheria. On the palate and tonsils says they consist, not of fibrin, but entirely of cells, which have undergo a peculiar glassy change, and have become fused together, so that a seri of little fissures alone indicates the original interspaces between the According to E. Wagner, these cells are all derived from the original ep thelial elements of the affected part. In the air-passages, on the other han Rindfleisch describes the pellicles as laminated, and consisting of layers cells which alternate at tolerably regular intervals with layers of a hom geneous substance, apparently fibrin.

Some writers have attached special importance to the fact that different affections of the throat there are great variations in the degree which false membranes adhere to the mucous surface. But these variatio depend mainly upon the *seat* of the lesion. As between the several diseas the distinction is valueless. The fact that in the very same case of diphther the membrane on the tonsils and that in the larynx differ essentially in the histology is of itself sufficient to show that our views in regard to the disea cannot be based on its pathological anatomy alone.

In the same patient we often find, after death, upon the fauces a thi grey, ragged and decomposing membrane, which bled and stank durin life—and the same characters mark the disease when it spreads from t palate and tonsils to the nares and the pharynx; on the posterior surface the epiglottis, in the larynx and the trachea, a thick, white, firm, an continuous layer with abundance of fibrin, along with leucocytes, but wit no blood-discs; and in the bronchial tubes very scanty shreds of membran with abundance of mucus and pus.

Moreover, in the fauces themselves, the morbid changes present wid variations of intensity. There is, in the first place, "*diphtheria sin diphtherâ*," a variety in which the tonsils and uvula are merely reddene and affected with catarrhal inflammation without a "false membrane, but which can be plainly recognised as diphtheria, because it occurs in th same family simultaneously with the more severe forms. Even when mem branes are present, they differ greatly in extent and thickness in differen cases. Rindfleisch, indeed, speaks of the morbid process in the pharynx a being always insular, and as consisting of circumscribed milk-white spots, o an intensely hyperæmic base, which are never raised more than half a lin above the level of the mucous surface. But no physician who has had muc experience of the disease can be ignorant that in many instances the whol fauces, tonsils, and uvula are covered with a thick tough layer, which look like wash-leather, and may ultimately be detached *en masse*. In 1858, D Fuller exhibited to the Pathological Society a cylindrical cast of the pharyn four inches long, which had been ejected by a girl aged eleven. Indeed, i

nbranes are removed, or become detached, they may be and again. This is well known to all who have observed s of the disease; and Dr Burdon Sanderson vouches for it as s own observation in 1859. "At Crowle," he says, "I had of seeing an example of extreme rapidity of reproduction of ryngeal concretion; and I have notes of a case, in a robust gfordbury, watched with the greatest care and attention, in of the mucous surfaces the pellicle must have been renewed thirty times in the course of three or four weeks." This portant, because Rindfleisch expressly denies that false mem- palate or tonsils ever recur in the same place, unless they ped off before their spontaneous maturation; and Oertel (in Ziemssen's 'Cyclopædia') likewise limits the possibility of ion by arbitrary conditions. Another point on which ts positively is the frequent occurrence of ulceration, which, hat he observed, led to perforation of the palate; and he stance in which the tonsil and the arch of the velum were hat a continuous sloughing surface extended to the pharynx. erved that in cases which presented exactly the appearance he uvula and tonsils in a state of "putrid dissolution," and st detached—the fauces were found after all to be intact membranes at length came away; but all recent writers, both rman, admit that Bretonneau went too far in denying the ngrene.

e think, clear that the definition of diphtheria as a disease n the histology of the exudation, but on its pathology and natural history: in other words, less on anatomical than on aracters.

ic course, the infective power, the kind of fever, the effect s, and the sequent paralysis—these are the distinctive n mark the Boulogne sore-throat or Cynanche contagiosa or ther in the fauces or the larynx.

primarily local?—An important pathological question has dered—namely, whether the disease is from the first consti- ether Oertel is right in maintaining that it is originally ts the system secondarily. His chief argument is based between the results obtained in the experimental transmission the lower animals, and those which follow the inoculation glanders. In the former case, he says, the affection always at the spot to which the poison is applied; in the latter it s the nasal membrane, even when the contagious principle d beneath the skin.

f this statement is accurate, it leaves diphtheria in the as erysipelas and syphilis. There can be no doubt that a special tendency to attack the fauces, and that the fre- al diphtheria does not merely mean that the poison comes h this mucous surface more frequently than with any other. etimes when the affection has begun elsewhere the throat ily, apart from any direct extension of the morbid process. as admitted into Guy's Hospital with an affection of the oubtful nature, but before his death the palate and tonsils l with a false membrane, so as to clear up the nature of the

case; and in epidemics of conjunctival diphtheria, of which several ha occurred in Berlin, the fauces have occasionally been attacked.

Ætiology.—The first point to be insisted on is that diphtheria is contagio This, indeed, is not always an obvious feature of the disease as it is se in private practice; for if several cases occur in succession in the same hou one cannot say whether they all depend upon a common cause, or whet one has given rise to another. But when a patient suffering from diphthe is admitted into a hospital, it often happens that persons occupying beds the same ward are subsequently attacked. Sir William Jenner rela several instances in which patients sent into the country infected those w whom they came into contact. The epidemic in East Kent in 1856 w ushered in by a striking instance. "No case of diphtheria had ever be seen in Folkestone during my time," says Mr Eastes, "until Isabella W- aged 4¾, arrived from Boulogne on the evening of July 2nd, being then an advanced stage of the disease. She died on the following day. On t 6th, her sister, aged ten, was attacked, who had always resided on t East Cliff; another case occurred in the same house three days after, a they all terminated fatally." But the most conclusive of all argument furnished by the unhappily frequent instances in which a medical man h fallen sick with diphtheria, immediately after having had phlegm cough into his mouth or nose by a patient whose fauces he was examining; or af having used his lips to inflate his patient's lungs, or blow through a trach tomy tube. Oertel mentions by name five physicians whose lives were th sacrificed, among whom was Valleix, the writer on neuralgia. A lamental case of death from such self-devotion occurred in the person of a hou surgeon at King's College Hospital, and similar cases are not rare in oth hospitals. It was believed that the late Princess Alice of Hesse contract the disease by kissing her child who was ill of diphtheria. A case belongi to the same class is that of Dr Wiessbauer's child, who was attacked short after having put into his mouth a cannula that had just been removed fr the throat of a patient suffering from diphtheria.

It must, however, be admitted that even when the conditions see favourable, diphtheria sometimes fails to spread. Those in attendance up patients suffering from diphtheria have recklessly or ignorantly blown throu tracheotomy tubes for the purpose of cleaning them over and over aga without suffering ill effects; and Trousseau in 1828 and afterwards Pet actually inoculated themselves on the palate and tonsils with diphtheri matter, and were none the worse. There is nothing very surprising in the facts, for persons exposed to contagion from other diseases not infrequent escape, and even inoculations designedly made sometimes fail with u doubtedly infectious maladies. Trousseau himself firmly believed in th contagious nature of diphtheria, and at present no one doubts it.

In the 'Guy's Hospital Reports' for 1877 Dr Fagge recorded series of fifty cases of what would be regarded as diphtheria in th common acceptation of the term; and whereas eleven of fifteen cas in which the larynx was free could be brought into connection with oth cases (by either ascent or descent) only eight out of thirty-five cases which the air-passages were involved could be so brought; moreover, all th eight cases just referred to were cases in which the fauces were severe affected; of ten cases in which there were only very slight patches of fal membrane upon the tonsils or palate, there was not one in which a

ctiousness was obtained. We shall hereafter have to discuss
acts must be accounted for by supposing that when diphtheria
-passages it is less contagious than usual, or whether they
arge proportion of the cases in question belong to a different
pecific inflammation, attended with the formation of false
the chapter on Laryngitis).
f the opinion that diphtheria is highly infectious must be men-
onal fact that its contagious principle sometimes adheres with
o particular houses or apartments. Squire mentions that in a
n Scotland a visitor was attacked while occupying a chamber
had occurred eleven months before. The infection from a
enerate a severe one, or *vice versâ*.
patient ceases to be infectious is not yet known. Bristowe
ease has sometimes seemed to be communicated by a child
ntly been well for two or three weeks.
—Since the study of micro-organisms in disease by modern
een applied to infectious diseases, the whole subject of
assumed a new shape. Buhl in 1867, and Hueter and
dently in 1868, discovered in the "false membrane" of
te spherical microbes, *micrococci*, either separated or joined
n chains, or as zoogloea; mixed with these are some which
To distinguish the micrococci from mere granules of
is difficult. The bacteria present a more characteristic
ecially when arranged in long chains; but similar bodies
ce in all putrid fluids.
tes are found not only in the false membranes of diphtheria
between the epithelial cells and the leucocytes), but also in
ructures. They are described as being present from the very
of the morbid process, and as extending beyond the limits
the naked eye to separate the affected from the healthy
re said to block up the lymphatic channels, to penetrate
of muscles, cartilages, and bones, and to be carried by the
neys, where they germinate afresh. Löffler finds that the
crophyte of diphtheria (which he identifies as a bacillus, not
only present in the affected mucous membranes, not in the
idneys.
question that micrococci are really present in the tissues
d by the diphtheritic process; but it is doubtful whether
detected in the blood, and great caution is required in
n of appearances which have been held to indicate the
ganic forms in parenchymatous organs, such as the kidneys.
is important to remember that the first observations of
de, not upon cases of "diphtheria," as we define the disease,
f hospital gangrene. In other words, his facts apply rather
inflammation" in the wider sense of that term than to

nclusion probably applies to a large number of the experi-
different observers, in which animals have been inoculated
tic" products. Portions of false membrane have been
the skin, or introduced into the trachea of the dog or
he cornea of the eye has been punctured with a poisoned
ult has been a disease which proved fatal in two or three

dogs, and the tissues in the neighbourhood of the spot to which the infecti material was applied have been found full of masses of micrococci. B Recklinghausen succeeded in generating a "diphtheritic keratitis" in rabbit by inoculating the cornea with matters from a case of hospital gr grene. In Oertel's series of experiments he passed on the infection fr one animal to another, choosing sometimes the trachea, and sometimes t muscles of the neck or chest, as the seat of the induced disease; and aft six transmissions he obtained a product capable of giving rise to the formati of a false membrane in the air-passages of the last animal experimented on

Probably the constitutional disturbance caused by the so-called "dip theritic" inflammation, excited by inoculation in animals, is in many ca only a form of septicæmia; but there remains strong though not conclusi evidence that there is also a specific diphtherial micro- or strepto-coccus.

On the whole it seems probable that the rod-shaped microbes abo mentioned are only the common bacterium of putrefaction (*B. termo*), a therefore most common when warmth and moisture are combined with fi access of air as on the fauces; but that the micrococci (particularly th which occur in chains ?) are specific and pathogenic organisms. It appe that the false membranes in the larynx and trachea contain scarcely a microphytes.†

Predisposing causes.—It must be admitted that diphtheria often sprin up in isolated houses, and under circumstances which accord ill with t theory that it must be due to a specific contagion. In this there is not, deed, anything to be wondered at, for the same thing is likewise true of the exanthemata. Dr George Johnson has advocated the opinion that ca not directly traceable to infection are often due to contaminated water, or the effluvium from foul sewers or cesspools. He relates the cases of fo children who were all attacked on the same day; three days previously cesspool, distant about twenty yards from the house, had been emptied; a the wind was blowing towards the house at the time, and conveyed to i very offensive smell. Soon afterwards two of the servants fell ill, and length the mother. The only members of the household who escaped w the father (who was away from home all day) and one servant. Scarlet fev however, is also associated with bad drains and foul smells, and probal in the same way—namely, by these conditions producing a sore-throat, which the germs of scarlatina or of diphtheria alight and find a favoura nidus; or perhaps we should rather say on which they are not opposed the antagonism of healthy tissues (cf. p. 18).

There is reason to believe that, even when diphtheria is epidemic, a when the specific contagion has perhaps already been introduced into system, exposure to cold may act as an exciting cause in bringing out manifestation of the disease upon the fauces. At least Dr Yeats ('Ed. M Journ.,' 1876) states that this was the case in an epidemic which occurred Auchtergaven in Perthshire, between March and June, 1875. Several pers he says, were attacked who had been working late in their gardens,

* "Experimentelle Untersuchungen ü. Diphtherie," 'Deutsches Arch. f. klin. m 1871, Bd. viii. See also Roux ('Annales de l'Institut Pasteur,' 1888, Nos. 11 and 12).

† On this difficult subject, compare Cohn's statements as to the distinction betw *Micrococcus septicus* and *M. diphthericus* ('Beiträge zur Phys. der Pflanzen,' 2tes H pp. 164 *et seq.*). Eberth ('Zur Kenntniss bact. Mycosen,' 1872), and the 'Report of American National Board of Health for 1882' are also referred to by Klein, whose rema at p. 72 of his "Micro-organisms and Disease," and those by Crookshank in "Bacteriology" (pp. 117, 136, 177), should be read by the student.

t on a damp lawn, or driving after sunset. Others, who had
o bed for a considerable time from other causes, were infected
ppearance in the open air. It is obvious that these cases
ant bearing on the question of the existence of a membranous
rom diphtheria, since the former affection is supposed to be due
sure to cold.

ld, altogether independently of epidemic influences, can ever
ecific inflammation of the fauces, attended with the formation
rane, it is very difficult to say; it is, however, certain that
process may result from the action of more powerful irritants.
n the 'Guy's Hospital Reports' for 1877 are related the
ildren in whom the palate and tonsils presented appearances
se of diphtheria; but in one of them the affection was caused
a piece of hot potato, in the other by a burning stick, which
into the little patient's mouth by another child. A similar
een met with in three or four cases of scald of the throat, in
ater or steam had been sucked out of a teapot or kettle.
found a well-marked separable false membrane on the lingual
piglottis, as well as on the base of the tongue, in a woman who
de with white precipitate, but who lived six or seven days after
poison. There was extensive "diphtheritic inflammation"
us, the stomach, and the lower part of the intestines.

nown that, instead of attacking the throat, diphtheria some-
lf on the mucous membrane of the genital organs, or of the
enuded parts of the cutaneous surface. The study of such
isease ought to throw light on its ætiology. Dr Braxton
ded in the 'Guy's Hospital Reports' for 1871 an epidemic
n the obstetric wards, in which two women were in turn
iphtheria of the labia, while two others had the ordinary
fauces. Moreover, in Berlin and some of the other cities
any, where conjunctival diphtheria has prevailed to a remark-
re have in each epidemic been some cases in which the disease
er downwards through the lachrymal passages to the palate
n the reverse direction upwards, or in which the throat and
een attacked simultaneously and independently. Moreover,
l affection, as in a case observed by the writer, sometimes
quela of measles, just like diphtheria of the fauces. The
close a relation between these different forms of the disease
argument for its specific character, and against the supposi-
ses spontaneously, as the result of defective hygienic condi-
must be added that both Jacobson (of Königsberg) and
it a sporadic variety of conjunctival diphtheria; stating, for
onorrhœal ophthalmia often assumes a diphtheritic character.
that the affection of wounds and raw surfaces known as
ne is a sporadic representative of cutaneous diphtheria.
that the conjunctival disease, even when it is of purely local
ead by contagion. If this is the fact it would be somewhat
e case of contagious porrigo of the skin, which often seems
out of simple eczema.

nd the rich are probably, in proportion to their numbers,
o diphtheria. It is notorious that the disease often carries
and robust-looking children.

Diphtheria is far more apt to attack *children* under ten years of ag than older persons; but perhaps the proportion of children appears undul large from some cases of croup being included under diphtheria.

It is doubtful whether one *season* of the year rather than another i favourable to the spread of diphtheria. Fothergill found it far mo frequent from September to December inclusive; but the very cold winter o 1860 did not hinder its epidemic diffusion in London; and it has ofte prevailed during the summer. Oertel says that diphtheria is of much le frequent occurrence towards the tropics than in the temperate zone. Ge logical conditions of the soil seem to have no part in its causation.

In England it is most prevalent in London and the south-easter counties, and in North Wales; while the mortality from it is below th average in the counties north of the Humber and in the central Midlands- including the manufacturing districts of Tyneside, Lancashire, and th West Riding, Birmingham and the Black Country, and also South Wale (See Dr E. Barnes' paper, 'Brit. Med. Journal,' July 28th, 1888.)

Diphtheria as a complication of other specific fevers.—In the chapters o measles and on scarlet fever it was mentioned that each of those complaint is now and then accompanied by an affection of the fauces resembling th of diphtheria. A fact related to Dr George Johnson by Dr Dewes, Coventry ('Lancet,' 1875), tends to show that both the specific contagia a present in some cases of this kind. Two brothers had been in successio attacked by scarlet fever, and had been separated from one another throug out the course of the disease. The elder had remained at his school to l nursed; the younger had been sent to a cottage in the country. The form passed through a mild form of the disease; the latter had it severely, a also had his fauces covered with diphtheritic exudation. After a time th boy at the cottage sickened with scarlet fever; and he, too, had a diphtherit throat. When both brothers had been convalescent for a fortnight the were allowed to be together, and even to lie in the same bed. Presently th elder one fell ill of diphtheria with laryngeal complications, and died on th third day.

Senator remarks that when scarlet fever is accompanied with th formation of a false membrane upon the fauces, the morbid process scarce ever extends to the air-passages.

Protection.—Does the fact that a person has had diphtheria imply su sequent immunity from the disease? In Simon's 'Report' for 1859 sever cases are recorded of individuals who suffered twice, at intervals of two three months or longer; and it is to be noted that the second attacks we always more severe than the first ones. Second attacks are not uncomm according to Dr Eustace Smith.

Incubation.—This period of diphtheria is said by Trousseau to be fro two to seven days. Oertel puts it at from two to five days; but he quot cases in which it was longer. One such instance is related by Jenner; th of a young lady who sickened eight days after having been sent into th country from a house where her brother and her sister had before be attacked. Senator says that the interval may be as much as three four weeks. Where there has been a direct transference of the poison fro one person's fauces to another's the period seems to be much shorte Valleix had a pellicular deposit on the tonsil the day after he became infect

a, and died in forty-eight hours. Oertel supposes that the
kely to be less prolonged during the prevalence of an epidemic,
the type is malignant.

nically we recognise diphtheria (the specific febrile disease
name, apart from so-called diphtheritic or necrotic inflam-
ed above, pp. 257–9) under two very different forms. In
e characters of an infective fever, epidemic in course, con-
, with febrile albuminuria, and other "typhoid" symptoms;
auces, and the membrane is grey and causes bleeding when
e other there is less constitutional disturbance, the cases are
hough very contagious, and the affection begins in the larynx,
more fibrinous membranes.
however, combine both characters, and beginning in the
ls descend to the larynx, trachea, and bronchi. As seen in
se cases appear to be varieties of the same disease.
theria.—The course of this disease is far from constant.
gins with marked constitutional disturbance—anorexia, head-
and loss of energy; nausea or vomiting, acceleration of
vering, followed by fever. There may from the first be
llowing, the sensation varying from a slight pricking up to a
or shooting pain which may quite prevent the patient from
this pain is rarely so extreme as in acute tonsillitis. On
auces one finds that the uvula, the palate, and the posterior
rynx are more or less swollen and of a red or violet colour.
ours, or at latest two days, one or more whitish grey spots
on some of those parts; they are at first small, and they
the same size for several days. From an early period the
f the neck, especially those near the angles of the jaws,
and painful.
stances the febrile disturbance is very slight, and in others
uration. The pellicles are sometimes detached and cast off,
seems to have entirely recovered within two or three days.
y cases the disease takes a less favourable course. The fever
emperature remaining at 102° or 103°, or mounting higher;
have fallen, it rises again on the fourth, fifth, or sixth day.
l spots on the fauces rapidly run together, and extend in
; so that the tonsils, the uvula, and all the visible parts of
in a few hours be covered with a yellowish or greyish
he cervical and submaxillary lymph-glands swell still more,
se at the angle of the jaw, just opposite the tonsil. Occa-
sues are infiltrated with inflammatory products, so that the
eck is obliterated, and the whole space from the chin to the
upied by a uniform brawny mass, with the skin red and
rysipelas. Before long the membrane begins to soften and
uiring a brownish colour, and separating here and there in
patient's breath then becomes horribly fœtid. An ichorous
run from the corners of the mouth, which excoriates the
ves rise to ulcers which in their turn become covered with
layer. At this time it is not uncommon for sore spots—
, for instance, or in the creases of the skin, even of remote
ody—to become distinctly diphtherial. If a blister should

unfortunately have been applied, the raw surface may be coated with a me brane; and leech-bites are apt to pass into unhealthy spreading ulcers.

From the commencement of these more serious local changes the con tutional symptoms begin to present those features of depression wh characterize the further progress of the case. The pulse may still be qui its beats rising to 120 or more in the minute, but it becomes daily smaller a weaker. The countenance rapidly acquires a waxy pallor, and the muscu power is remarkably enfeebled. Purpuric spots sometimes appear upon t skin. The tongue becomes dry and brown, sordes collect on the teeth a lips, and in fact a typhoid state may be developed. Such cases are genera fatal, and on *post-mortem* examination the serous membranes, and even t endocardium and the substance of the lungs, are commonly found to ecchymosed, as in septicæmia. Pneumonia or œdema of the lungs is, many instances, the immediate cause of death.

Occasionally there is delirium, but as a rule the mind remains perfec clear. At length the temperature falls below normal, to 97° or 96° F., a the pulse may become irregular and intermittent, its beats scarcely amount to 50 or even 40 in the minute. Death may either occur very gradually asthenia, or more often it takes place suddenly while the patient is in act of sitting up in bed or making some other slight movement. Such ca generally terminate between the tenth and the fourteenth days.

Fatal syncope, however, is not limited to those cases of diphtheria which the disease has been severe throughout its whole course; it so times happens when all the symptoms have been of the slightest. Jen relates such an instance. A boy, aged ten, who was convalescent from very mild attack, was attacked with vomiting, and the pulse (which h been becoming less frequent for two days) fell to 36 in the minute. Th was nothing in the patient's appearance to suggest that he was in immin danger, but notwithstanding the free use of stimulants the pulse contin to fall; by the next afternoon its beats were only 24, and soon af wards they ceased altogether. The following is one of two similar ca recorded by Dr Fagge. A little girl, in whom there were all along w marked diphtheritic patches in the fauces, seemed to have so little matter with her that she was allowed to play with other children in garden in front of the house. Early one morning she was being brou downstairs from the nursery as usual before being dressed, when it w noticed that she looked very pale. On being hastily sent for he found pulseless with her extremities perfectly cold, and in spite of all that co be done she died about eighteen hours afterwards, without having rallied the least from her collapsed condition.

Even when characteristic diphtheritic patches exist on the mucous me brane of the fauces, it sometimes happens that the real nature of the dise may be overlooked from the patient failing to complain of difficulty of sw lowing or pain in the throat. Children have been brought to the out-pati room at the hospital suffering from febrile symptoms for which no cause be found until, as a matter of routine, the fauces are examined. And so years ago a chlorotic girl, who had been an inmate of one of the wards a few days, died without anyone suspecting the real nature of her dise The chief symptom was a weakness so extreme that she could not sit even when supported. After death the fauces were found covered w false membrane.

But perhaps the most obscure of all the forms of diphtheria is

d to the *nasal mucous membrane*, or which, at least, may be
the presence of any obvious pellicles upon the pharyngeal
hief local symptom is then the escape of a thin sanguineous
nt fluid from the nostrils, the orifices of which become more
l and excoriated. Epistaxis is not infrequent, and it may
nat one would have attributed to it the bloodless appearance
out for the fact that anæmia is so constantly present in all
diphtheria. With a speculum we may sometimes perceive
ated bones are covered with a membranous layer, or casts of
ischarged from the nostrils. After the first day or two the
the affected parts is commonly of a brownish colour and
The disease not infrequently extends along the nasal duct
iva, which then becomes coated with a perfect diphtheritic
it may pass through the Eustachian tube to the tympanum,
ng or buzzing noise in the ears and deafness; perforation
place, and matter be discharged through the external

rection in which diphtheria sometimes spreads is from the
he *œsophagus*. In one fatal case at Guy's Hospital we found
all ulcers in the stomach close to the cardiac orifice, some of
ted with a distinct layer of false membrane; and instances
rded in which the whole œsophageal and gastric mucous
e taken part in the morbid process. This condition seems to
to no special symptoms.

iphtheria.—The continuity of the false membranes is often
ly over the epiglottis and the aryteno-epiglottidean folds;
this is not the case, and certain observers have in conse-
d to regard the spread of the disease to the larynx and
esult of what they have termed "auto-infection," an inocula-
cous membrane by particles of secretion drawn downwards
nspired through the mouth. Upon the epiglottis and the
false membrane is firmly adherent; it may pass straight
between the true and the false cords. Below the glottis it is
ttached to the mucous surface. It becomes thinner as it
In the trachea, at a variable distance down, it commonly
omes continuous with a muco-purulent layer which lines the
assages. But in some cases even the bronchial tubes within
ent a delicate tubular diphtheritic lining. Oertel quotes
ng pointed out that this is always confined to those tubes
ards or downwards in the back parts of the lungs, and never
which course forwards towards their anterior edges. These
intain that the existence of old pleuritic adhesions over any
g favours the penetration of the fibrinous exudation into the
ronchial tubes.

n practicable to employ the laryngoscope to determine the
lse membrane in the air-passages in a case of diphtheria, and
ctised observer would be able to obtain a satisfactory view
of the larynx, at least in a child. The diagnosis of laryngeal
mainly upon the fact that the entrance of air into the lungs
his is shown not only by the rapidity and gasping character
on, but also by the way in which at each breath the soft
clavicles and sternum are sucked in, as well as the lower

intercostal spaces, and in young children even the sternum and ribs, wh at an early age are soft and yielding. Every time that the patient inspi a loud crowing or croupy noise may be audible, and the cough is often h and brassy. In other words, the symptoms are as those of Croup, and so pathologists believe that croup, when attended with the development of me brane, is always an effect of the diphtheritic poison. The dyspnœa is apt become greatly aggravated from time to time, a circumstance probably d either to the supervention of spasm, or to the accidental impaction of portic of membranous or other secretion in the narrow chink of the glottis.

When diphtheria affects the larynx, extension of the disease to t part commonly occurs within from three to six days after the commenceme of the disease. Jenner, in 1861, had never known it delayed beyond t end of the first week; but Oertel says that it is not infrequent on the eigh or tenth day, and may be as late as the thirteenth. In Jenner's cases de always occurred in five days from the setting in of laryngeal symptom and he says that out of twenty-six fatal cases of Bretonneau's there w only five in which life was prolonged after the third day, and that th was but one in which it was prolonged after the sixth day, except as result of operative interference.

Were it not for the circumstance that the presence of false membra in the air-passages is in itself dangerous to life, from mechanical interfere with respiration, it is probable that many cases in which the disease assum this form would be of a mild type. In other words, one need not won that the laryngeal variety of diphtheria, even when it terminates fatally, often unattended with those symptoms of bodily prostration, anæmia, depression of the heart's action, which play so prominent a part in ma uncomplicated cases of the disease.

It is said that subpleural emphysema is of frequent occurrence fr distention and rupture of the pulmonary alveoli, and that the escaped may diffuse itself into the mediastinum and even into the subcutane textures of the neck and of the body generally. Bartels, in one case, he a loud sound on auscultation, which he supposed to be due to pericardi but which proved to depend upon the presence of a quantity of air in areolar tissue outside the heart.

In certain cases diphtheria seems to commence in the air-passag there being no primary affection of the fauces. In the epidemic form the disease, however, this is of rare occurrence. Bretonneau states that met with but two instances of it; of one of these he gives details (Case in his fourth 'Memoir'). The patient was an infant, a year old, in cha of a nurse at Tours, where no case of diphtheria had been seen for mont The nurse was a native of a hamlet some miles distant, where the dise was prevailing, and a nephew of hers had died of it a few days before t infant fell ill. Bretonneau puts the relative frequency of primary laryng diphtheria at one in thirty cases; Guersant at one in twenty cases. the epidemic which occurred at Auchtergaven (cf. p. 262), Dr Ye observed among one hundred and eighty-three cases, fifteen in wh laryngeal symptoms were present from the commencement, but in wh there was no visible affection of the fauces when they were first seen; a in six of these the pharynx remained free throughout the whole progres the disease.

In the reports made in 1859 by Greenhow and Sanderson to the Pri Council, in reference to the epidemic of diphtheria, which had been p

e or four years in various counties of England, extension to poken of as exceptional. Among a large number of cases lding in Lincolnshire, and recorded by Dr Capron, there are h it was mentioned. Out of twelve fatal cases that were seen , of Highgate, nine terminated by "asthenia," three only by Birmingham it is reported that croupy symptoms were com- Mr West had never seen any marked affection of the Heslop did not think that such complications occurred in r cent. of the cases. Only at Stalham and at Smallburgh is majority of patients died with croupy symptoms, or that such very common. So, again, at Crowle, Sanderson reported o means of determining in how many cases the symptoms of ication existed; it is certain, however, that they bore a small he fatal cases. At Launceston, on the other hand, out of t appears that eighteen were preceded by symptoms of croup." ns of French physicians as to the extreme frequency of such eprived of most of their value by the very fact that these gnise no membranous croup apart from diphtheria. Breton- rs' contain forty-five cases, related in detail, and in the m the air-passages were involved. But one must remember bject of this writer was to "establish the identity of croup angina," under the common name of diphtheria; and in one rding an ordinary instance of "pharyngeal diphtheritis," he number of special cases of this kind would present no interest." ement that diphtheria extending to the air-passages is the form of the disease requires allowance to be made for the al belief on his part. For he proceeds to say that this is diphtheria takes when sporadic, and also that which it ex- es in severe epidemics. Now, the former assertion involves that sporadic membranous croup is a manifestation of Fagge found in 1877, in putting together a series of cases d croup which had occurred at Guy's Hospital, that among as would commonly be regarded as examples of the former ere thirty-five in which the air-passages were affected. But the disease may have been a non-specific laryngitis.

.—The urine is not only scanty and high-coloured, as in other but it very commonly contains a considerable quantity of fact, which was first pointed out by Dr Wade, of Birming- of great value, as sometimes aiding in the diagnosis of cases rwise be obscure. There may also be epithelial and hyaline h more rarely, blood. Eberth is quoted as having found rience albuminuria was present in two cases out of three; ion seems to vary in different epidemics. In some patients ithin a day or two from the commencement of the disease; until convalescence already seems to be established. It is transitory, and may be detected only once or twice, even in the urine is repeatedly examined; but it more often lasts o. It is generally supposed to be devoid of prognostic signifi- tel, although he admits that the urine of some patients who ains very little albumen, yet says that he has been able to e relation between the quantity of this substance excreted in hours and the general intensity of the disease. From one

to three drachms was the amount usually passed in cases so severe a threaten life, or to terminate fatally; and the albuminuria persisted, those who recovered, for six or eight weeks after the subsidence of the di theritic affection of the throat.

When an opportunity is afforded of observing the state of the kidn they are said to be found large and congested, the epithelial cells be swollen, opaque, and granular, and filling the tubes. Minute extravasati of blood are also described as present in many cases; and, according to Oer masses of lymph-corpuscles often surround the capsules of the Malpighian tu The observations which have been made at Guy's Hospital would, howe suggest the belief that the affection of the kidneys differs widely in deg from scarlatinal nephritis, that observed in cases of diphtheria being comp tively of a slight character; and this accords with the well-known fact t dropsy very rarely occurs after diphtheria. A few instances of general anas have, indeed, been recorded by different observers—one, for example, Oertel in the 'Deutsches Archiv' for 1871. But when this writer speaks Ziemssen's 'Handbuch') of fifty fatal cases, mostly attended with suppres of urine and dropsy, as having occurred in Kiel and the neighbouring villa the doubt arises whether the epidemic was not really one of scarlet fe with diphtheritic complications.

It was maintained by MM. Bouchut and Labadie-Lagrave that *endocar* is of frequent occurrence in diphtheria. The author repeatedly searched such an affection in making autopsies in children who had died of the ease; but the valves have always appeared to be perfectly healthy. Sanné (who has had good opportunities of testing the value of the statem in question) declares that the slight irregularities that are commonly fo under normal conditions on the upper margins of the mitral and tricu valves have been mistaken for vegetations.

Paralysis.—Convalescence from diphtheria is sometimes attended b most remarkable sequela—*diphtherial paralysis.* This was observed by Spanish physicians in the seventeenth century. It commonly begins during second or third week after the subsidence of the throat affection, but is sai be sometimes postponed until the lapse of a month or six weeks. First, the palate is affected; it hangs flaccid, the uvula cannot be drawn up; its se bility is lost, so that neither pricking it nor applying the solid nitrate of si causes any pain. One consequence is that the patient speaks indistinctly "through his nose;" another, that when he attempts to swallow any liq part of it passes upwards and escapes through his nostrils. The pharyn muscles are occasionally involved: deglutition is then difficult, and som the food is apt to find its way into the air-passages. Next, generally afte interval of a few days, one or more of the ocular muscles may be attack the patient then sees double and squints; or paralysis of accommodation in, affecting both eyes, so that he is not able to distinguish near obj properly, and cannot read small print with comfort. The limbs are c monly affected somewhat later still, but sometimes they are the part which a loss of power is first observed. Sensations of numbness or pai the feet are complained of; and presently the legs grow weak and trem the gait is shuffling and uncertain, or the patient may be unable to st without support. The arms are much more rarely involved, but someti the patient cannot dress himself or hold anything in his fingers. He ca bend the feet on the ankles, nor extend the hands on the wrists.*

* According to Oertel, the electric sensibility and contractility of the affected mu

ances the bladder and rectum take part in the paralysis, en complete loss of sexual power in adults. Again, the larynx may be affected, in which case the vocal cords the laryngoscopic mirror to lie motionless in a position that of respiration and that of phonation—the position ys occupy in the dead body, but which is never seen under s during life. Wilks has insisted on this affection as a fre- ffocative dyspnœa, when a tracheal tube is removed, after n for a few days. The inspired current of air draws the inwards, until they meet and close the glottis. The eck and of the trunk may be affected, so that the patient is is head supported, or to raise his body from the recumbent turn over in bed. Lastly, the diaphragm or the inter- ay be paralysed, and lead to death by suffocation.

thology of this sequel of diphtheria there is still difference one extreme case Oertel says that he found extensive nal cord and its membranes; hæmorrhages surrounding the es, proliferation of nuclei in the grey matter, and fibrinous central canal. In another instance Buhl observed hæmor- nembranes and substance of the brain, and a red, swollen, the roots of the spinal nerves. See also Dr Percy Kidd's dico-Chirurgical Transactions' for 1884. The muscles have atrophied and in a state of fatty degeneration. But it is e that similar conditions are present in those cases which y in recovery. The fact that the palate is so commonly bviously favourable to the theory of a "*neuritis migrans*," a mation starting in the nerves of the part originally attacked, ong the fibres until it reaches the centres. The tenderness the affected nerves, the loss of knee-jerk, and anatomical Charcot, Vulpian, and Lépine in France, and by Leyden and any, have rendered probable the hypothesis of diphtheritic ing on peripheral neuritis (cf. Dr Buzzard's 'Harveian 85, p. 108). Senator declares that an abscess of the tonsil paralysis; and mumps is sometimes attended with a remark- of the vital functions, very like that which occurs after the other hand, some writers maintain that nervous sequelæ eria only as a member of the large group of specific fevers, s may arise after enterica, typhus, or smallpox. But this e. The only case in point that has ever come under our nat of a boy, aged five, who was admitted into Guy's Dr Wilks in the autumn of 1877 for a paralytic affection, nenced four months before, after a very mild attack of s unable to speak, and had difficulty in protruding his allowing. He could not stand, and his hands and forearms d.

The distinction between diphtheria and scarlatina, or non- is comparatively easy; the difficulty is to distinguish

d or entirely abolished, and their substance undergoes rapid wasting. denied the loss of electric contractility, and Buzzard agrees with him. action is certainly not constant. Atrophy is usually moderate, in some he muscles have a characteristic flabby feel. There is more or less ain. The knee-jerk is sometimes abolished.

between it and croup, or rather between "croup" as the result of laryn diphtheria, and "croup" from non-specific laryngitis. Both points come under consideration again in the chapters on Diseases of the Lar and on Local Affections of the Fauces.

Prognosis.—The result of diphtheria is always doubtful; no case, h ever mild, is to be regarded as free from peril. The average morta varies greatly in different epidemics, but according to Oertel it gener ranges between 30 and 40 per cent.

Age is the most important element of prognosis. The larger the portion of children among those who are attacked, the more fatal disease; for in adults it comparatively seldom assumes the fatal laryn form. However, according to Trousseau, the nasal variety is almost equ dangerous. It was observed by the Spanish physicians in the seventee century that "at its first coming it was most severe, but by degrees bec less violent—as is usual with other epidemical disorders" (Fothergill, 17

In faucial diphtheria the chief dangers are inability to swallow septicæmia; in laryngeal diphtheria, asphyxia before tracheotomy and a wards bronchitis, which more slowly but not less surely stifles the pati in both, failure of the heart.

In 70 consecutive cases of diphtheria under the writer's care (en December, 1889), the mortality was 15 in 17 under three years old, 1 33 between three and ten, 2 in 8 between ten and seventeen, and 2 in 12 in adults between twenty and fifty.

Of purely faucial cases only 5 out of 26 were fatal, of faucial laryngeal 24 out of 33, and of purely laryngeal all. In 39 cases trac tomy was performed, with 27 deaths.

Treatment.—The main indications in treatment are to maintain patient's strength, and to control the various tendencies to death. F the very first he should be strictly confined to bed. If the skin be and the pulse good, stimulants may for a time be withheld. But in m cases the heart soon begins to flag; brandy or port wine should the prescribed freely, and nourishing soups should be given in small quant at short intervals, night and day. Jenner mentions the case of a c three years old, who took from three to five ounces of brandy in twenty- hours with apparent advantage. The tincture of perchloride of iro supposed to be the best medicine. For some cases quinine, or bark ammonia, is preferred.*

As to the *local treatment* of the throat affection, there has been m tainty of opinion. All observers are now agreed that membranes upon tonsils and palate should never be forcibly removed. Bretonneau re mended that the diseased surface should be brushed over every day strong hydrochloric acid, or a mixture of one part of the acid to tw three of honey; and in 1861 Jenner expressed the opinion that powerfully corrosive agent, if once efficiently applied, would frequently the spread of the inflammation; but he advised that it should no

* Dr Hermann Weber has drawn attention to the fact that the practice, usu in England, of giving abundance of nourishment in diphtheria, is far from having ob the liability to the occurrence of the sudden collapse, which we have seen to be one o principal modes in which the disease proves fatal; but whatever may be said with r to this complication, I do not think there can be any doubt that the general tende such treatment must be good.—C. H. F.

must remember that mucous membranes with which the into contact show whitish patches for at least twenty- wards; for the writer has seen Bretonneau's practice con- day when no disease of the fauces was left, except what y the application. The white marks left by nitrate of e solid caustic or solutions in distilled water of gr. xx to so long. Liquor Ferri Perchl. Fortior has also been much ntly with local benefit.

years the practice of applying these powerful agents to the heria has been almost abandoned in all parts of Europe. come recognised that the local affection within reach is not rous part of the disease. At the present time disinfectants oyed—for the purpose of destroying the micrococci, by the parasitic theory of the disease; to lessen putrefaction surface of the affected parts, by those who reject that chlorine-water is said to be the most useful of such s accords with the results obtained experimentally by ed various disinfectants to liquids in which diphtheritic been repeatedly washed, and afterwards tested their up a putrefactive process in "Pasteur's fluid." The other h he found most effective were alcohol, solution of per- otash (gr. iss—gr. iiss ad ℥j), and solution of carbolic acid He recommends that one or other of them should be used or twice at least in every hour. Most English physicians ffering from diphtheria to suck small pieces of ice, which relief and helps deglutition.

plication on which Dr Squire lays stress is the weaker loride of iron; he adds a little glycerine to it, and paints ith a camel-hair brush two or three times a day. This acts rom the undiluted Liquor Ferri Fortior. He also recom- for a gargle, as first suggested by Küchenmeister, on account power of dissolving diphtheritic membranes. The editor has alkaloid of lecithin) locally in diphtheria of the fauces, fol- s made at Vienna :* it clears away the membrane without jury, and leaves a clear surface. Another preparation intro- hysiological laboratory is papain, from the Brazilian papaw- erful digestive agent dissolves false membrane with great have used it at Guy's Hospital, to that extent with success. ounds Oertel makes a great point of setting up a suppurative ace of the affected parts as early as possible; and he therefore vapour of boiling water should be inhaled, for a quarter of e, twice every hour, and would even reduce the patient's three or four hours, in order to keep up this treatment peaks highly of solutions of common salt and of chlorate alation. Siegel's spray apparatus is a convenient way of s to the throat, and is now much used in cases of diphtheria, ed. With very young children syringing the fauces with ime-water and milk, or with some antiseptic solution, is t efficient and least injurious procedure. Experience shows ground for the fear expressed by the elder Heberden

rkung des Neurins bei Diphtheritis," von Prof. E. Ludwig in Wien. d. Wissensch.,' 1877, No. 12).

that syringing the fauces would poison the patient by introducing pu matter into the stomach; for the danger is not in digestion, but in sorption of this matter unchanged. That admirable author concludes chapter in his 'Commentaries' which deals with the malignant sore-th (under which he probably included some cases of scarlatinal as wel of diphtheritic angina) as follows: "The gargle may be injected wi syringe into the throats of children, but this should by no means be d so often as to tease or fatigue them. Similar reasons would forbid us more strongly to take great pains in rubbing off the sloughs from t ulcers or in scarifying them. . . . . My only reason for suspec that I ought to lay more stress upon applications made immediately the throat than I have here done, is that several physicians of deserv great authority have judged them to be of more importance than they l appeared to me." *

When the disease has its seat in the *nasal cavities*, these parts shoul frequently cleansed with disinfectants (dilute solutions of the permanga of potash or of carbolic acid, lime-water, or brine), which may be inje with a syringe every two hours, or even oftener. If the patient is enough, Weber's douche should be used. This consists of a flexible t with a nozzle fitting tightly into one nostril, through which a strea fluid is made to run by syphon-action from a vessel at a higher l The patient is directed to incline his head forwards, and to bre quickly with his mouth widely opened. The soft palate is thus draw against the wall of the pharynx, so that the fluid, instead of escaping the fauces, passes round and runs out of the opposite nostril into a bas

For diphtheria affecting the *skin*, the local application of calomel is by Trousseau to be useful; we now use iodoform. On account of liability to development of false membrane wherever blisters are appl they should never be ordered in cases of diphtheria.

When diphtheria attacks the *larynx*, an emetic of ipecacuanha or of phate of zinc should be given, and if a good result is obtained it may repeated after an interval of some hours.†

In most cases of laryngeal diphtheria the question of *tracheotomy* to be considered. Even if the air-passages can be freed from the obst tion which immediately threatens the patient's life, there is always fear that the general symptoms may increase and prove fatal; and, m over, the edges of the incision may become coated with a diphthe layer, although the mere fact that the wound looks unhealthy, and beco coated with an ash-coloured layer, is no proof that it has been expose the action of the specific poison of diphtheria. Lastly, the disease m and unhappily often does, spread below the glottis and obstruct the trac itself or the bronchial tubes.

* 'Commentaries,' chap. vii. Compare the excellent remarks by Senator on the ca and mischievous attempt to apply local remedies to the fauces of young children in efficient manner.—'German Clinical Lectures,' 2nd Series, p. 447.

† Oertel recommends that a camel-hair pencil attached to a suitably curved handle sh be passed down into the larynx, and moved about so as to entangle and bring away any of false membrane that may be loose. This practice, he says, proved successful in t out of eight cases in which he employed it, the patients being respectively five, six, eleven years old. The membrane did not always come out with the instrument, but sometimes ejected afterwards by a short choking cough. He also advocates the inhalatio lime-water from a spray apparatus, but (as he remarks) very little can be expected f this procedure unless the patient spends nearly half his time over it, and most physic hold that this is too heavy a tax upon his strength.

tions for tracheotomy are rapid respiration, with laryngeal th deficient breath-sounds at the base of the lungs; pallor a purplish tinge of the lips; and sucking in with each inspi- ft parts at the root of neck and at the epigastrium, as well as os and ensiform cartilage. It is better not to delay the opera- ting has failed to relieve.

eotomy it is probably best to abstain from all treatment g the tube and removing membrane and muco-pus from the soft feather. Children under a year old generally die; the f the trachea, the yielding nature of the chest walls, and the eding them are the causes of this high mortality. When a o swallow, liquid food must be poured down one nostril is closed, not through a catheter, but by a funnel inserted . Nutrient enemata are rarely successful for more than a children. Brandy in frequent doses is of great service.

ied intubation instead of opening the trachea, but so far the t been encouraging.

ers.—After death from diphtheria, the lungs are found in *collapsed* in several lobules; and in all cases the tubes are and mucus. Ammonia and senega, sweetened with treacle u, is the best medicine, and brandy the best form of stimu- h to prevent this condition.

nger is *failure of the heart.* A boy of eight or nine under re went well through a severe attack of diphtheria, then plegia, and died suddenly from syncope; we found acute he heart, as after some cases of scarlet fever. Steel and digitalis are probably the best means of preventing this catas- ion to food and alcohol. Feebleness of the pulse and the first art resembling the second are indications of the danger.

tients may die from *septicæmia*, and this must be met by local ntiseptics, and by such remedies as quinine and alcohol.

is very rarely fatal; if so, it is from a branch of the external bly from the trunk of the internal carotid artery.

f sequelæ.—Diphtheritic paralysis is the most important of rally subsides spontaneously within three or four months, and h earlier. A case under Donders in which recovery did not ten months had elapsed is exceptional. Some years ago, how- e to Guy's Hospital as an out-patient who had been in our s previously for this affection; he was still unable to swallow fluids sometimes returned through his nose if he tried to This paralysis is very rarely fatal; but sometimes the patient ntly with slightly impaired power of certain muscles.

cated in cases of this kind, and often seems to be very useful. o the administration of nux vomica or strychnia, and he does that galvanism or faradisation should be employed, except at a

SYPHILIS*

"Consumptions sow
In hollow bones of man, strike their sharp shins,
And mar men's spurring. Crack the lawyer's voice,
That he may never more false title plead,
Nor sound his quillets shrilly: hoar the flamen,
That scolds against the quality of flesh,
And not believes himself: down with the nose,
Down with it flat, take the bridge quite away
Of him that his particular to foresee,
Smells from the general weal: make curled-pate ruffians bald."
Timon of Athens.

History of the disease—Its present extent—Nomenclature—Its place among specific exanthems—Origin and incubation—Primary lesion—Infecting and soft sores—Premonitory stage—Secondary lesions of skin, throat and mouth, eyes, &c.—Tertiary lesions of skin, tongue, palate, bones, and testes—Diagnosis—Conditions of infection—Special questions in syphilitic pathology—Prognosis and protection—Treatment.

Congenital syphilis—Its transmission to the child—Syphilitic placenta—Late manifestations—Its further transmission—Its treatment—Later effects.

THE disease which was first recognised about three hundred years ago and received the name of Syphilis, was then regarded as a pestilence—an epidemic disorder—spreading like the plague, or putrid fevers, by conditions of the air, and conveyed by infection at a distance. After a time it was discovered to be closely connected with foul local disorders of the genitals, and to be as a rule communicable by sexual congress. In the seventeenth and eighteenth centuries syphilis was generally known as the venereal disease (*lues venerea*), and was supposed to spring from any form of local disease acquired in impure connection. Its constitutional effects were until lately compared with those of Gout and of Tubercle, and thus syphilitic "diathesis" was placed side by side with assumed "arthritic," "scrofulous" and "malignant" diatheses. Inherited syphilis was unrecognised after infancy, or was confounded with what was called "scrofula;" and "scrofula" itself was often supposed to be the expression of a distant syphilitic taint. Owing to the labours of Ricord, Bassereau, Hutchinson and many other observers, the accidental connection of this disease with others of venereal origin no longer obscures its distinctive characters: it is completely separated from tuberculosis both in its acquired and hereditary forms, and it now resumes its original place among specific contagious diseases, as not always venereal in origin, and not more different from typhus, measles, or smallpox, than are diphtheria, erysipelas, and cholera.

* *Synonyms*—Lues venerea—Lues—Morbus gallicus—The pocks or great pox.—*Fr.* grosse vérole, *Germ.* Lustseuche.

either the writings of antiquity, nor those of the Middle descriptions of syphilis. What first drew attention to this epidemic prevalence in Italy at the end of the fifteenth Charles VIII of France invaded and occupied Naples. It sed to be a new malady; either generated by the filth of a trange wickedness of mercenary troops, or introduced from overed Western World by the sailors of Columbus, who pe about the time when it broke out. However, there are belief that the disease had been observed in France, in n Italy, as well as Spain, before the expedition of Charles), and when Columbus had not yet reached the port of Palos or at least had only recently landed his men there on his first voyage to the West Indies. Critical inquiries seem to probability syphilitic affections were by no means unknown s centuries, although they were confounded with other ularly with leprosy.

epidemic of 1494 and the subsequent years was unusually ancereaux supposes that almost a twentieth part of the e attacked, and although few died, fewer still were cured. on lost its pestilential character, and by the middle of the ry its type resembled that with which we are now familiar. , its diffusion was at first ascribed to climatic influences, in real mode of conveyance.

demics of syphilis have since been observed, which have entirely misunderstood, and described under a variety of ne affections, long regarded as peculiar to certain regions, tly been recognised as syphilitic. A disease which attacked d eighty persons at Brünn, in Moravia, in 1578, one which a in 1780, and the "Scherlievo" of Fiume at the beginning were almost certainly local epidemics of syphilis; and s was called "the Sibbens" in the West of Scotland, " in Norway.

ent time the disease is found in almost every part of the different degrees of frequency, according as the conditions r otherwise for its usual mode of propagation. Among the celand it is said never to have established itself, although it been introduced by sailors; according to Livingstone, in the blood in the centre of Southern Africa it is mild and effects; and in China and Japan it seems to be seldom eaux cites French physicians, who had studied it in these have suggested that its virulence has gradually become ts wide diffusion through the closely packed population nerations. Upon the same authority it is stated to acquire ate degree of intensity when it passes from a Chinese Similar assertions have been made with regard to its om one race to another elsewhere,—in the case, for in- British army in Portugal during the Peninsular war. Its ear in seaport towns, where vice and intemperance prevail

e term "Syphilis" was invented by Fracastorius, a learned rona, who in 1521 published a poem under that title, in d how *Syphilus*, a shepherd, was stricken by Apollo with

the new disease—even then not recognised as venereal.* It is a great pi
that we have not for every specific disorder a name as distinguishing,
short and flexible, and as free from meaning.

The French early contrasted *la petite* with *la grosse vérole* (i. e. *variol*
just as in England the word "smallpox" formerly conveyed a similar d
tinction.† Many other names, employed in various countries, indicated
belief that the disease was of foreign origin. Thus, while it was *mal*
Naples to the French, it was to the Italians *mal francese*;‡ and, unhappi
the Sandwich Islanders first knew it as the "English disease." At o
time the most common designation was *lues venerea*, which dates back
Fernelius (1556). But the venereal disorder was held to include both gon
rhœa, proved to be distinct by Ricord in 1831, and the soft chancre, wh
nearly all pathologists now regard as an independent affection. Inde
the conception of syphilis as a general malady, comparable with the ex
themata, had no existence until about forty years ago. It is true that Jo
Hunter described a constitutional form of the venereal disease, but
expressly taught that the action of the poison on the blood was differ
from that which occurred in any kind of fever. Since the modern vi
has been accepted the name of Syphilis has superseded most others,
Lues and its adjective "luetic" are still used abroad.

Pathology.—Like other results of purely scientific and speculative inqui
the recognition of the true nature of the disease has proved of the utm
practical importance. The physician must almost forget the local prim
disease and put aside the idea that the diagnosis of syphilis carries wit
the stigma of impurity. There are many ways in which a person may
a victim to syphilis without illicit intercourse; and in exceptional instan
one must be prepared to recognise its manifold varieties in patients
either sex, at any age, and in every position of life.

In the second edition of this book Syphilis was accordingly inclu
among the specific febrile diseases. It arises by contagion alone,
breeds true; inoculation is followed by a latent period of incubation;
onset is febrile, with an exanthem and a local lesion in the throat; lastly
has definite sequelæ, and protects against a second attack.

The contagium has not yet been certainly identified. Lustgarten
discovered that a bacillus, resembling those of tubercle, lupus and lepr
occurs in the cells of the diseased tissue.§

The chief peculiarities of Syphilis are the slightness of the pyrexia,
slow evolution of its stages, the importance and frequency of its sequela

* The *locus classicus* is as follows:— " . . . . primus
Syphilus ostendit turpes per corpus achores:
Insomnes primus noctes convulsaque membra
Sensit, et a primo traxit cognomina morbus
*Syphilidem*que ab eo labem dixere coloni."
—HIERON. FRACASTORII, Veronensis, Syphilidis sive de Morbo Gallico lib. iii, v, 2

† My learned friend, Dr Norman Moore, informs me that the term "pocks" freq
occurs in Irish and other mediæval MSS., and probably refers to cutaneous syphilis.

‡ It was called the French disease also in England:—
"News have I that my Nell is dead i' the spital
Of malady of France."—*K. Henry V*, v, 1.
The passage from 'Timon of Athens' (iv, 3), which stands at the head of this ch
shows how familiar the symptoms of secondary syphilis had become by the beginning o
seventeenth century.

§ 'Med. Jahrb. der k. k. Ges. der Aerzte,' 1885. See also 'Brit. Med. Journ.,' Oct.
of the same year, p. 757.

mission, and, we may add, its reaction to remedies. As
g hereditary, Mr Hutchinson well argues, that smallpox and
e, like syphilis, occasionally transmitted from mother to
revity and severity of these and other specific fevers make
ible for them to be imparted by inheritance.

disease.—Incubation—When a person is affected with the
here elapses, in uncomplicated cases, a considerable interval
ny symptom is observed. This is the period of incubation.
kable that this fact was unknown until, between 1856 and
xperimenters inoculated syphilis upon healthy individuals.
ler, in 'Ziemssen's Handbuch,' brings together thirty-one
his kind, in the very large majority of which the incubation
n to twenty-five days. Once it was only ten days; four
irty-five and forty-four days. In 1865 Fournier recorded
in which no treatment was adopted, so that the disorder
naturally. The incubation was more often over than under
not infrequently reached a month or six weeks, and once
o ten weeks. In one patient of Bäumler's, in whom the
posure to the poison was known, the incubation was twenty-
other twenty-nine days. Frequently incubation is prolonged
more. The idea that no such period occurred was based
atural tendency to ascribe the disease to the last impure
ding its appearance, partly upon the circumstance that
at of the soft chancre), which produces an effect almost at
nsmitted in association with that of syphilis proper, and
recognised as distinct.

.—The earliest symptom of syphilis is manifested at the
it is spoken of as the *primary* lesion, while the most remote
appear elsewhere are termed *secondary*. It is often called
ian "chancre," but upon the skin its typical character is that
d papule. This, which is at first very small, soon increases
grows larger becomes indurated, so that to the touch it
ce of cartilage let into the part. After a week or ten
quamate slightly; or a little moisture may ooze from its
ently dry up into a thin scab; or it may continue to look
zed; or, lastly, it may become excoriated, and slightly
centre. Upon a mucous membrane, the primary affection
s a very small itching vesicle with a reddened base, which
ning an erosion, and afterwards a shallow ulcer; this, too,
rated floor as it enlarges.

of these lesions has been investigated by several observers.
d an abundant infiltration of nucleated cells, not only
dles of connective tissue in the cutis or mucous membrane,
dventitia of the blood-vessels, which are thereby narrowed.
s that there is an actual development of connective-tissue
he periphery of the indurated mass, and that this is the
ness, which by Auspitz had been attributed to the presence
adation between the cells and the spaces in which they lie.
f the primary lesion of syphilis varies in different cases.
ses quickly away, leaving no trace of its presence; but when
it generally takes many months in subsiding, and upon the

skin its site remains marked by a brown pigmented patch, with more or l superficial scarring in its centre. On the other hand, mucous membra never show pigmentation, and all that is left by even the largest papule i little vascularity and looseness of texture. Mr Hutchinson has dra attention to the fact that in some individuals an induration, like cartil appears again and again during a period of several years exactly whe former primary syphilitic lesion was situated, without any fresh infecti Ultimately even the pigment disappears, and it may then be quite impos to discover that the patient has ever suffered from primary syphilis.

Hard and soft sores.—The typical primary syphilitic lesion is still o called a Hunterian or indurated chancre; but if (as is often the case) it the characters of a chancre, or "sore," they are accidentally present ra than essential. Bäumler calls the primary lesion of syphilis, not a chan but an "ulcerating sclerosis."

In 1852 Bassereau took the pains to trace to their origin in the oppo sex a number of venereal cases; and this method of "confrontation" (a terms it) showed that, whereas sores which were followed by second symptoms had been derived from persons who themselves suffered un similar effects, other sores which remained simply local, or which at m were attended with suppurating buboes, came from individuals in whom disease had likewise failed to produce any constitutional effects. His vi were soon afterwards adopted by Ricord, who indicated a number of dist tions between the two kinds of lesions in question, of which one bec known as the "indurated," "Hunterian," or "infecting" chancre, and other as the "soft" or "non-infecting" chancre. His account of the infec chancre need not be quoted in this place, except to mention the import fact, which was now brought out for the first time: that the ordin slight secretion of such a sore cannot be inoculated upon the pati nor upon anyone else who has already had syphilis. Bidenkap indeed, since found that there are occasional exceptions, it being so times "auto-inoculable" during an early stage of the disease, when th have as yet been no constitutional symptoms. But such observations dently do not affect the principle on which the rule is explained, since t are strictly parallel with the fact that vaccination can be successfully formed before the eruption of smallpox appears, *i. e.* before the infection become generalised throughout the body.

Ricord's description of the non-infecting chancre was that it beg (without any incubation) in a pustule, which in two or three days brea that there is then formed a deep, punched-out ulcer, with irregular slightly undermined edges, a grey surface, and a soft, or at least not indurated, base; that it secretes pus freely, and that this is inoculable ag and again upon the same individual, and also upon all other persons, whet affected with syphilis or not. One consequence of the difference in inoculability of the two affections is that whereas there is seldom more t one indurated chancre, several soft chancres are often seen together. soft chancre is scarcely ever seen except on the genital organs. T histology of the soft sore is not so different as might have been expec from that of the primary lesion of syphilis. There is a similar infiltrat of cells into all the tissues, including the walls of the blood-vessels, but it said that the channels of these vessels are dilated, instead of being narrow The cicatrices left by non-infecting chancres are often irregular in form, much more conspicuous than that which follows a typical Hunterian sore

not appear to be any doubt as to the correctness of Basse-
ons, nor as to the general validity of Ricord's distinctions
kinds of venereal sore. But subsequent experience has shown
al application of those distinctions in the prognosis of such
le to certain sources of error. And the theoretical question
der discussion, whether the poison of the soft chancre is or
gin independent of syphilis. It is hardly necessary now to
nfusion which at first prevailed in consequence of the way
l and those who followed him formulated their doctrines.
king of the "duality" or "unity" of the chancre, they in-
uality of the syphilitic virus. Thus they drove from their camp
egarded syphilis as a single specific disease, comparable with
fevers, while yet there was no place for the dissentients
opponents, who advocated the *unity* of the local soft sore
is followed by constitutional symptoms. This, however is
han a verbal difficulty.

onsiderations bearing upon the theoretical question as to the
ontagious principle of the soft chancre are the following :—
net with a case in which an indurated chancre on the scrotum,
ondary symptoms, had inoculated itself upon the skin of the
h it came into contact, producing there a sore which remained
ation. Another patient under his observation had an indu-
he grey surface of which suggested that its secretion would
the experiment was tried on the patient himself, and the
t sore which reached the size of a franc-piece. It is evident
are easily explicable on Rollet's theory, that "mixed sores,"
the virus of syphilis and the contagion of the soft chancre,
non. But the view which Clerc adopted was that the soft
ing else than the product of inoculation from an infecting
person who is suffering or has suffered from constitutional
as if there were not already sufficient confusion, he proposed
se of the term chancre to the indurated primary lesion of
call the soft sore a "chancroid." Shortly afterwards Mr
wed that an indurated sore, when irritated by savine oint-
red savine, could be made to pour out a purulent fluid which,
l upon the bearer or upon other patients, produced, without
a sore having the characters of a soft sore, and capable of
d again and again. And lastly, Pick, of Prague, found that
e of scabies, or pemphigus, or acne, was also capable when
syphilitic subjects, of generating a similar soft sore, although
t healthy individuals in the same way. From these experi-
ypothesis derives far more support than from his own observa-
he alternative theory of Rollet can be altogether set aside.
ne case that the sores obtained by Lee and Pick are identical
res, one may be justified in concluding that such chancres are
-product of syphilis, and that their contagious principle is
vo in persons suffering under that disease. There is reason
in mucous patches (or flat condylomata), themselves due
re often arises a virus which is capable of transmitting them,
of all other symptoms of syphilis, to healthy individuals.
irly comparable with those which seem to show that in various
f the human body animal poisons may be developed which

may set up erysipelas, or even perhaps diphtheria, in other patients ; bu we, with Mr Hutchinson, speak of soft chancres as "abortive inoculatio the difficulty remains of explaining how their contagion possesses a virule of a certain kind which is wanting in the secretion of a true infect chancre. That it is not a mere question of the presence or absence pus is shown by Ricord's observation, that the matter from the inte of a suppurating gland produces a soft sore when inoculated, wher this is not the case with the matter which is formed in the connect tissue outside it.

From a practical point of view, however, the origin of the soft chan is of minor importance in comparison with the question whether it conta the virus of syphilis : that is, whether it remains local and can only pro gate itself, or whether it is liable to be followed by constitutional sympto In principle the first of these statements appears to be correct, but unfor nately it is open to exceptions, which interfere with its application in prognosis of individual cases. If we accept the theory of "mixed chancr promulgated by Rollet, of Lyons, in 1858, it follows that, if a perso infected at the same time with the poisons of the soft chancre and syphilis the necessary result must be the immediate production of w appears to be nothing but a soft chancre, which will not assume an indura character until the lapse of three or four weeks, corresponding with incubation of syphilis. Such sores are believed to constitute an act majority of the primary syphilitic lesions which are seen in the hospitals large cities. It is, however, a question whether the rapid spreading of base and sides of a soft sore, which sometimes occurs, may not lead to destruction of any syphilitic poison that may be present in it, and thus s the patient from further ill-effects. In this way it may be possible account for an occasional failure in the transmission of syphilis from a per suffering under that disease to one who has never had it, notwithstandi the occurrence of a chancre in the latter. Bäumler states that there only seven experiments on record in which the artificial inoculation unquestionably syphilitic secretions upon healthy persons has not commu cated the constitutional affection. On the other hand, it not unfreque happens that a male patient derives syphilis from a woman in whom a sore is the only discoverable primary lesion ; and the best observers h been obliged to allow that they have seen instances in which a sore t has at no period been indurated was followed in the same individual secondary symptoms. Mr Morgan, of Dublin, has especially insisted the fact that in the Lock Hospital of that city he scarcely ever saw indura chancres in the very class of women from whom the men who come to institution derived their venereal disease. But this proves less than mi at first sight appear, now that we know that the secretions of moist second eruptions contain the specific virus : and Mr Cooper Forster stated t at Guy's Hospital he generally succeeded in finding a characteristic prim affection in female patients when he carefully looked for it.*

* All difficulties in the matter are sometimes explained away by saying that the ind tion is occasionally very slight and parchment-like (*parcheminée*), so that it can be dete only by taking the sore between the finger and thumb in a particular manner, whic impracticable in the case of a female with a sore situated within the vaginal orifice. this is in reality equivalent to the admission that the primary lesion of syphilis does invariably present the distinctive characters which are assigned to it. Conversely, typical induration is sometimes closely simulated by inflammatory thickening of the flo a soft chancre as the result of irritation by caustics.

practice, although it is easy to say that certain "hard" sores are sure to be followed by secondary symptoms, it is seldom, if ever, e to assert that any sore, however "soft," will *not* be so followed. It consequently be unadvisable to attempt to limit the use of the term cre" to sores from which the syphilitic virus is absent, as has been ed by some writers, including Mr Forster. We had much better keep original meaning, and apply it to all *ulcers*, whether syphilitic or not, are directly due to venereal infection.

nstitutional symptoms of syphilis are sometimes seen in persons in no primary affection is known to have occurred. One source of in these cases is that a primary lesion sometimes assumes the appear- a secondary one. Thus, a parchment-like glazed papule on the glans nay be undistinguishable amid a general papular eruption, while on bium the affection may be so modified as to simulate a "broad oma" or "mucous tubercle." In other instances, perhaps, it is so picuous that the patient never notices its presence, and it quickly es, leaving no mark. But some observers admit cases of true "*syphilis* ," *i. e.* the invasion of the general malady without a local lesion.

-exanthemic period.—The primary, or latent, stage of syphilis is cha- sed by swelling and induration of those *lymphatic glands* which bear an ical relation to the seat of the primary chancre or papule. So constant symptom that Fournier failed to detect it in only three out of 265 cases , and in the same number among 223 cases in women. The glands correspond with the genital organs are, of course, those in the groins. lurated sore upon the finger leads to enlargement of glands in the or at the bend of the elbow, particularly of the small lymph-gland lies just above the internal condyle. One upon the lip affects those ne lower jaw. These "amygdaloid" glands reach the size of almonds, y be a little more, but they never approach the dimensions of the ating bubo which accompanies a soft chancre. They feel hard, ely moveable, and are seldom tender or painful. There is generally ldening of the skin over them, and they scarcely ever suppurate. mes along the dorsum of the penis an inflamed lymphatic vessel can like a cord.

is "indolent bubo" of syphilis, as it is termed, appears a few days han the primary lesion. A chain of glands is commonly affected, ten those in both groins at the same time. The swelling runs an ingly slow course, so that it often does not begin to subside until six s have elapsed. Thus it may be very useful in diagnosis, by indicat- e probable seat of a chancre or papule, the marks of which might ise have been overlooked. One must, however, remember that is is not the only cause of a similar affection of the inguinal glands; oper Forster insisted on the frequency of the occurrence in rowing nd it may also be seen in young men accustomed to much walking. s in other regions are liable to become enlarged apart from syphilis, and gh the firm, painless, separate bullet-glands are usually characteristic, ay occasionally feel something very similar in cases of irritative ement, and in Hodgkin's disease.

ne appearance of the primary papule or chancre of syphilis is followed interval during which we may suppose that the virus is undergoing lication in it and in the adjacent glands. The lesion itself now and ubsides in the meantime, and so the period is sometimes spoken of as

a second incubation, or latent period. Its length is generally s
seven weeks; but it is liable to wide variations, as is shown by the
that where the disease has been inoculated experimentally it has
found to range from twelve days to twenty weeks. Probably it is sh
in persons who are weakly, or whose habits are dissolute or in
perate, than in those whose general health is good. During its co
some patients look and feel well; but others—it is said, particul
women—complain of malaise and depression, and have a pale and hag
aspect.

Secondary stage.—The general or *constitutional* symptoms of syp
begin differently in different cases. In some there is well-marked fe
the temperature rising suddenly, or gradually, until within a few day
may reach 104°. With the appearance of an eruption it sometimes decl
but it may run on for several weeks, assuming, as Bäumler has shown
intermittent type resembling that due to malaria. The proportion of c
attended with febrile disturbance is stated by Güntz at 20 per cent.;
Bäumler thinks that it is really higher.

A more frequently noticed and very characteristic symptom of
period is what French writers term *bitemporal neuralgia;* the occurrenc
more or less violent pains, which shoot upwards along each temple. T
are not felt at all periods of the day, but come on, often with surpri
regularity, in the evening or at night. A similar tendency to noctu
exacerbations, indeed, belongs to all other painful syphilitic affecti
Ricord used to ascribe it to the warmth of the bed, and said that in per
whose occupations compelled them to sleep during the day, the tim
which the pains returned was reversed. But, whether or not this is
fact, there can be no doubt that his explanation of it is incorrect. For
hour at which the bitemporal neuralgia sets in is often early in the even
while the patient is still up; and Bäumler is probably right in thinl
that it coincides with an increase of fever, and is in some way depend
upon augmented vascular excitement. Pains in the back and limbs ma
present, and there may even be swelling of the joints. The writer has se
distinct, though slight and painless, enlargement of some of the artic
tions of the fingers; and Bäumler speaks of patients seeking advice for
and swelling of the metacarpo-phalangeal joint of the thumb, but leavin
to the physician to discover that they have syphilis.

Syphilis of the skin.—Unlike all other specific poisons, the virus of syp
produces not merely one or two definite cutaneous affections, but an imm
variety. They are known as *syphilides* or collectively as syphiloderma (n
properly syphilodermia); they shade off into one another so as almos
defy description, and are classified with the greatest difficulty. W
regard to the causes which lead to the evolution of one of these erupti
rather than another, all that we at present know is that a patient who i
a bad state of health is most likely to have those forms which suppu
and ulcerate. The differences between them are by no means attribut
to tendencies towards particular non-syphilitic cutaneous diseases;
instance, a person liable to ordinary psoriasis is not specially apt to
affected with a squamous syphilide, nor one who has lichen with a pap
one. We should, therefore, avoid using such names as "syphilitic lich
"syphilitic ecthyma," or "syphilitic psoriasis." However difficult
diagnosis may occasionally be in individual cases, syphilitic affections

y distinct from the non-syphilitic eruptions which may simulate them, hology, in prognosis, and in treatment; and therefore, since a name hould indicate a diagnosis, the terms psoriasis and roseola alone become , if liable to be contradicted by the far more important qualification litic." A non-syphilitic disease, as psoriasis, is not more "modified" hilis, than is scabies or a typhus rash; any of the three may be seen incidence accompanying cutaneous syphilis, but unaffected by it.

ere are certain features which belong, more or less, to all the des. (1) One of them is a peculiar colour, which is commonly said to le raw ham, or to be "coppery:" according to Bäumler, the former rison has been traced to Fallopius, who wrote about three hundred go; the latter only to Swediaur, at the commencement of the present y. The cause of the tint is probably a chemical change in disintegrat- od-discs which have been extravasated into the tissues, and therefore t present at first; while, on the other hand, a very similar appearance times displayed by non-specific eruptions, of chronic course, especially legs, where the venous circulation is apt to be embarrassed. (2) A character of syphilitic cutaneous affections is their multiplicity or norphism" in the same patient and at the same time. Sometimes s, papules, pustules, scaly patches, are so intermingled that we cannot ich of them preponderates. (3) A third point, which is, however, not r to the syphilides, is their tendency to arrange themselves in circles or cles, or less completely annular forms. (4) Unless their development rapid, they are seldom attended with itching; in many cases the experiences nothing whatever to draw his attention to the skin. (5) rliest rash, or any universal syphilitic eruption, may be symmetrical, the exanthem of measles or smallpox is symmetrical, because it affects nmetrical human body. But syphilides do not show the characteristic try of psoriasis, eczema, and some other cutaneous diseases, in picking rresponding parts of the trunk or limbs. They are irregular in distri- . Their favourite localities are the forehead, the soles of the feet, and ms of the hands, and they often affect regions rarely visited by other ous affections.

important distinction has been drawn between the earlier and more cial syphilides, and those which are later in appearance, and affect the t layers of the skin. The former, to which some writers limit the *econdary*, may relapse several times, but their first appearance is very postponed beyond twelve months from the time of infection. The which are often called *tertiary*, seldom appear within the first year, ey may break out for the first time after an interval of many years. listinction of time is not, however, constant. "Tertiary" lesions e occasionally seen very early, but the more superficial secondary des seldom appear very late, and probably never after tertiary forms ecome developed.

thologically, the roseolous and other secondary eruptions may be com- to the exanthems of variola and typhus, while the tertiary syphilides less complete counterpart in the suppurations which occur as sequelæ asles and enteric fever, or perhaps they may be more justly compared secondary growths which follow a malignant tumour.

art from the period of their development, secondary syphilides are mperfectly symmetrical; tertiary almost always avoid even an approach teral symmetry. The secondary rashes commonly consist of numerous

isolated spots or patches. They seldom affect the hands or feet, are paratively superficial, have little tendency to ulcerate, and leave no cica behind them. On the other hand, the so-called tertiary dermatos syphilis consist of comparatively few separate lesions, but they gen run together; they affect the deeper layers of the integument; they de the tissues, and are followed by scars. These distinctions are not abs nor do they apply equally to every form of syphilide belonging to the or to the late group, but the exceptions are few.

The special character of the several syphilides will be most usefull cussed along with other diseases of the skin from which it is of the u importance to distinguish them, but the following brief account has re to them as parts of the pathological evolution of the general specific di

1. *The macular or exanthemic syphilide* ("syphilitic roseola") i earliest and most constant of all, and answers to the exanthem of m and scarlatina, or perhaps more closely still to the occasional early rose of smallpox. It consists of rather ill-defined, pale or dark, rose or "cop coloured spots, irregular in form, of small size, or approaching that threepenny piece, or even larger; scarcely if at all raised above the su and generally disappearing under pressure.

This is most constantly seen upon the chest and abdomen, but some it covers most of the trunk, and appears on the neck and face; o limbs it is less common, and it avoids the hands and feet. As a ru takes a week or more in coming fully out, but sometimes it is develop rapidly that the case may be mistaken for one of measles. A pale scanty eruption often fades within a fortnight. One which is dark col and abundant may remain visible for several weeks. It may then assu papular form or disappear with slight desquamation even when no ment has been adopted.

2. *The papular syphilide* ("lenticular syphilitic lichen") is the common form to follow the initial rash. It consists of red shining tions often of a marked coppery tinge. In size they vary consider some being scarcely bigger than millet seeds, others as large as peas develop themselves very rapidly, reaching their full magnitude in a few They do not, however, generally all come out at once, but rather in succe crops over a long period.

The eruption is sometimes scattered irregularly over the whole b sometimes the papules are grouped together in clusters. They are times most numerous on the neck and forehead; sometimes they are th crowded in the naso-labial grooves, or at the angles of the mouth. generally remain for some weeks and then subside, leaving dark st but not unfrequently they desquamate, so as to pass into a squa syphilide, or their summits soften down and become covered with y or brown crusts.

In consequence of the thickness of the cuticle of the palms and these parts show, not raised papules, but flat round horny plates, each a reddish-brown border. After a time the plates become detached, for so many little ulcers, or the adjacent surface becomes rough and and cracks or fissures may be formed. Thus a complicated affe arises, constituting what used to be called "syphilitic palmar and pl psoriasis." This form is the most constantly symmetrical of all syphilides.

3. *Mucous patches.*—Another modification of the papular syphilide is

rts of the skin which are thin, and habitually in contact with other and also upon mucous membranes. The lesion is known as a ous tubercle" or patch—"*plaque muqueuse*,"—a 'flat, broad, or moist *loma latum*," so called to distinguish it from the "pointed condy- (*C. acuminatum*) or "wart" of the genital organs, which is often yphilitic in origin.

consists of one or more broad raised patches, from the size of a to a half-crown, with a sharply defined edge, and a surface which is imes dry and warty-looking, but usually moist and coated with a dirty secretion, of a peculiarly nauseous, pungent odour.

ucous patches occur, sometimes in large numbers, about the genitalia, lso along the perinæum and round the anus, in the fold of the in the groins, at the umbilicus, at the folds of the axillæ, beneath easts, in the neck, between the toes, at the angles of the mouth, and here. Not infrequently they are so placed upon opposed parts of the s strongly to suggest the idea that they spread by local infection. If s the case it is one of great theoretical interest, since a patient who y has syphilis is believed to be absolutely protected from the further nce of the virus, so that flat condylomata would possess an independ- ontagion peculiar to themselves. They may certainly be the means ich syphilis is transmitted to other persons. In such cases they gene- give rise to a typical primary indurated papule, which is followed after ual interval by constitutional symptoms; but it is equally certain that he cases the affection which they set up in non-syphilitic individuals is inguishable from a flat condyloma, and cannot be proved to contain philitic virus. This is admitted by Kaposi, but he passes it lightly by he remark that secondary symptoms sometimes fail to appear, even an indurated primary sore. Yet so common is the occurrence of flat lomata, apart from all other indications of syphilis, that careful ers have in some cases attributed them to mere irritation of the e by dirt and moist secretions. Moreover, in some countries they been known to prevail endemically in such a way that their connection yphilis has been altogether a matter of inference. These considera- seem to point to the conclusion that, although the affection owes its to syphilis, it yet acquires a contagious principle of its own, which is le of surviving and reproducing itself by contagion after the original itic virus has in some way disappeared or become exhausted; if so, ends strongly to corroborate the doctrine that the soft chancre arises preads in a similar manner.

The squamous syphilide ("syphilitic lepra or psoriasis").—Contrary to once was taught, this, as an independent eruption, is infrequent. We seen that the macules and papules of syphilis often after a time he scaly. If such spots go on spreading still further, there arises ffection which may resemble psoriasis; but it is distinguished by nall and scanty scales, the copper-like tint of the patches, and the ce of the definite and symmetrical distribution so characteristic of sis.

The pustular syphilide ("syphilitic ecthyma and rupia").—This is a collection of several different varieties of eruption than a single well- cterised form. It consists of pustules of all sizes, each of which is ally seated upon a firm red base. They are sometimes present in nse numbers, especially upon the face and trunk. They come out

rapidly and sometimes with febrile disturbance, but in successive c which may be prolonged over many weeks; and they may relapse after a year has passed. They dry up into brown, or dark green, or b scabs, and leave large stains, which ultimately pass into shallow, flat, w cicatrices.

Concomitant early affections.—One of the points in which syphilis resem some of the acute exanthemata is its strong tendency to affect the *fauc* well as the skin. Indeed, the throat sometimes suffers before any cutan eruption can be discovered; and in other instances an inspection c reveals morbid changes, which had caused neither pain nor discomfor that the patient had no suspicion of their presence. The acute angin early syphilis appears as a diffused redness, and not in the form of dis maculæ, contrasting in this respect with the sore-throat of measles. T is often much swelling, particularly of the tonsils. The follicles bec enlarged and prominent; or they may rupture, and cause shallow excoriat In more chronic cases flat, greyish mucous patches are seen on the tonsi the palatine arches; or there may be yellow ulcers, with sharply defined borders. The most curious affection is one which is seen not only upon fauces, but also upon the hard palate, the inside of the cheeks, and the It consists of scattered milk-white spots, which have been termed *pl opalines*, and which are perhaps best compared with those caused by ap ing nitrate of silver to a mucous membrane. Their shape may be ro oval, or indefinite; they vary in size, and may run together so as to cov large surface with an irregular pattern. Sometimes they are slig puckered, and parts of their surface may be reddened, with only a l white opacity here and there. All their varieties are deserving of ca study, for they are very characteristic of syphilis. They run an exceedi slow course, and may break out again and again, not only during the e stage, but also long afterwards.

In many cases the *eyes* suffer. The affection most commonly observ an iritis, attended with the formation of yellowish-red nodules near edge of the pupil. This is generally bilateral. In Hutchinson's experi it arises within the first six months of the disease, if at all. It is gener associated with one of the more severe forms of eruption. Later attack said to be always relapses; they are often limited to one eye at a time. retinitis may occur, in which case the fundus of the eye has a hazy app ance when seen with the ophthalmoscope; the disc is reddened and swo and its margin is indistinct; there may be many small extravasations of bl The recognition of this marked change is very important, because of insidiousness; Hutchinson speaks of the patient as noticing "nothing ex that his sight is very dim; he has no pain, no congestion of the front of eye, no intolerance of light."

The *lymph-glands* in various parts of the body become swollen at stage of syphilis; we are especially accustomed to look for enlargemen those which lie one above each elbow, and of those that are seated at back of the neck.

Another symptom is partial *alopecia;* the hair may come away with comb so freely that the patient fears becoming prematurely bald. The s hairs from the limbs are shed, as well as those of the scalp.

The cerebral arteries are sometimes diseased at an early date, the more often in the tertiary stage, and, according to Fournier, the same ap to diseases of the cord and nerves.

ory *albuminuria* is not uncommon. Dr Fagge had one patient whose as albuminous at a time when it could hardly be supposed that the had already become lardaceous; and he mentioned two cases of jaun-e of which quickly subsided, but the other ended in acute atrophy of r (cf. Dr Wilks's case, 'Path. Trans.,' 1867, vols. viii and xvii).

e present writer has for some years had a patient under observation the subject of secondary syphilis, and passes albumen in his urine any other sign of renal disease.

r syphilides.—After the lapse of the six months, or the year, or two luring which one or more of the early syphilides has developed itself oftener, the disease often becomes altogether latent. In many cases run its course, and the patient henceforth remains perfectly free. other instances he continues to be troubled, at varying intervals, with manifestations of the disease. Perhaps small scattered pustules on the scalp, which are scratched by the comb, and, scabbing over nd again, cause him great annoyance; perhaps the palm of one w and then becomes scaly and fissured in its centre; perhaps some ails grow rough and thick and discoloured. Affections such as these on for a long time.

in some cases, possibly many months or even years after the subsi-f the early syphilides, there appears one of the late or *tertiary* es, with the characters described above.

1869 a woman was in Guy's Hospital who had been infected by her l twenty years previously, and in whom the disease assumed the form reddish-brown glazed patches, covering the greater part of the face; re not at all raised, and there was only the slightest possible desqua-; in fact, they were scarcely more than maculæ. In other cases, hite scales appear around a red ring or festooned line. Or, again, atches of skin may become thickened, raised, of a reddish-brown and rough with a bran-like scurf. But the most common of the uptions of syphilis are lurid reddish-brown nodules, like those es seen in the early papular syphilide, except that, instead of attered, they now cohere in rings or patches, so as to cover a large or to form straggling festooned lines. Another peculiarity of such lules is that they leave cicatrices even when they have not ulcerated. most instances they either become covered over with crusts or eaten as to form small deep ulcers with vertical edges. It is no uncommon o see patches a foot or more in diameter, some parts of which have cicatrised, while others show recent nodules, or serpiginous lines of preading over the healthy skin around them. All these varieties are of the most patient clinical study, for they are absolutely characteristic ilis, and the patient has often no suspicion of their nature. They have al tendency to affect the face (particularly the forehead, nose, and e nape of the neck, the shoulders, the back, and the extensor surfaces imbs.*

r lesions of mucous membranes.—The tertiary period of the disease is

an illustration of the importance of the correct diagnosis of such affections I may the case of a gentleman aged fifty-five, who came to me for the most obviously ulcerating patches, one on each shoulder, which had been steadily getting worse ar. When a young man he had some venereal complaint, attended with swelling stis. He had been married for many years; his wife had had three miscarriages, hildren. Iodide of potassium cured one of the patches in a fortnight, and the hin six weeks.—C. H. F.

also characterised by lesions of the throat and mouth, most of which d from those of the secondary stage. *Plaques opalines* may, indeed, cont to form on the cheeks or lips; but the surface of the tongue now for first time becomes affected in a variety of ways. Sometimes more or extensive patches appear unnaturally smooth and glossy. Sometimes t arise oval greyish ulcers, especially on its centre or edges. Sometime mucous membrane becomes greatly thickened, with deep grooves intersec it in all directions. All these changes render the contact of salt and pe exceedingly painful to the patient, and make him glad to confine himse the blandest possible diet.

In other cases, again, the soft *palate* becomes affected with deep sh cut ulcers, which very rapidly perforate it, and eat away a considerable of its substance. Such ulcers, when they heal, leave well-marked cicatr Some years ago a woman died in Guy's Hospital whose velum had before been extensively destroyed on one side, so that the uvula was hel its place by two thread-like processes of mucous membrane, which looke if they could not possibly have escaped being torn through during deg tion: the preparation is now in our museum. Sometimes the palate become adherent to the pharynx, so as to cut off the communication betw the cavity of the nose and the air passages below.

*Concomitant tertiary lesions and nodes.**—Some writers characterise tertiary period as the "gummatous stage" of the disease. In the su taneous tissue, or in the substance of the tongue, such tumours often acq a very considerable size; and the skin or the mucous membrane over t may at length become ulcerated through, so as to expose a grey degener mass, of the most typical kind. But we cannot call tertiary eruptions g matous, to the exclusion of the earlier syphilides. The scattered lenti papules which may constitute one of the first manifestations of cons tional syphilis are not merely inflammatory; and recent observers h shown that the nodules which are found in iritis are small gummata, mere fibrinous exudation. Again, Hutchinson has mentioned a cas which definite gummata were found in both testes and also in the sp although a secondary rash was still out on the skin of the patient, died of "syphilitic disease of the heart—myocarditis with a gumma."

The gummatous affections of internal organs are by far the m important of the effects of syphilis, since they produce various sympto and not rarely destroy life. They will be described among the local dise of the meninges, larynx, lungs, liver, &c. It is sufficient here to urge importance of carefully examining the bones and the testes whenev syphilitic taint is suspected.

It is believed that those *bones* which, like the skull-cap, clavicles, u and tibiæ, are but thinly covered with soft parts are more than others li to be affected. By running one finger along the surface of these bones i easy to discover whether there is any swelling in them, or whether the pat shrinks from pressure. The enlargement caused by syphilitic periost generally rises gradually from the surrounding surface, but in one inst

* The terms *gumma* and *gummositas* are of considerable antiquity. Fallopius, in sixteenth century, spoke of tumours of bones as having been called "*gummata gallica*," account of their containing a matter resembling *gummi eliquatum*. Ernst Wagner posed to substitute the name "syphiloma" for gumma; but the change is not worth mak "Tubercles" is rightly abandoned, for *tubercula syphilitica* have nothing to do tubercle in the modern sense of the term, although v. Bärensprung supposed that they "Node" is still often used as a synonym of gumma, especially in superficial regions.

ontal and the parietal bones presented bold hemispherical tumours that at ppeared as if they must be malignant growths, especially as one upper was enormously swollen and in part gangrenous: the effect of treat- with iodide of potassium in this case was wonderful. When such s affections are recent they often feel soft and semi-elastic, and at an y one may find that there is a grey succulent material, more or less ively caseating, which can be shelled out of the excavated substance bone. At a later stage the soft substance may be absorbed, and there e left a central depression, with a thickened zone around it. But the bones of the limbs the whole of the gummatous growth is more converted into a uniformly dense, raised, bony mass.

he *testes* are often affected with syphilis in the secondary stage. Later n yellow gummata form in the proper substance of the organ, or epididymis, and can often be easily detected during life. But in cases a diffused fibrous thickening, with atrophy of the secretory ure, is all that one discovers at an autopsy. This suffices to account e fact that so many of these patients are unable to beget children. he could hardly in the living subject recognise such an affection by ion, for flaccidity often may accompany any serious disorder of the l health. Nor, again, is it in itself a proof of syphilis; at least it is in the *post-mortem* room in many cases where there is no other evidence t disease. Still more doubtful is the significance of closure of the nities of the Fallopian tubes in women.

mong the later results of syphilis are *ocular paralysis*, *tabes*, and *ous disease*.

agnosis.—From the foregoing description of syphilis it must be suffi- evident, not only that the recognition of a disease of which the oms and effects are so numerous and varied must often be very difficult, at to attempt to lay down any rules for its diagnosis would be of little Sometimes the first glance at a patient reveals to a skilled observer ture of the case beyond a doubt; and he is bound to treat it actively, ularly in a married woman, without asking questions which might endless domestic misery. In other instances, even when all inquiries pushed without the least reserve, one is unable to arrive at a positive n until one has watched the effects of remedies for a considerable time. mportant to remember that the body should always be stripped as ghly as possible, and that every part of it should be looked at before ronounces as to the character of a doubtful eruption. Some of the puzzling cases are those in which several cutaneous diseases are present same time; as when a macular syphilide is almost hidden among the es of acne indurata or the papules and pustules of scabies.

ansference of contagion.—Few theories in medicine have been better ved than the doctrine of Ricord that primary syphilitic lesions alone s the power of propagating the disease. Clinical experience has fur- numerous instances of contagion from flat condylomata, and experi- have been made which show that it is possible to convey the virus to y persons by inoculation with the blood of syphilitic patients, or with r from pustules of a secondary eruption, or from an ulcer of the tonsils. her pus from a late or tertiary affection of the skin contains the poison to be still doubtful. There seems to be no doubt that the surface of

the skin must be abraded, or fissured, to allow of the penetration of the vir and probably this is true even of mucous membranes.

There are undecided questions as to the contagiousness, apart fr admixture with purulent discharge, of the natural secretions of the saliva mammary, or lachrymal glands; of the semen, without impregnation of ovum; or of inflammatory exudation from a mucous membrane—as, instance, the gonorrhœal pus in one who has also had syphilis. With reg to this last point, it is suggested that if pus-cells are wandering leucocy from the blood, they must almost certainly contain any virus which blood itself contains. But it is generally held that the saliva and the n are not in themselves capable of conveying the disease. That mixed flu from the mouth may form the vehicle by which the virus passes from person to another is shown by cases of infection in the act of kissing, glass-blowing, when the same metal tube is employed by different workn in turn, in smoking, if one cigar or pipe is used by several individuals, (as in a case related by Dr Baxter) in cleaning the teeth with anot person's brush. Nor must we forget that we may unintentionally in our patients if we omit thoroughly cleansing a Eustachian catheter o laryngoscopic mirror, after using it in a syphilitic case. The disease also been transferred by one person biting another in the hand. Las accoucheurs and midwives have acquired primary sores on the finger w they have overlooked a slight abrasion upon it during their attendance o woman with flat condylomata or other specific affections of the genitalia.

With regard to the ways in which syphilis is transmitted, it is perh worth notice that among married women of the lower class the disea often traced back to a confinement, when probably the patient really deri it from her husband after her convalescence, he having become infecte the result of adulterous intercourse while she was in childbed.

Other special pathological points.—Concerning the nature of the prim indurated papule or chancre two opposite opinions are entertained. S think that it is itself a local expression of an infected state of the bl Others, following John Hunter, suppose that the virus does not reach blood until after it has multiplied itself at the seat of inoculation and passed along lymphatic vessels, so as to be discharged with their conte into the great veins. In support of the second view, attention has recen been drawn to the fact that, in the ordinary indolent inguinal bubo, glands which lie along the iliac vessels become swollen as well as those wh lie superficially in the groin. This is said to be shown by three preparati at the Lourcine Hospital in Paris, each of which was taken from a wo who died of some intercurrent disease while suffering from sores upon vulva. But, as Bäumler remarks, if such were the mode of transmissio the syphilitic poison, one would expect that under ordinary circumstan the constitutional symptoms should be longer delayed than when, the s of the primary affection being on the lip or on the finger, only a single gr of glands would have to be traversed. Moreover, even if the *irritation* wh causes the glands to swell does spread from one gland to another, it by means necessarily follows that *infection* of the blood takes place by the sa slow and circuitous route. The analogy of vaccination seems to suggest t it is quite possible for an indurated papule or chancre to be the direct re of inoculation of the specific virus *in situ*, and yet for the blood to be abs poisoned from the moment when such a lesion begins to be develop The rarity of multiple primary syphilitic sores, and the fact that the se

f a primary sore is scarcely ever inoculable upon the patient, point y in the same direction. One might have expected to settle the point e by excising indurated chancres, or destroying them with caustics r very commencement, but at present the evidence as to the effect of perations is conflicting.

other question concerns the relation between the secondary and the y stages of syphilis. Wilks has long thought that visceral lesions st frequently met with in cases where cutaneous eruptions and other cuous symptoms of the disease had been absent or but slightly marked; is opinion has not been positively proved to be correct; and, if it we should still have to ask whether it is not the omission of early e treatment in the cases in question which determines the occurrence ote effects. Hutchinson regards the tertiary lesions as sequelæ rather mptoms of syphilis—"regrowths" in morbid structures left behind he secondary period. Of the points which he makes in support of gestion, the most important seems to be the fact that the liability to k of tertiary symptoms sometimes continues after the patient has ed a family of healthy children, and when one can therefore hardly e that his blood still retains the virus.

gnosis.—A patient affected with syphilis is apt to be exceedingly cious in his inquiries as to whether the disease will leave a per- taint. He has heard of instances in which, after the lapse of many tertiary symptoms have appeared; and he has acquired a notion that philitic poison differs from the contagious principles of all other diseases in being never eradicated. But we are probably justified ring him that the distinction is one of degree rather than of kind. immense majority of cases a person who has had syphilis is after a rs free from it in every sense in which it can be said that one who d scarlet fever or smallpox is free from those diseases. In each e the only appreciable difference between the patient and other people he is no longer capable of taking the infection. Nevertheless there any cases in which, notwithstanding careful and long-continued ent, relapses occur again and again. True, they can be cured again ain, but the liability to them seems to be ineradicable.*

inions probably differ widely as to the extent to which the pre- of a syphilitic taint *modifies* other diseases. Some surgeons have hat it may interrupt the natural course of almost every morbid ; that a broken bone may fail to become united, and a wound refuse l, until the virus is neutralised by the administration of specific nes. All this is, however, very doubtful. On the other hand, it is stablished that an injured part may gradually take on the characters yphilitic sore; but then psoriasis or eczema may develop itself upon which have been bruised or lacerated in a person liable to one or the of these diseases. There is no reason to believe that the effects of e diseases like scarlatina or diphtheria on the throat, rheumatism on ints, or psoriasis, scabies, and eczema on the skin, are the least ed by the presence of syphilis, for the two diseases may occasionally n in the same person, each unaffected by the other.

ith regard to *protection*, it is well known that a person very rarely has

n this question see Dr Gowers' somewhat too gloomy statements in his admirable on syphilis ('Brit. Med. Journal,' 1889).

an indurated chancre and afterwards a secondary eruption and sore-throat two different occasions; but several instances of such an occurrence h now been recorded. In a case of Hutchinson's the interval of health only three years; it has generally been much longer. The second att has always been very mild. On the whole syphilis protects against it more perfectly than measles or scarlatina, though perhaps not so perfec as typhus.

The *prognosis* of syphilis depends partly upon the treatment of syphi for over the manifestations of the disease the administration of drugs immense power. To adults, indeed, syphilis is scarcely ever directly fa Bäumler mentions, as the only two ways in which it can destroy hæmorrhage dependent upon spreading ulceration (*phagedæna*), and exha tion appearing when the early symptoms are very acute and severe. only case that has come down to the *post-mortem* room at Guy's Hosp during the last thirty years, in which death was attributed to syphi apart from any visceral lesions, is that of a man, aged thirty-one, who in one of the wards for seven months, with a rupial eruption on the sh and complaining of pain and swelling in his joints. The most extre wasting took place; his knees and his left arm were rapidly contracted; wrists and ankles were enlarged. Towards the last he was drowsy listless, and at the autopsy the brain appeared to contain an excess of flu

Indirectly, as we shall see in the chapters on diseases of the br larynx, lungs, liver, kidneys, syphilis is the cause of a very large mortal If, therefore, its prognosis is almost always favourable, it is because proper treatment we can not only cure the early symptoms, but prev their more dangerous after-effects.

Treatment.—Mercury has been employed as a remedy for syph during nearly four hundred years,* but unfortunately the experience wh appeared to justify its use was long vitiated by an obvious source of err the natural course of the disease, when unmodified by drugs, had never b carefully studied. Moreover, the metal was generally given so freely as cause profuse salivation, with the most pernicious results. Thus, wh certain English army surgeons, in the early part of the present centu introduced a "simple treatment," in which mercury found no place, th had the immense advantage that their patients remained free from ma evils to which persons suffering under syphilis were then commonly s jected by their medical attendants. And what made the difference more striking was that the real nature of syphilis as a specific disease be then altogether unknown, the same methods had been employed indiscri nately for all venereal sores.

It is now an established fact that it is possible for not only the prima lesion of syphilis, but also the various secondary and tertiary eruptions subside spontaneously without any specific treatment; indeed, this res would occur (sooner or later) in most cases if the patient were placed und favourable conditions.

Nevertheless, experience tends irresistibly to the conclusion that patient who has syphilis derives immense advantages from a course of m curial treatment, provided that it is judiciously managed. It has be

* Its use against syphilis was known as early as about 1510, when Fracastorius wrote
Argento melius persolvunt omnia *vivo*
Pars major; miranda etenim vis insita in illo est.—'De Morbo Gallico,' ii, 2

r tried than any other specific remedy, and has stood the test of
nulated experience. Among those who throughout the civilised world
est qualified to form a judgment upon the matter there is at the
nt time hardly any difference of opinion; and what adds to the force
is consensus is that it has been arrived at by many paths, after pro-
d controversy, and by many who were originally in doubt or in
ition.

is certain that mercury, properly administered, is in no way injurious
e patient's general health if he is otherwise sound. Even in the case
erson with a tubercular tendency, the drug may almost always be given
perfect safety, although one should perhaps be more cautious with it.
hould the actual existence of visceral disease prevent our prescribing
xcept that one naturally would refrain from treating a mild form of
lis at all, if the patient were obviously about to die in a few months of
sis, cirrhosis of the liver, or renal dropsy.* It is generally thought
yphilitic affections attended with much ulceration should, if possible,
ated by other remedies rather than mercury, but Hutchinson is now
ed to doubt the correctness of this opinion. The cachexia resulting
syphilis, so far from being a reason for avoiding mercury, is often
ly curable by its use.

Iercury has a most marked effect upon the induration of a primary
ing papule or chancre. The treatment of such affections belongs, how-
to surgery rather than to medicine.

Iercury if administered systematically during the existence of a
ry indurated sore will often prevent the development of secondary
toms. This is perhaps more important than any other point connected
the treatment of syphilis, and it deserves the most careful verification.
esent the best evidence in favour of it is the statement of Mr Hutchin-
a paper read before the Hunterian Society in 1874, especially when
asted with his previous opinion on the same subject in Dr Reynolds'
em of Medicine.' Formerly he thought that secondary symptoms were
e most part inevitable whatever treatment might be adopted. But in
es of eleven cases of vaccino-syphilis in which he began to give mercury
months after contagion, and about a fortnight after the appearance of
ecific induration, not only did every patient quickly get rid of chancre
landular enlargement, but only about half of them ever showed secondary
toms: these, moreover, were almost always of an exceedingly mild type,
lid not appear until many weeks later than they would have done if no
cine had been taken. . Now, where syphilis has been intentionally inocu-
upon healthy persons constitutional effects have scarcely ever been
ing; and the same may be said of cases of vaccino-syphilis in which early
urial treatment has not been adopted.

Iercury renders the secondary symptoms of syphilis mild and of
duration in proportion as its administration is begun early and is
d on regularly and without intermission for a considerable time.
this point also Hutchinson has recently spoken very decidedly.
erly he cited instances in which a patient, while actually salivated for
in one eye, became attacked with the same affection in the other eye,
nding to establish an opposite conclusion; but now he says that in such

*Even in such cases the supposed "phthisis" might be syphilitic peribronchitis and
itial pneumonia, the supposed "cirrhosis" syphilitic gumma of the liver, and the
ed "Bright's disease" lardaceous degeneration from syphilis.

cases the failure is probably due to the fact that the "salivation is pre ture," that is, that the remedy has not yet exerted its full influence u the rest of the body, notwithstanding that it has affected the gums severely. "The secret of success is to avoid any interruption of this kin and he insists upon the fact that cases in which the secondary symptom syphilis are exceptionally severe are almost always cases in which merc has not been given.

Mercury if administered efficiently during the primary and second stages of syphilis will probably diminish the frequency and severity tertiary symptoms. Here, indeed, we come to a point as to which it is must be difficult to obtain direct proof, for there are no certain data as the proportion of cases of syphilis which result in tertiary symptoms w no treatment is used; nor do we yet know to what extent their occurre may depend upon the severity and character of the secondary sympto But there is at least a strong presumption that if the drug can prevent earlier and more constant of the phenomena of syphilis, it will not have power over the more remote and exceptional effects of the disease.

If these statements are correct, it evidently is the bounden duty o medical man to prescribe mercury to all patients suffering under primary secondary syphilis, who place themselves unreservedly under his care.

With regard to the relative advantages of the various preparation mercury, differences of opinion prevail. We should prefer those which mild; and it is rarely advisable to prescribe calomel or blue pill, in freque repeated doses, except, perhaps, when it is necessary to produce a rapid ef as when iritis or retinitis is present. Plummer's pill is very useful in t not infrequent cases. It is hard to believe that the hypodermic injectio the bichloride of mercury has any advantages which counterbalance its and its tendency to set up inflammation; yet this is almost exclusively as a method of treatment in some Continental cities, where patients unde hundreds and thousands of subcutaneous or intermuscular injections. preparation is better administered by the mouth in doses of one sixteenth one twelfth of a grain (3j—3iss of the Pharmacopœial solution) three tim day. Another preparation which may often be used with advantag grey powder, in doses of two or three grains: when the gums appear lik to be too quickly affected, further inconvenience may be obviated by lett the patient at the same time take the chlorate of potass; but, as a rule, not desirable to mask the effect of the mercury in this way.

Sometimes, when no mercurials, taken internally, could be borne, the unction of blue ointment has succeeded perfectly. But best of all, in re difficult cases, is the calomel vapour-bath of Mr Lee. About fifteen gr may be volatilised by a spirit lamp over a water-bath, the apparatus be placed beneath a chair on which the patient sits naked, and wrapped ro with blankets. The bath should last from fifteen to twenty minutes.

Except during the cold seasons of the year, the patient may go out, even continue at his occupation, while he is carrying out a mercurial tr ment. He should, however, keep early hours and take plenty of rest; clothing should be made of flannel, and other warm materials; he sho have good food, but should generally abstain from stimulants. Pure air important to him; there are greater advantages in residence at the sea-s than at Aix-la-Chapelle or other Continental spas, where, if report says t the real method of cure is neither the baths nor the waters, but mercury.

Even when syphilis has reached the tertiary stage, mercury should,

be prescribed, if the patient has not already taken it. But in most the remedy which is then most serviceable is Iodide of Potassium. s, indeed, often employed with advantage, even during a protracted lary period; but over the early macular and papular eruptions it has if any power. The most striking effect of this drug is seen in the ment of tertiary nodes and gummata, and of periostitis: over these it has a rapid and sure effect than mercury itself. On the other hand, it does or no good in arterial disease; and after the pains and other symptoms umma have been removed by its use, it is desirable for the patient to go a prolonged course of perchloride or biniodide of mercury. With l to the dose, there has been much difference of opinion. Of late it ecome the general practice to give from ten to twenty grains three a day. It is generally prescribed with aromatic spirit of ammonia. me persons take it more readily in milk, which they keep on a table them and sip from time to time. When the iodide of potassium pro- unpleasant symptoms, they may sometimes be avoided by substituting dium salt. Some patients appear to benefit by this in a remarkable er. Very frequent small doses (four or five grains every or every other is probably the best way of giving either of the iodides when it is tant to bring the patient rapidly under the influence of the remedy.

dide of potassium often exerts so marked an influence over the oms that the patient will go on with it more or less continuously for In such cases it is generally desirable to interrupt its use from time e; and important services are then rendered by the Chlorate of Potass. nate forms of cutaneous eruption may be removed by this salt, given ses of ten grains three times daily. Another medicine prescribed in tervals of specific treatment is dilute nitric acid, and still more valuable is parilla. The fluid extract given in full doses is sometimes followed by testable results. Guaiacum, which three centuries ago had so high a ation as a sudorific in syphilis, has now completely lost it.

is sometimes well to apply remedies directly to the eruptions of lis; some of the local tertiary affections rapidly subside when they are ed with the diluted blue ointment, and calomel should always be dusted mucous patches (*i. e.* flat or moist condylomata). Iodoform ointment 3j) is the best application to foul and sloughing sores. The severe rnal pains produced by nodes may often be relieved by blistering.

ONGENITAL SYPHILIS.—This is a modification of the disease, which, d of being acquired by direct infection, is transmitted to the patient one or both parents. It differs in some respects from ordinary syphilis symptoms, and is of course without a primary stage. Some modern rs prefer to call it "hereditary" or "inherited," because it does not ly manifest itself by well-marked symptoms until some weeks after The discovery, however, has been made that the bones in the fœtus ften affected by syphilis, and there can be no doubt that the malady, if latent, is really present from the very commencement of extra-uterine ust as it is in an adult during the period of incubation, or in the inter- between successive eruptions. The term hereditary is better reserved iseases such as gout and phthisis, which usually begin at a later age. ase of congenital syphilis does not, it may be observed, raise the difficult ion whether acquired peculiarities are ever transmitted. For syphilis , like supernumerary fingers or scars, a structural variation; nor is it, like

grey hair and gout, the result of a supposed transmitted tendency: it poison which is directly conveyed by a process of infection from the pa to the sperm-cell or the ovum.

Method of transmission.—At one time it was thought that infection to fœtus *in utero* must necessarily come from the mother. But observa proved that in many cases the mother of a syphilitic child showed no of the disease. All writers admit that the semen may convey syp directly to the ovum. A man sometimes transmits the disease to his chil notwithstanding that he did not marry until long after the disappearanc all secondary eruptions; but such cases are rare and exceptional. If o consulted about the propriety of marriage on the part of the patient has had syphilis, one should perhaps never declare it to be impossible fo offspring to be tainted: until two years have passed after the complete sidence of the secondary symptoms of the disease marriage must be altoge forbidden; but lapse of time seems, as a rule, quickly to diminish the Hence each succeeding child of the same parents is less likely to suffer the previous one; sooner or later the taint wears out, and the children sequently born remain free. Sometimes, however, one infant may es notwithstanding that both older and younger ones are attacked. There modifying circumstances which may cause the later children of a ma pair to suffer more than the earlier, even when the syphilis had been tracted long previously. A healthy woman, impregnated by a syph husband, gradually becomes herself syphilitic without ever having suf from a primary lesion, so that she forms a second source of infection fo children whom she afterwards brings forth. For there is reason to be that the offspring are much less likely to escape, and that they are often affected severely, when both parents are tainted.

The full extent and subtlety of the contamination which affects the mo of a syphilitic child were first pointed out by Colles, of Dublin, in 1 He formulated the remarkable law which now bears his name, that newly born child, even although it may have symptoms in the mouth, n causes ulceration of the breast which it sucks if it be its mother who su it, though it is still capable of infecting a strange nurse." In other w although the mother may have shown no sign of syphilis, she has yet un gone a modification of the disease, bearing somewhat the same relation t ordinary form that vaccinia does to smallpox, and no less protective i action. The *choc en retour* (as French writers have termed the infection woman through her fœtus) is not, however, always unattended with ptoms. She not infrequently becomes pale and thin, her hair may fal perhaps certain lymphatic glands become swollen, or there may be swelling of bones. Hutchinson has observed that symptoms may first ap at the menopause in the form of so-called "psoriasis palmaris," or sore the tongue. He suggests, as an interesting point for inquiry, the que whether a woman infected in this way *per fœtum* can transmit the tai children subsequently born to a healthy father; one would certainly sup that she could.

Effects on the ovum.—The results of syphilitic infection on the fœtus greatly in different cases. Very often it dies *in utero*, and is thrown off an interval in a more or less decomposed condition, with its cuticle pe in large flakes as the result of maceration in the dark and fœtid liquor a Thus abortions and miscarriages, especially if several times repeated, a valuable indications of a syphilitic taint.

e occurrence of a specific lesion of the *placenta* had long been suspected ; 1863 Dr Wilks cited in the 'Guy's Hospital Reports' some observa- f Mr Wilkinson King, in which the chorion had been found thickened riaceous, and the amnion lined by a false membrane, as the result, it pposed, of syphilis ; in almost every instance abortion had taken place third month. Virchow, however, afterwards described the decidua e maternal part of the placenta as the structures liable to morbid s in this disease. If his view had been correct it would have followed e changes in question must be absent whenever the fœtus derives its n solely from the father ; but there is reason to believe that most hard yellow masses in the placenta which have been taken for gum- ave been mere residues of accidental hæmorrhages. At any rate, ta spreading from the maternal into the fœtal part of the placenta nly once found by Fränkel, of Breslau, who in the 'Archiv für Gynä- ' for 1873 recorded a series of investigations with regard to this sub- In sixteen cases, however, he discovered a peculiar affection of the emselves, a fact of great interest, since it is obviously compatible with ire absence of infection from the mother. The lesion in question s in a dense growth of round- or spindle-cells in the substance of the ttended with a more or less complete destruction and disappearance of ascular loops, and ending ultimately in a process of fatty degeneration. altered are less easily isolated from the structures in which they are ded ; they are swollen and opaque, and have irregular outlines and s extremities. Sometimes the whole placenta is uniformly affected ; en remarkably large and heavy (even to the weight of two pounds), ough, and of a pale greyish-yellow colour. In other instances only portions of it are diseased ; these appear as opaque wedge-shaped surrounded by zones of congestion ; in the healthy portions there en hæmorrhages. Once Fränkel found that the cell-growth had ex- from the villi into the maternal part of the organ. He is in doubt r the premature expulsion of the fœtus, which in his cases took place ing periods from the sixth month onwards, is dependent upon the al lesion directly, or rather upon the death of the fœtus. In some es gestation appeared to have gone on to its natural limit, and he morbid change was partial the child was sometimes born alive. The t period at which the affection of the villi was detected was at the nonth ; it often seemed to be only just commencing. We have still n whether a similar lesion exists when abortion occurs at the third or rth month.

casionally a syphilitic fœtus shows gummata in the *liver*, but in the majority of cases what alone characterises the placental disease as tic is a peculiar morbid condition of the *bones*, which is rarely absent. ffection, first described by Wegner, of Berlin, in 'Virchow's Archiv' 70, but independently observed by a French physician, Parrot, at the same time, seems to begin at a very early period of fœtal life. hat which constitutes rickets, it has its chief seat at the meeting etween the shafts of the long bones and their epiphysial cartilages, n the case of the ribs, between their anterior extremities and the ges which tip them. The meeting lines in question become much r than natural, uneven, and irregular. But here the resemblance There is in syphilis comparatively little increase of the normal of proliferation," whereas the "zone of incrustation of cartilage" is

enormously exaggerated. It forms a thick layer, dense and homogene but friable, white, and opaque, like mortar; long processes project fro into the substance of the cartilage beyond. As the affection advances layer becomes separated from the shaft by a soft, or even semi-fluid, grey red or yellowish material, consisting of granulation tissue, which may s off into pus. Another feature is the formation of new bone round the side of the shaft in the neighbourhood of the epiphysis.

Wegner terms this lesion a "syphilitic osteochondritis." It is wi diffused throughout the body, but it is said to be always most marked w the growth of osseous tissue is naturally most active; and what is interes is that precisely the same distribution is stated to obtain in the cas rickets. Thus the lower end of the femur is the favourite seat of diseases, while the corresponding part of the humerus is least often affe by either. Parrot, however, says that in syphilis the new bone is most a dant just where the other changes are least developed; and this corresp exactly with a remark long ago made by Hutchinson to the effect that ostitis in congenital syphilis is more often met with just above the elbow than anywhere else.

In a few exceptional instances, when an infant infected with syphi born alive, the soft material between the shafts of the long bones and epiphyses increases to such an extent as to detach them from one ano and form fluctuating purulent swellings beneath the periosteum. The then complete loss of power in the limbs, attended with so little pain it has actually been mistaken for paralysis. The child's hands are desc by Parrot as lying pronated by its side; its legs are extended, and it is lifted up they hang helpless and swing to and fro. After almost all the principal epiphyses may be found separated from shafts.

Henceforth there ought to be little difficulty in diagnosing cases of severe kind, but it is otherwise with the ordinary form of the disea which there is seldom a sufficient degree of enlargement of the ends of bones to justify one in speaking positively about it, at least in very y infants. Indeed, Köbner has shown that even after death the microsc often required to reveal the presence of the lesion.

These lesions of congenital syphilis are to be carefully distinguished those of Rickets, as will be explained in the chapter on that disease i second volume of this work.

Effects at birth.—As a rule, a child infected with syphilis does not ma symptoms of the disease when it is first born.

One exception is, a peculiar and rare form of bullous eruption, whi known as *pemphigus neonatorum*, and which may either be present at the of birth or come out a few days later. In 1851 there was a discussion this affection in the Academy of Medicine in Paris; Paul Dubois dec that it was syphilitic, while Cazeaux maintained the contrary. Prof in future cases an examination of the bones would easily settle the que The bullæ are flaccid, and contain opaque serum or pus; their favourite are the soles of the feet and the palms of the hands. This eruption gene proves fatal.

The *thymus* has been found in a state of suppuration by Dubois; was no abscess, but the organ when squeezed emitted drops of yell pus, easily distinguishable from the opaline liquid which it may co when in a normal state. Another morbid condition, first pointed ou

ıl as occurring in the bodies of syphilitic children, consists in the ıce of yellowish-grey indurated nodules in the *lungs*, softening in their s into cavities. Lastly, Gubler has described a peculiar change in the the organ is not only enlarged, hard, and elastic, but its cut surface a number of small, white, opaque grains on a uniform yellowish d. Dr Wilks showed a specimen of this affection at a meeting of the logical Society in 1866; it came from an infant one month old. The is often larger and firmer than usual. Acute peritonitis, or pleurisy, ningitis has sometimes been found to be the cause of death. In cases ortion from syphilis, the state of the skin may suggest the cause of ath of the fœtus.

ffects after birth.—The more common symptoms of congenital syphilis ally begin to show themselves towards the end of the first month of uterine life or in the course of the second month, rarely after the end third month.

he earliest is, in most cases, *nasal catarrh*, producing what is popularly the "snuffles." This is attended with the discharge from the nostrils duid, at first thin and serous, but afterwards viscid, so that it dries up rusts which obstruct the passage of air. Consequently the child is no r able to breathe while it is at the breast; it takes the nipple into the ı only to drop it again, and thus, as Diday pointed out, it fails to get h food, and rapidly loses flesh. Indeed, although syphilitic infants ometimes at birth well-grown, plump, and fat, and may even remain so ghout the whole course of the disease, the rule is that they are from rst, or soon become, pale and wasted; they have a dull, opaque, yellow, rinkled skin, and "look like little old men."

oon after the catarrh there appears an *eruption*. Its favourite seats are ates and the face, but it may cover the whole of the body and limbs. It sts of maculæ, blotches, or flat papules, sometimes bright red, but ly brownish or copper-coloured. They are sometimes isolated, some- so confluent that they have been said to look like erysipelas. It is times difficult to distinguish between syphilis and the effects of the 's negligence in allowing the parts about the anus to remain soiled with ta, or in drying them insufficiently after washing. As a rule, however, ed blush produced by mere irritation is ill-defined and fades away at dge, whereas at the margin of a specific rash there are seen isolated, ly outlined blotches. The inflamed surface, moreover, is uniform and t red in ordinary dermatitis, it is patchy and yellowish or coppery in caused by syphilis. The papules themselves may have smooth and l surfaces, or they may be dry and horny, raised here and there into l bullæ, or superficially ulcerated. At the corners of the eyes, about ngles of the mouth, in the folds of the neck or of the groins, or round nus, the papules often become covered with a moist opaque layer, and e more or less the appearance of flat condylomata. But typical ous patches," occurring chiefly at the anus, are comparatively seldom n children less than eight or ten months old. Sometimes the eruption a pustular form; the greater part of the body may then become ed with moist scabs, separated by cracks, from which a sero-purulent is constantly oozing.

n the interior of the mouth ulcers are often present, or there may be a e *stomatitis*. The latter affection involves the gums and dental sacs; hinson has seen it lead to necrosis and exfoliation of the alveoli. The

mucous membrane of the nose may likewise ulcerate, and discharg sanious fluid, perhaps containing fragments of bone from the septum.

Another but a rare symptom is *iritis*. Of this Hutchinson, some y ago, cited twenty-three cases, the majority of which occurred in girls. was usually seen at about the age of five weeks. Sometimes it was lim to one eye, sometimes it affected both eyes. It was attended with irr larity of the pupil, alteration of colour in the iris, and the exudatio white, yellow, or red lymph. But it required to be carefully looked fo the cornea was generally clear, with only a faint pink zone round its mar

Transference from the child.—Although, in accordance with Colles's la child suffering under congenital syphilis is incapable of infecting its mot it may give the disease to any other woman who suckles it. Diday c records cases in which women advanced in years appear to have contra syphilis by kissing such infants; and the use of a spoon that had previo been employed in feeding them has been followed by the same terrible co quences. Probably the source of the virus is always the secretion of ulcer about the lips or within the mouth. Hence Diday lays great stres the importance of searching for such ulcers, when a child born of syphi parents is to be placed with a wet-nurse. But as it is practically impos to be sure that the back of the throat is healthy, French writers advise as soon as any cutaneous eruption, or even a nasal catarrh is discover one should, for the nurse's sake, insist that she should no longer suckle an infant. If one knew of a wet-nurse who had had syphilis she migh engaged for the service; but otherwise recourse must be had to the feed bottle. The primary lesion on the nipple of a nurse is described by Di as a small red papule, slightly desquamating on the surface. When infected, she in her turn may convey the disease to other infants. That milk is not the vehicle of the poison is shown by an observation of Henry Lee's. A woman, who was nursing a syphilitic child with one bre acquired an ulcer upon that breast, and afterwards an eruption; but own infant, for whom she reserved the other breast, went on sucking for weeks and remained healthy.

Syphilis contracted by an infant from a wet-nurse does not appea differ in its symptoms from the inherited form of the disease.

Later effects.—The subsidence of the usual symptoms of congenital syph is occasionally followed by the development of others, which are obvio analogous to those that constitute the "tertiary" stage of syphilis in adult. Thus nodes may appear. Hutchinson says that the bone most o affected by them is the humerus at its lower end, and that they often reach a size as to impair the movements of the elbow-joint. A girl of eight or years old originally came to Guy's Hospital on account of large ulcerating rupial patches, covered with thick brown crusts, on her trunk and li Under iodide of potassium they healed with marvellous rapidity, but nevertheless returned with extensive periosteal swellings upon the tibia ulna, and it was two or three years before she was finally cured. Ano form of eruption consists of raised red rings or zones, somewhat like "circinate squamous syphilide" of adults. At the Evelina Hospital we had a girl, eight or ten years old, with a sloughing ulcer which had destro the soft palate, and with great enlargement of the metacarpal bones of hand. Hutchinson cites the case of a boy, aged eight, almost the whol whose calvaria was involved in disease at first regarded as "strumous," which proved to be syphilitic; he was the son of a clergyman, but his mot

contracted the disease from a former husband, an officer in the army. lar swellings, doubtless gummata, in syphilitic children's testes have or twice been found at Guy's Hospital after death. Of lardaceous se, as the result of congenital syphilis, nothing appears to be known.

The most interesting of the remote effects of the hereditary disease are, ver, some which differ altogether from those of ordinary acquired syphilis. discovery is due to the acumen and patience in observation of Mr hinson, to whose masterly account very little has been added by any rs but himself. One peculiarity is that on each side of the forehead *ontal eminence is protuberant.* M. Parrot maintained that, in addition to ntra-uterine osseous lesions already described, syphilis gives rise, during infancy and childhood, to a morbid change in the bones resembling that kets; and Hutchinson now accepts this account of the appearance in ion. A character on which Parrot has further insisted is the presence of eminences upon the bones forming the sides of the anterior fontanelle; their resemblance to the buttocks he calls such a calvaria "natiform." lso claims for congenital syphilis the affection known as Craniotabes, n has hitherto been deemed rachitic. But this is more doubtful.*

The other signs of congenital syphilis established by Hutchinson are far distinctive than the shape of the forehead. One is a *sunken bridge of the* resulting from long-continued swelling of the parts within, or from loss pport by exfoliation of the septum. Another is the presence of radi- *linear cicatrices* at the angles of the mouth, running outwards towards heeks. A third is a thick, pasty, opaque condition of *the skin*, the hair scanty, and the nails broken and apt to split.

Most characteristic of all is a peculiar change in certain of the permanent , and particularly in the *central upper incisors*, which for this purpose hinson terms the "test-teeth." Not only are they much smaller than , but they are "notched" and "pegged;" the former peculiarity con- g in a single deep crescentic excavation of their free edge, the latter gradual convergence of their sides towards this edge. It is supposed this alteration in their form is the result of their having been disturbed eir growth by the stomatitis which is so marked a feature of the dis- during infancy. When they first protrude from the gums the notch is present; in its place there is a row of minute projections; these soon wards break off. Similar changes may sometimes be observed in the of the other incisors. It is important to note that the above single notch is alone indicative of syphilis. Horizontal grooves on the sur- and numerous small notches in the edge, are common enough in per- who are altogether free from any congenital taint. Also, it is certain the defect of the teeth due to mercurial salivation during dentition is distinct from that of syphilis.

The appearances just described enabled Hutchinson to identify as litic an affection of the eye, the real nature of which had not previously suspected, and which was always known as "strumous corneitis." e can be no doubt that the "ugly form" of scrofula described by on and other writers was for the most part syphilitic, just as the tty form" was tubercular. *Interstitial keratitis*, as it is now called, occurs y in children between eight and fifteen years old, but sometimes in s up to the age of twenty-five or twenty-six. It is more common in girls in boys. It seldom remains limited to one eye, but commonly attacks both

* See on this and other points Fournier's 'La Syphilis héréditaire tardive,' 1886.

in succession at an interval of a few weeks. It begins as a dotted haz near the centre of the cornea, which spreads until almost the whole of opaque, like ground glass. The patient often complains but little of int ance of light, and there is not generally much congestion of the conjun or sclerotic at first. Ulceration never occurs, but at a certain stag cornea may become so vascular as to be uniformly pink or salmon-colo When the affection is at its worst, vision is often reduced to a bare pe tion of light; but in the course of a year or eighteen months a surpr degree of improvement takes place. The opacity slowly clears up, perhaps only a few hazy patches remain; and the patient's vision ultimately be little impaired. Sometimes iritis occurs as a complicatio choroido-retinitis. Interstitial keratitis is very rare in acquired syphil

Another morbid condition occasionally seen in the subjects of congé syphilis is *deafness*, apparently from some disease of the deeper par both ears. It belongs to the same age as interstitial keratitis.

Protection.—Is a subject of congenital syphilis less liable than o to acquire the disease later on in life? Hutchinson has recorded inst in which chancres have been contracted under such circumstances, an in which constitutional symptoms occurred; and his opinion is that th of a patient's parents having had syphilis renders the acquired di milder but does not altogether prevent it.

Transmission.—Is it possible for the taint to be handed down to a generation? Hutchinson has about eight times had opportunities of ex ing the children of persons undoubtedly the subjects of congenital syp and with one exception they appeared to be healthy.

Treatment.—Congenital syphilis seldom proves directly fatal, except after birth. In mild cases the eruption may subside in a few weeks wi treatment; and in the more severe forms of the disease specific treat is very successful. The usual plan is to prescribe the mercury chalk powder, in doses of a grain or two grains, two or three times a according to the age of the infant; a minute quantity of Dover's pow or of carbonate of soda may be given with it, according to circumsta Many prefer, however, to use the diluted mercurial ointment externall may be applied on the inside of a flannel band which is sewn round the as Sir Benjamin Brodie recommended; or ten or fifteen grains of it be rubbed into the child's armpits once or twice daily for a few minutes time; or the solution of perchloride of mercury may be ordered in of ♏xx to ♏xxx three times a day. Mucous patches about the anus be dusted over with calomel.

In the later forms of congenital lues, mercury and potassic iodide a be used as in the acquired disease, and they are no less efficacious.

TUBERCULOSIS

these facts speak as it appears to me so eloquently and decisively for the infectious ? Tuberculosis, that we are not shaken in this conviction by the direct demon- of the tuberculous virus being up to the present time an unsolved problem."

COHNHEIM, 1881.

of tuberculosis—Anatomy and histology of tubercle: views of Laennec, yle, and Louis: of Virchow, Schüppel, and Ziegler—Tubercle a product modified inflammation—Infiltrating tubercle—Caseous and fibroid trans- rmations of tubercle—Ætiology—Koch's tubercle-bacillus—Result of experi- nts on animals: inoculation of tubercle: Perlsucht: infection by the mouth d by the lungs—Predisposing causes—Invasion and spread of tubercle in body—Course of acute tuberculosis—Varieties of chronic tuberculosis: thisis pulmonum, laryngis et ilei; tuberculous inflammation of serous mem- anes; genito-renal tuberculosis; Addison's disease; tuberculosis of the mph-glands and spleen; tuberculous disease of joints: multiple tubercle erally—Concluding historical retrospect.

of the most important diseases which, under various forms and ry different local characters, affects almost every organ of the body rculosis; and this must now be included in the extensive group of infective fevers. Like syphilis, it usually comes before us as a chronic and it differs widely in its clinical aspect from the continued fevers nthems, particularly in the apparent absence of an origin from con- but in this respect it does not differ widely from pyæmia, or even teric fever, erysipelas, and diphtheria, in all of which it is difficult to trate a specific and constant contagion.

presence of the infecting virus is so wide-spread that it is absent few parts of the world, but susceptibility to it is happily far less ; hence the predisposing causes are much more important in this any other infective disease. The healthiest organisms appear to have any power of resisting the invasion of smallpox or of plague, but re exposed to scarlatina who do not take it; and this power of re- is far more common in the case of tubercle.

en the virus has gained an entrance it is most active locally. ntoxication" of the lymph, blood, and tissues is less rapid, and less complete, than in the typical specific fevers; there is, in fact, a ation of local granuloma-growths (the tubercles) with general febrile ance (so-called hectic fever). In this respect it forms one of a f infective maladies, from purely general to purely local: typhus, , syphilis, tubercle, leprosy, lupus.

remarkable are the affinities of tubercle to specific fevers that Cohn- redicted that one day phthisis and other tubercular maladies would rded as due to a specific contagium: see the passage at the head of apter from his 'Vorlesungen ü. allg. Pathologie' (Bd. ii, S. 709–712). s prediction has been fulfilled by Koch's discovery of the bacillus of e.

We must not commit the error of ascribing all the symptoms and m changes of tuberculous diseases to this cause; they are local inflamma as well as specific infective lesions, so that we shall best consult pathol accuracy as well as practical use by treating in this place of tuberc generally, among specific fevers, and leaving the account of its local var to the chapters which relate to diseases of the brain, of the lungs, abdo and other parts; just as we have dealt with syphilis as a specific dis among infective fevers, and reserve the account of its local effec subsequent parts of this book.

Anatomy of tubercle.—One great stumbling-block in the way of a understanding of tubercles has been imperfect observation of the chang appearance which they present in different stages of their forma Laennec described them as first "having the appearance of small semi-t parent grains, greyish or colourless, varying from the size of a millet to that of a hemp seed. . . . Afterwards they gradually increa size, and as they do so they become yellowish and opaque, beginning i centre. . . . Several unite together to form larger masses, pale ye opaque, and of the consistence of very firm cheese. . . . At l they soften and finally liquefy; this change also begins in the centre progressively approaches the circumference." The grey semi-transp granules he termed "miliary tubercle;" to the yellow cheesy mass applied the unfortunate name of "crude or immature tubercle."

Louis, whom Laennec's premature death left the leading patholog Paris, limited the application of the word tubercle to the yellow op stage, his method of statement being that the grey semi-transparent gra "undergo conversion into tuberculous matter." By most subsequent w the early grey stage was passed over altogether. Perhaps they wer fluenced by the misleading term "crude" tubercle, applied to the yellow s At any rate, they taught that the yellow material was deposited as such the blood.

Virchow, therefore, effected a most important reform of pathological trine when he showed that a yellow cheesy substance, identical with "cr tubercle, may arise out of decaying matters of various kinds, from ordi inflammatory exudations up to sarcoma and carcinoma; in other words, caseation is merely a mode of retrograde metamorphosis. Neverth Virchow certainly underrated the extent to which cheesy masses in the l and in other organs are really of tuberculous origin; though he never so far as one or two of his followers, who almost deny that tubercles ca at all.

A proof that grey and yellow tubercles may be stages of the morbid product is, as Louis long ago pointed out, that the successive ch described by Laennec may often be observed to occupy definite positio the lung; towards the base there are grey semi-transparent tubercles; hi up they are of opaline aspect and yellowish in their interior; higher still are yellowish-white throughout their entire substance. He might added that they become larger and approach one another more closely below upwards. Now, since it is certain that the upper lobe of the lu almost always the earliest seat of tubercles, and that they gradually sp downwards through the organ, the conclusion seems indisputable that are first grey and afterwards become yellow. It is, however, also true tubercles often undergo caseation before they are large enough to be vi

the naked eye. Both in the pia mater and in the liver the writer has atedly found tubercles of microscopic size which were already opaque, not in the centre, but in the greater part of their substance. It is therefore possible that no grey tubercles may be discoverable in a diseased organ, when tuberculosis has been rapidly destroying it. Again, another fact, h is still more important, is that tubercles, instead of caseating, often ergo a different change, which leaves them permanently grey. The earliest rvation of tubercles in this condition dates further back than the writings aennec himself. A few years before the publication of his great work, 827, another French physician, Bayle, had divided phthisis into different es, only the first of which was termed by him "tuberculous." To the nd species he gave the name of "Granular Phthisis," and described the s as "stuffed with transparent shining granulations, of cartilaginous re and consistence, never opaque, and without any tendency to soften." , it is a curious circumstance that those pathologists who (as we have) came to regard a yellow cheesy condition as typical of tubercle, habitu- spoke of *recently-formed* grey tubercles as the "grey granulations of e;" yet a perusal of the four cases of granular phthisis, related in detail hat writer, shows clearly that in three of them, at any rate, the disease of a *chronic* kind. Such a degree of hardness as he attributes to his granu- ns is, indeed, inconsistent with their having reached only an early stage eir development; and he also expressly mentions that they are some- s pigmented or speckled with brilliant black dots and lines, which will own elsewhere to be characteristic of *chronic* tuberculous affections of the s and bronchial glands. In fact the grey granulations of Bayle are really rcles which, instead of caseating, have undergone a fibroid change, and thus become permanently hard and semi-transparent.

Miliary tubercles, then, may be of three kinds:

1. *Soft grey granulations*, always of recent formation, and essentially tran- y in their characters.
2. *Yellow granulations*, of either recent or old formation, tending, if they ergo further change, to soften or liquefy.
3. *Hard grey granulations*, always chronic, and liable to no change, except ientation.

Histological characters of tubercle.—It may well be supposed that histolo- s have had much to say with regard to the nature of the tubercular ess. But the present writer ventures to think that their views on this ect are fundamentally erroneous. Their object has been to discover e microscopical characters which should serve to distinguish tubercular ns from all others. Thus Lebert many years ago thought that he had d a specific "*tubercle-corpuscle*" in the yellow cheesy material which was hat time taken for the typical form of the morbid product. Since how taught that this material is already in a state of decay and degene- on, the search has been actively prosecuted in tubercles still grey and nt. Virchow himself held that their histology is that of a form of our, namely, *lymphoma;* according to him, individual tubercles are com- able with the Malpighian bodies of the spleen; and, as he believed that are apt to arise where no such lymphoid structures are naturally sent, he regarded them as "heteroplastic" new growths. Subsequent ters further developed this view by insisting on the existence of a reticu- d stroma, such as belongs to other lymphomata. It was also suggested t the true seat of tubercles was perhaps the interior of the lymph-

channels throughout the body, and that their cells might probably formed by a proliferation of the lymphatic endothelium. On the ot hand, Dr Sanderson endeavoured, in 1868, to demonstrate in those parts which tubercles occur the normal presence of lymphoid tissue, an ov growth of which might lead to their formation, and thus render th "homœoplastic."

But about that time the attention of histologists became drawn another element of tubercle, which has played a principal part in t various theories that have since been advanced. That very large cells sometimes to be found in a tubercle had long been known; Virchow hims described cells containing twelve or more nuclei. Langhans and Schüp now insisted on the frequent presence of such bodies, and applied to th the name *Riesenzellen* or giant-cells, which had previously been invented Virchow for similar corpuscles in certain sarcomatous tumours (v. *supra*, 80). According to Dr Hamilton, whose papers in the 'Practitioner' 1880 are illustrated by beautiful drawings, giant-cells occur with the utm regularity in all tubercles. They are from ten to thirty or forty tim larger than the lymphoid cells, which are also present. They are sea times placed in the centre of a tubercle, sometimes laterally. When giant-cell is young, it seems to consist of a large mass of granular pro plasm, sometimes with many nuclei in it, sometimes without them. As grows older, the peripheral part of it (the "periblast") becomes organis and constitutes an almost fibrous sheath, in which great numbers of rou or oval nuclei may be perceived, and from which ultimately processes ar in continuity with the reticulated stroma of the rest of the tubercl Further on, Dr Hamilton speaks of a tubercle as made up of one or mo "giant-cell systems." He imagines that even the "lymphoid" and "e thelioid" cells which are found in the tubercle have originally been co nective-tissue nuclei, which have become detached and thrown off into t meshes of the reticulum.

Histologists are by no means agreed as to the origin of the giant-cells tubercle. Dr Hamilton's view is that each of them arises by the pr gressive growth of a single large connective-tissue element. But Schüpp himself attributed them to the coalescence of leucocytes in the interior capillary vessels. And both Dr Klein and Prof. Julius Arnold ha recently stated that they may be produced by the fusion together of number of epithelial cells, of which the nuclei persist. If such opinio are correct, it is impossible that giant-cells can possess the significance regard to tubercles which Dr Hamilton and others would assign to thes After all, giant-cells are far from being as conspicuous or as easily demo strable elements in a tubercle as they are in a myeloid sarcoma.

Again, the conception of giant-cells as the essential and distinguishi elements of tubercles has of late years been rudely shaken from anothe side. The experiments of Ziegler, quoted above (p. 56) have shown what had been to some extent recognised before him, that giant-cells ar formed in ordinary inflammatory exudation, during the course of it "organisation" or development into tissue. In fact, Ziegler's researche were undertaken with the special object of throwing light upon the stru ture of tubercle, and upon the relations between tuberculosis and inflamma tion. Moreover, nodules, having the histological characters of tubercle but of microscopic size, had already been found by other observers in place where their presence certainly would not have been expected. Köster di

ed tubercles (sometimes visible even by the naked eye) in the granula- which project into the interior of diseased joints; and Friedländer l similar bodies constantly present in lupus and in the walls of scro- s ulcers. It is true that both these affections were previously believed ar a more or less close relation to tubercle. But it is impossible to the same of certain other lesions in which Friedländer or Köster ohnheim found microscopic tubercles. Among them are a shallow of the uterus in an old woman who died of apoplexy, the stroma of a rous tumour, the floor of a phagedænic chancre, and bands of adhesion e pleura or in the peritoneum. Friedländer, indeed, maintains that all cases are examples of "a local tuberculosis," and compares them with fulous testis, or with pulmonary phthisis, which does lead to a general culosis. But this is surely a strained and partial view of the facts.

robable connection of tubercle with inflammatory exudation.—The general usion which we may draw from all these observations with regard to istology of tubercle is that there is nothing in them to prevent our ting the doctrine that *Tuberculosis is a modification of the inflammatory s*, if it is commended to us on other grounds. Some of the reasons led the author of this chapter to accept it will be stated further on; apart from these, it seems to afford the only possible solution of a lty which histologists used to regard as insuperable. Niemeyer's denial tubercles are an essential feature of pulmonary phthisis was based on icroscopical observation that in many specimens of phthisical lungs the id process which precedes the occurrence of caseation is not a deve- nt of lymphoid tissue, but a filling of the alveoli with epithelial cells, other words, a "catarrhal pneumonia." He accordingly declared that onic catarrhal pneumonia, ending in cheesy infiltration, really constitutes rimary anatomical lesion in many cases of phthisis. His opinions have been adopted by many other pathologists. Dr Hamilton, for example, s that dry yellow nodules, such as were described in the lungs by nec, are generally nothing but patches of catarrhal pneumonia; bodies g an aspect similar to that of a tubercle, but as large as millet seeds, ly (he says) prove to be groups of air-vesicles affected with catarrhal monia. But it is the greatest mistake to suppose that the cases in catarrhal pneumonia must be separated from tuberculosis can be d to those of ordinary phthisis, if the separation is to be made at all. he contrary, the principal point on which Dr Wilson Fox insisted in ng the discussion on tubercle at the Pathological Society in 1873 was in the disease known as acute tuberculosis, occurring in children, lations "composed of epithelial proliferation" are generally found in numbers than those made up of lymphatic cells. And in that very a German observer, Hering, actually went so far as to declare that the ary fatal miliary tuberculosis of the lungs ought to have its name ged into that of acute disseminated catarrhal pneumonia. But this l, after all, leave the difficulty untouched; because, as Dr Fox pointed granulations made up of an interstitial small cell-growth are generally resent in such cases, and some granulations consist partly of one kind ments, partly of the other. Moreover, it has been shown by Julius ld that precisely similar epithelial changes to those in the lungs occur liver and in the kidneys, when affected by acute tuberculosis; while has demonstrated a caseating catarrh of the seminal tubules in the so-

called scrofulous disease of the testis. Now, if the tuberculous proces regarded as a modification of inflammation, it is possible to solve the culty without giving up the use of the term tubercle, and without igno the important distinctions that undoubtedly exist between ordinary inf matory affections and those that are tuberculous. The relation of catar inflammation to the inflammatory process in general is still imperfe understood (cf. *supra*, p. 51). But at least this is certain, that the kind of irritation which in most other tissues leads to an exudatio leucocytes, causes, when applied to epithelial tissues, an overgrowth epithelial cells. Ziegler argued that a tubercle owes its rounded form its definite size (varying only within rather narrow limits) to the fact th is produced by the circumscribed action of an irritant of but slight inten upon the spot which afterwards becomes its centre; and he contrasts effect of such an irritant with the far more powerful operation of a s particle, such as gives rise to a miliary abscess. Now it is easy to conc that whereas the circumscribed irritation of a minute area of connec tissue results in the formation of a granulation made up mainly of le cytes, the same kind of irritation applied to an epithelial area may cau granulation consisting of epithelial cells. The two bodies may differ c pletely in their histology, and yet we may be quite right in giving bot them the same name. It is from this point of view that the attempt to a criterion of tubercle in its histological structure appears to be fundament erroneous.

Specific character of tubercular inflammation: infiltrating tubercle.—The ception of tubercle unfolded in the last paragraph is one which Dr F for several years past taught in his lectures on pathology. Koch's disco of a tubercle-bacillus supplied the "irritant of but slight intensity" th needed for the theory. The slow growth of this organism, to which K himself draws attention, accounts for the fact that the morbid process w results from its action is so much less acute than infective inflammation general, with which tuberculosis must henceforth be regarded as c parable.

It is not the least advantage of this view of tubercle that it enables u understand how an affection (whether of the lung or of any other or may fail to present the characteristic granulations, and yet be really tu culous. Laennec long ago described what he termed a tuberculous "infil tion of the lungs," when considerable portions of the pulmonary tissue bec solidified by a greyish and semi-transparent, or by a paler and yellowish-w material; either without any previous development of distinct tubercles around tubercles already formed. Subsequent observers have very gener declared such lesions to be "pneumonic." But in their naked-eye charac these "infiltrations" differ altogether from anything that is seen in ordi pneumonia. Even when the growing edge of a patch of pinkish-grey filtration appears quite homogeneous, one often finds that the less re parts of it, where caseation is commencing, show yellow granulations co sponding exactly with tubercles in size and in general appearan Now, it is easy to see how a uniform consolidation may result from irritation set up by bacilli if they happen to lie very close to one anot through the pulmonary tissue. Again, in association with acute tubercul of other organs, an affection of the membranes of the brain is not frequent, which seems to be a simple inflammation, since no tubercles discoverable. In such cases, the presence of recently formed tuber

here is strong evidence that the meningitis must be essentially of the nature.

aseation of tubercle.—Another peculiarity of tubercular lesions, which is ly less characteristic than the presence of the grey granulations, e tendency to caseate. Most pathologists since Virchow have been eady to assume that caseation is of frequent occurrence in merely mmatory affections. It is of course indisputable that the pus con- l in a serous sac or in a large abscess may, and often does, dry up into esy mass. The same thing is observed in the crypts of the tonsils, in ecal appendix, and in other pouches of mucous membrane, and it is in the walls of arteries when they become atheromatous. But in every f these instances the inflammatory exudation which caseates is more or eyond the range of the blood-vessels. Is it a fact that simple inflamma- ver leads to caseation in the substances of a vascular solid organ—in iver, or in the spleen, or even in a lymph-gland—unless a definite ss has been formed? Such an occurrence is commonly spoken of as if re the most natural thing possible; but it would be difficult to find nces that will bear hostile criticism. Surely, in a healthy person mmation generally runs its course so as to leave no *débris* behind, except an abscess forms and fails to discharge itself. In the solitary follicles e intestine it used to be supposed that caseation was often met with, independently of the tuberculous process; but the writer's own experi- has been that it scarcely ever occurs unless other parts of the body in tubercular lesions.

ne of the most typical features of tuberculosis is the presence of ding ulcers in cavities, with yellow walls of definite thickness, the ance of which regularly undergoes caseation almost as soon as it is d. This process, wherever found, whether as a pulmonary vomica, the liver, the kidney, the prostate, or the testicle, may be safely set as tuberculosis.

hose pathologists who have recognised that caseation is especially apt cur in tuberculous lesions have commonly attributed it to the absence od-vessels in tubercles. Thus Virchow's comparison between tubercles he Malpighian bodies of the spleen was objected to, and with justice, e ground that the latter contain capillaries. Every museum of morbid my contains injected specimens of phthisical lungs, from which it appears not only isolated tubercles, but also patches of caseous infiltration, are utely non-vascular. No new vessels seem to be formed in them, and re-existing vessels of the pulmonary tissue undergo obliteration. The er process of softening and liquefaction, by which vomicæ are formed, rs to be essentially of a chemical nature.

ibroid transformation.—In many cases, as already mentioned, tubercles, d of caseating, undergo fibroid transformation. What determines them e the one course rather than the other is as yet uncertain; but it would that caseation is less frequent in proportion as the patient is older. In s other than the lungs, comparatively little is known about "fibroid" cles. But Schüppel has demonstrated their occurrence in lymph-glands, as shown that the reticulated stroma grows at the expense of the cells, s converted into a transparent, almost homogeneous, indistinctly fibril- material; and Rindfleisch has recorded a remarkable specimen of s tubercles in the great omentum. The occurrence of a similar change bercular infiltration is, probably, the origin of the disease known as

fibroid phthisis. Even in such cases, there seems still to be defici vascularity; for Rindfleisch has insisted on the impossibility of forcing in tion into the interior of indurated masses in the lungs, and contrasts t state with the abundant supply of vessels to the newly formed connec tissue in cirrhosis of the liver and in granular disease of the kidn No writer has laid more stress on the fibrous transformation of tubercles t Dr Hamilton, who, indeed, declares that the natural ultimate destiny of th bodies is to become little fibrous tumours, and that many of the so-cal cirrhoses of organs, both in children and in adults, are in reality the rem of outbreaks of tubercle.

The tubercle-bacillus.—In the 'Berliner klinische Wochenschrift' for A 10th, 1882, Dr Koch made known the important fact that he had discove bacilli in the tubercular diseases of man and of animals,* that he had succee in cultivating these bacilli upon the coagulated serum of blood, and had b able to set up tuberculosis in healthy animals by inoculating the cultiva products. The organisms themselves he described as in length equal t quarter or half the diameter of a red blood-disc (3–5 μ), and in breadth fifth to one sixth of their length. They are slightly curved and have roun ends. When prepared by a particular method of double staining,† t present a blue colour, whereas nuclei and other tissue-elements appear bro This not only facilitates their recognition, but also serves as a point of tinction between them and all other kinds of bacilli, except those of lepr which, indeed, resemble them very closely, and only differ in being still n slender and in having pointed ends. The bacilli of tubercle, while lying in living tissues, produce spores which are oval in form, from two to fou number, and placed at equal distances in their length so as to give the ba a beaded appearance. When grown upon the coagulum of blood-serum, organisms become aggregated together into flat, scale-like masses, hardl large as poppy seeds, which can be lifted off entire, and which are so that it requires some force to break them up. Their development takes p very slowly, about ten days elapsing after infection of the coagulum be any change is discoverable. Another condition necessary to their growt a uniform temperature of 86°—106° F. Hence they cannot grow in open air in this climate.

Koch does not think that previous observers who have described mi cocci,‡ or even rod-shaped bodies, in tubercle, had seen the same organi which were discovered by him. They are not generally present in la numbers, except where the tuberculous process is recent and acti if it is slow, they are often to be found only within the giant-c perhaps one or two in each cell. They do not exhibit spontaneous mo ment.

The original cases in which Koch found his bacilli were the followi (1) Eleven cases of miliary tuberculosis. In miliary tubercles of the lu

* There is still some doubt as to the identity of Perlsucht in cattle (v. *infra*, p. with tubercle in man. Klein finds that the tubercle-bacilli in man are much larger t those in the tubercular lungs of cattle.

† Viz. by methylene blue and vesuvin. Ehrlich has since devised a more deli method by which the bacilli appear magenta-red with fuchsin (hydrochlorate of rosani Weigert, Heneage Gibbes, and many other histologists have described methods of do staining. See an account of them in Dr Crookshank's 'Introduction to Practical Bacte logy,' pp. 162–7, Gramm's method.

‡ Professor Toussaint, of Montpellier; Professor Klebs, of Prague. See Che 'Practitioner,' April, 1883.

ere never absent, but in those which had caseated they often existed
t the edges. They were present, too, in miliary tubercles of the
liver, and kidneys, and in great numbers in the grey tubercles of
neningitis. And they also occurred in the cheesy bronchial glands
he same cases. (2) Twelve cases of caseous bronchitis and pneu-
(3) One case of solitary tuberculous tumour of the brain, the
lying within giant-cells contained in the tissue immediately surround-
cheesy mass. (4) Two cases of intestinal tuberculosis. The bacilli
resent also in the corresponding mesenteric glands. (5) Two out of
ases of freshly extirpated scrofulous glands. (6) Two out of four
f inflammation of joints.*

vious experiments on the artificial production of tubercle.—Koch's discovery
utes the final link in a chain of evidence which has been long accu-
ng, and which places tuberculosis among the specific diseases. In
French observer, M. Villemin, made known a fact which roused the
t interest in the medical world; he found that in certain animals,
larly rabbits and guinea-pigs, tuberculosis could be set up by the
tion of tuberculous matter from the human subject. His experiments
on repeated in this country by Mr Simon, Dr Sanderson, Dr Wilson
nd others. Tubercles from the pia mater or from serous membranes,
substances from the lungs of phthisical patients, even the sputa yielded
n patients during life, were introduced beneath the skin of animals.
were afterwards killed, or were allowed to die of the disease which
d the operation and generally proved fatal in from six to ten weeks.
then found that pus or a dry, cheesy substance had been formed at
t of inoculation, and that bands of induration extended away from it into
acent subcutaneous tissue. The lymph-glands in the neighbourhood
nlarged and caseous. Nodules of various sizes were present in the
the liver, the spleen, and the peritoneum. These resembled tubercles
the naked eye and in their histology. They consisted of a grey,
ansparent material, which underwent caseation from the centre out-
The infective character of the process was further shown by the fact
atter taken from the body of an animal after death was capable of
up the same disease in other animals.

er a time, however, certain observations were made which seemed to
ne bearing of these experiments. Dr Sanderson found that in guinea-
"artificial tuberculosis" arose after the inoculation of pus from the
ary abscesses of pyæmic patients, or even after the mere introduction
an seton of unbleached cotton. Dr Wilson Fox independently arrived
e result by inoculating guinea-pigs with pieces of putrid muscle, or
accine lymph. In Germany, Cohnheim and Fränkel introduced into
ritoneal cavity of animals of the same species portions of new growths,
of healthy organs from the human body, or even bits of charpie or
ercha; and they succeeded in setting up a tubercular process not only
serous cavity itself, but also in the lungs and in the liver. It even
ed that guinea-pigs in which incisions were made, without any injec-
erished afterwards with inspissated abscess at the seat of injury, and
iliary tubercles in their organs; so that it became necessary to give
erimenting upon such animals, and to have recourse to dogs, as not

the twenty-first volume of the 'St Bartholomew's Hospital Reports,' Dr Vincent
states that in twelve ancient specimens of tuberculous lungs, from forty to seventy
d, he succeeded in demonstrating the presence of Koch's bacillus microscopically.

being liable to be made tuberculous by trifling wounds. But, after injec into the jugular veins of these dogs pus from guinea-pigs which had th selves been inoculated with non-tuberculous materials, it was found that dogs likewise became the victims of miliary tuberculosis.

These observations fitted in perfectly with a theory of tuberculosis w had been propounded a few years before the publication of Villemin's exp ments, but which at the time had attracted little notice. In 1857, Buh Munich, had suggested that in the human subject acute miliary tubercu was due to the absorption into the blood of caseous matters from non-en suled yellow tubercles, or from pulmonary cavities not surrounded on sides by dense fibroid tissue. Indeed, a similar opinion had been expre earlier still by Dittrich, of Erlangen. It afterwards became the fashio regard tubercle as always a secondary product, the origin of which was so for in "caseous foci," of which the formation was supposed to precede, i cases, the development of tuberculous lesions. In ordinary cases of pht it was thought that a catarrhal pneumonia, with secondary more or extensive caseation, was the earliest change, and that this might go on great length of time before any tubercles made their appearance, so perhaps the disease might become tubercular only at its very end. Su view was of course in entire accordance with the doctrine of Niem already referred to (p. 310). The acute tuberculosis of children was tr back, in a large number of instances, to a simple intestinal or bron catarrh; this was supposed to lead first to swelling of the correspon abdominal or thoracic lymph-glands, and then to their caseation; and once caseation had commenced the conditions for the development of tub were assumed to be present. Dr Hamilton still maintains this doctrine imagines that in the process of caseation some material, probably a ferm is elaborated, which acts as an irritant upon the tissues to which it is car But surely this theory of tuberculosis is inconsistent with the broad fac human pathology, whatever may be its application to the artificial tu culosis of animals. Nothing is more certain than that, in man, the in sated pus of a common abscess, or the caseous matter of an atheroma artery, or of a degenerating new growth or gumma, does *not* produce tu culosis. The few cases that have been recorded as illustrations of such occurrence serve, by their rarity, to tell against the conclusion they are tended to support. Thus Dr Murchison some years ago exhibited at Pathological Society a specimen in which a simple ulcer of the duode (an affection generally entirely free from all trace of caseation) had followed by tubercular disease in the lungs. Is there the smallest re for supposing that this was anything more than an accidental coincide Dr Hamilton insists upon a case in which a woman died, thirty-three after her delivery, of general miliary tuberculosis. She had been attac by a rigor on the seventh day, and her symptoms were at first mainly th of peritonitis. In many places, says Dr Hamilton, more especially beh the uterus, the peritonitic fibrinous effusion had become caseous, and and there softening had occurred. He assumes that this was the start point of the tuberculosis; but it seems to be very doubtful, according observations made by Cohnheim on the rate of development of acute tu culosis, whether it would have been possible for the process to have run course within twenty-five to twenty-six days, even supposing that the tu culous "ferment" became "elaborated" at the very moment when peritonitis first began. Moreover, there is no difficulty in supposing

man's illness was, from the first, acute tubercular peritonitis, forming, ften happens, the clinically obvious part of a more widely diffused tuberculosis. In fact, cases of acute tuberculosis in man, attributable ction from non-tubercular lesions, are so scarce that it is impossible to any significance to them.

antime, however, it has turned out that the experiments which were ed to prove that tuberculosis could be set up in animals by the tion of non-tuberculous matters had been misinterpreted. Klebs that in all probability they were vitiated either by the accidental e of the specific poison of tubercle at the time when the operation rformed, or by the wound becoming infected with it afterwards. im afterwards frankly acknowledged that, having repeated his inves- ns both at Kiel and at Breslau, he has utterly failed to obtain the same as before; and he became one of the warmest supporters of the nature of tubercle. On referring to Dr Wilson Fox's observations that all the animals which were inoculated by him, whether with ulous or with non-tuberculous substances, were placed in a single so that it was quite possible for them to have infected one another tally.*

nheim made a further advance, by showing that in rabbits and in pigs tuberculosis has a tolerably definite period of incubation: from n to twenty-one days. When a minute fragment of tuberculous is introduced into the anterior chamber of a white rabbit's eye through sion in the cornea, the slight reaction which arises after the operation subsides, provided that the tubercle is perfectly fresh. The fragment w be seen through the transparent cornea, and it diminishes in size day until it may altogether vanish. Then, at the end of the period or three weeks, an eruption of small transparent grey granulations on the iris. Afterwards they caseate, and a destructive inflammation whole eyeball often results. Ultimately the animal may die of a miliary tuberculosis, in from five weeks to three months, or later; diffusion of tubercles may be limited to the lungs or to the peritoneal ; or, lastly, there may be no development of them in any organ outside eball.

complete the account of the experimental study of tuberculosis in the animals, we must describe two sets of observations which illustrate ls by which the specific virus of tubercle may be supposed to invade man body.

The disease of cattle which is known in Germany as *Perlsucht*, England as "the grapes," is now generally admitted to be al with tuberculosis, although the morbid appearances, as seen by aked eye, differ considerably from those with which we are r in man. It is a chronic affection, which may run on for years without impairing the animal's health. In fact, milch cows almost the only instances in which symptoms appear during life: re chiefly cough and wasting. It appears that animals thus affected en sold to the butcher, so that besides the risk of drinking their milk the early part of their illness there is the further risk (at least to

t a meeting of the Pathological Society on December 4th, 1883, Dr Fox stated that equest the experiments referred to in the text had been recently repeated by Dr Williams. The details of this series of experiments were stated, with the precau- gainst accidental infection; the results were absolutely negative.

the poor) of eating the diseased tissues, perhaps concealed in uncook sausages. The frequency of Perlsucht may be judged from the fact that Augsburg 2 per cent. of all cattle slaughtered were proved to be tuberculo and of cows as many as 5 per cent. In Paris bovine tuberculosis l become less common of late (1889). Its experimental propagation in otl than bovine animals has been studied by Gerlach, Klebs, Orth, and othe Not only have pigs, lambs, rabbits, and guinea-pigs been successfully infect by the milk from diseased cows, but a like result has been attained feeding animals with portions of the affected tissues removed after the co were slaughtered, and also by inoculating them beneath the skin with su materials. Klebs found that even boiling the milk did not, in two expe ments, render it inert.*

A further step in the investigation has been taken by Bollinger, w thinks that a broad distinction can be drawn between the Herbivora a the Carnivora as regards their liability to be affected with tuberculo through the alimentary canal; he supposes that flesh-eating animals secr a gastric juice which has a more active power of destroying the bacill Man, as an omnivorous creature, ought to occupy an intermediate positio

One is strongly tempted to find in these experiments an explanation the frequency of mesenteric disease in children, in whom milk forms important an article of diet. The following observation, recorded in 18 by Dr. W. H. Spencer, of Clifton, may perhaps bear upon this point. M than twenty boys in an industrial school fell ill of what was supposed to enteric fever. Most of them recovered, but four died; and in each fa case the organs were found to be full of tubercles. In every instance, ho ever, caseation of bronchial glands was present, which could scarcely ha occurred in the three or four weeks of the boys' illness.

(2) The other set of experiments was made by Tappeiner, in order determine whether tuberculosis could be induced in animals by the inha tion of the sputa of phthisical patients diffused in the air as a spray. I method was to employ six grammes of the sputa at a time, and the inha tions were continued for six hours a day during fourteen days in successi Dogs were used in this inquiry, and the earliest period at which tuberc

* Dr Creighton has recorded several cases in which he believes that *post-mor* appearances found in the human subject justify the conclusion that the disease was deri from the cow. His point of view, however, is that Perlsucht, or bovine tuberculosis not identical with the tuberculosis which chiefly occurs in man, and that it will hencefo be possible to separate by certain characters cases having such an origin from those wh may be regarded as proper to the human species, and he cites Gerlach as having sta that in some of the animals that were infected by him from the cow the "peculiarity Perlsucht was unmistakable." In addition to there being grey translucent tubercles, l those which are seen in man and in apes, the characters on which Dr Creighton relies the following:—The peritoneum and the pleura present round or oval nodules, of the s of lentils, sometimes pendulous, sometimes connected with one another by fibrous co sometimes flat and cohering together so as to resemble the duckweed on the surface of a po In the lungs there may be soft, whitish masses up to the size of a walnut, some rou others wedge-shaped and like infarctions. These organs may also have in their inter closed smooth-walled cavities, giving them somewhat the appearance of a crumpet. It is be observed that most of the cases in which Dr Creighton found such lesions occurred, i in children, but in adults of all ages. Now the prospect of our being able in futu to distinguish from other cases of tuberculosis in man such as are of bovine orig does not seem to be promising. But even if what Dr Creighton terms the "stru tural mimicry of infection" really goes so far as to give special characters to bovine cas it yet would not follow that the two diseases are fundamentally distinct. Koch fou his bacillus in ten cases of Perlsucht, chiefly in the interior of giant-cells surroundi the calcified nodules in the lungs, but also in the bronchial and even in the mesente glands.

were found in the lungs after the commencement of the inhalations was on the twenty-third day. In no single instance were the results negative.

Bearing of the above experiments on human pathology.—If we now turn to consider the bearing of these experimental observations upon tuberculosis, as it is seen in the human subject, we shall be obliged to conclude that they are only applicable within narrow limits.

There are two questions to be discussed which must be carefully kept apart, although they are often confounded together. One concerns the ætiology of tubercular diseases, or invasion from without, the other refers to the way in which acute and chronic tubercular lesions spread through the patient's tissues, or infection within the body.

(*a*) *The origin of tuberculosis.*—The view that infection from without is the essential part of the ætiology of tubercular diseases is inconsistent with clinical observation. There is the clearest evidence that phthisis does not ordinarily spread from patients to other patients, or to relations, nurses, or medical men. On the other hand, it has been proved that its prevalence is greatly augmented by such conditions as overcrowding and defective ventilation, and also by exposure to cold and wet, and by dampness of soil. Overcrowding and defective ventilation may be supposed to act by lowering the general health. But it is difficult to see how exposure to cold and wet can induce the disease except by their liability to set up catarrhal affections of the pulmonary and bronchial tissues; probably this may also be the way in which dampness of soil acts. Moreover we shall find reason to believe that the affections of the lungs due to inhalation of dust are really tubercular, and not (as is generally taught) purely inflammatory. Again, hereditary transmission plays a most important part in the ætiology of phthisis.

It is not easy to frame a theory of the ætiology of tubercular diseases which shall accord with these facts. There are probably great differences in the *susceptibility* of different persons to the virus, or, perhaps more correctly speaking, in the *resistance* which they offer to it. Moreover, notwithstanding the enormous mortality from phthisis and the affections allied to it, this resistance must be far greater in the human subject than in the rabbit or in the guinea-pig; for one must remember that those who die of tubercular diseases have commonly lived for several years, during which time they have most likely been exposed to tubercular infection again and again. It is also necessary to admit that this resistance varies in the same individual at different periods, and that it is not absent even among those in whose families phthisis is hereditary. Otherwise we should find that when one member of such a family was attacked by the disease it would quickly spread to other members. Now, the fact is, that even if several brothers and sisters die in succession, their illnesses are usually separated by considerable intervals of time.

The greatest difficulty is to understand how an affection produced by the growth of a bacillus can bear any relation to causes which might be expected to set up a simple inflammatory process. One possible solution is that the microbe may be so widely diffused that it is continually finding access to the body, but fails to implant itself until the local resistance of the tissues happens to be lowered by inflammation. But this seems to be inconsistent with Koch's statement that the bacillus cannot grow unless the temperature is above 86° F. Cohnheim thought that the bacillus may remain latent in the body for an indefinite time until some accidental cir-

cumstance calls it forth into active growth. He even imagined that hereditary transmission of phthisis means the direct transference of tubercular virus, either in the spermatozoa or in the ovum, as virus transmitted in the case of the *pébrine* of silkworms. There is perhaps third possibility, namely, that under certain unknown conditions the occ rence of what is at first a simple inflammatory process may cause mic phytes already existing in the body to acquire infective properties that th did not previously possess, and to convert the simple inflammation int tuberculous one.

(*b*) *The spread of acute tuberculosis.*—Let us now pass on to consider what extent the recent experimental observations upon tubercle thr light upon the *mode of spreading of tuberculous lesions within the human bo*

Acute general miliary tuberculosis, with its myriads of minute cent of tissue-irritation, is hardly explicable in any other way than by the semination throughout the blood-stream of minute particles of the tubercu virus in the form of bacilli or of their spores. Buhl himself failed to fin caseous mass which, on his view, could have been the starting-point general tuberculosis in no fewer than thirty out of three hundred cas and this notwithstanding that he accepted as satisfactory such lesions a small embolic plug in an organ, or a degenerating purulent exudation i serous cavity. The probability now seems to be that the real starting-po is often a very recent patch of tubercles which happens to be so situa that an infection of the blood-stream is a necessary result of its presen Thus, for instance, Weigert discovered, in many instances of acute tube losis, a caseous mass of tubercle, growing into the interior of a pulmon vein, by extension from the pleura, or the bronchial glands, or the med tinum. In quoting this observation, Cohnheim alludes to Ponfick's case tuberculous infiltration of the wall of the thoracic duct as another poss source of blood infection : this, however, is a very rare lesion.

But it must not be supposed that the growth and the distribution tubercles, when they are ever so widely scattered throughout the body, regulated solely by the way in which the bacilli are disseminated thro the blood-stream. There seems to be a curious difference between artificial tuberculosis of animals and the disease as it is seen in men w respect to the distribution of the tubercles. In the former affection they found beneath the pleural surface more abundantly than in the substa of the lung, and Dr Sanderson speaks of them as having sometimes the fo of blunt cones. In both respects artificial tuberculosis resembles the other great examples of tissue infection by the blood—pyæmia and malign tumour. A third point of similarity between the three affections is that all of them the liver forms a conspicuous seat of the secondary lesions. artificial tuberculosis Dr Sanderson seems to have found tubercles in liver more often than in the lungs, but Dr Wilson Fox observed li difference between the two organs in this respect. On the other hand, the general miliary tuberculosis of man the liver is much less frequently less obviously affected than the lung, although no doubt the microsc shows that hepatic tubercles are far more often present than used to supposed. In the lung itself the tubercles are not more numerous bene the pleural surface than elsewhere, and they do not possess the conical wedge-like form.

Further, in many if not in most cases of acute tuberculosis, the tribution of tubercles in the lung is such as to show clearly that the p

clivities of the affected tissues play a part in determining it. In the upper lobe the tubercles are more abundant, larger, and more advanced in their development than in the lower lobe. They often gradually diminish both in number and in size from the apex downwards. They may be quite caseous above, grey and semi-transparent below. In other words, even when the virus is carried by the blood-stream to all parts of the lungs at once, the result is an affection which bears some resemblance to an ordinary local phthisis so far as concerns the distribution of the tubercles. This fact helps to explain what would be otherwise unintelligible—the frequent limitation of acute tuberculosis to a single organ or tissue. In tubercular meningitis, for example, it often happens that not a single tubercle exists anywhere except in the cerebro-spinal membranes; and yet one can hardly doubt that bacilli in immense numbers must have been distributed by the blood equally to all other parts of the body.*

(*c*) *The distribution of chronic tuberculosis.*—In chronic tubercular affections the mode of spreading seems to be by the gradual invasion of the lymph-channels from point to point. Among the most striking appearances are the lines of progressive thickening and caseation, with or without the formation of distinct tubercles, which run in the course of the sub-peritoneal lymphatics from the floors of intestinal ulcers. It does not often happen that the process of local infection fails to respect an anatomical boundary line between two widely different tissues; but in children the present writer recorded two or three cases in which a continuous cheesy mass, with a festooned growing border, extended from bronchial glands directly through the lung-substance, and one similar instance in an adult. A still more striking case seems to have been met with by Buhl. A child had caries, with caseation, of the lower dorsal vertebræ; the pleura became adherent, and the ulcerative process spread continuously into the base of the left lung. And Grancher ('Arch. de Phys.,' 1878) mentions a case of tubercular peritonitis, in which tubercles penetrated the diaphragm and infected the pleura covering the inferior surface of the lung, without adhesion having taken place. But in most instances of chronic tuberculosis the special proclivities of the several organs and tissues play a most important part in determining and in limiting the distribution of the tubercles. It is indeed difficult to see how the bacilli can fail to enter the blood-current in greater or less numbers in any of these cases; and if so, the resistance of the tissues in general can afford the only reason why lesions are not always found scattered irregularly through all parts of the body. Instead of this, the fact is, as the writer pointed out in the 'Pathological Transactions' for 1874, that several distinct varieties of chronic tuberculosis can be recognised, each of which has peculiarities of its own, in regard to the organs and tissues attacked. A parallel can generally be found for the distribution of each of these several varieties in the similar distribution of some other non-tuberculous disease; so that one is forcibly reminded of what Darwin calls the "correlated variation" of organs.

The following are the chief local varieties of chronic tuberculosis:

1. Pulmonary phthisis is very commonly accompanied by tuberculosis and ulceration of the air passages (especially of the larynx), and of the intestine (especially the lower end of the ileum). Some observers think that the affection of the lungs directly causes the lesions in question, the larynx being

* It is, however, possible that, in this very acute disease, infection is carried through the sub-arachnoid space by the movements of the cerebro-spinal fluid.

infected by the sputa which pass over it, and the ileum by the sputa w
are swallowed, and there is some experimental evidence in support of
view. But it is worthy of notice that, in enteric fever likewise, ulcer
of Peyer's patches and the solitary follicles occurs together with ulcer
over the bases of the arytænoid cartilages; and also that, in cases of
pneumonia, ulcers are apt to form at the same spots within the larynx.

2. Dr Wilks long ago pointed out that tubercle sometimes attack
pleura, the peritoneum, and the pericardium, without affecting any of
solid viscera. As already remarked (p. 60), the same distributio
observed in some cases of simple inflammation.

3. Tuberculous disease which began in one kidney not only spreads
extraordinary extent by continuity along the genito-urinary mucous memb
and the corresponding submucous tissue, but is also commonly attended
like affections of the opposite kidney, and (in the male) of one or both o
testes. If infection occurs by the blood-stream, tissue proclivities
surely play an important part in the result. Have we not an analogous
in the occurrence of a "sympathetic" orchitis in a case of renal calc
There is also a relation between tuberculous disease of the kidney and
disease of the lumbar vertebræ at the same level: but in this instance d
extension probably occurs.

4. Addison's disease of the adrenals (the tuberculous nature of w
is well ascertained) is sometimes associated with spinal caries at
same level, which, indeed, probably precedes the adrenal lesion in poi
time. The writer saw two examples of a still more remarkable conne
between solitary tubercle of the brain and tubercle affecting the adrenals i
form of one or more scattered cheesy masses. Virchow taught that gliom
growths sometimes occur simultaneously in the brain and in the adrena

5. Tuberculous disease of the lymph-glands often spreads widely thr
out the body, and this not merely by obvious continuity, for it may
groups of glands widely distant from one another. It is also very gene
attended with tuberculosis of the spleen. A precisely similar associ
is seen in Hodgkin's disease.

6. Several of the joints are often affected by tuberculous disea
succession, without any corresponding affection of other structures.
again, one is reminded of the frequent limitation of pyæmia to joi
another illustration of the effect of local proclivities in determining
locality of lesions, even when they are due to infection of the blood.

It is still an open question whether multiple tuberculous affectio
the same subject are not sometimes independent of one another.
author was once much struck by finding in an infant, six months
two large masses of tubercular glands, entirely distinct from each other,
in the chest, the other in the abdomen; and we not infrequently meet
cases in which the distribution of tubercular lesions seems to be capric
without any one of them being obviously of older date than the rest.
we have found phthisis associated with a tuberculous affection of one t
and of the vesiculæ seminales in a man, and with a similar disease o
uterus and of one adrenal in a woman. In a child who died with a t
culous growth in the cerebellum there was a mass of caseous gland
the abdomen. Similar instances might be multiplied indefinitely. B
is possible that all, or any of them, may have been due to chance infecti
the blood-stream, the virus being carried to a spot where it met wit
resistance sufficient to check its action.

Historical retrospect.—It is interesting to ask whether any of the writers in the early part of this century expressed opinions approaching those which seem to accord best with our present state of knowledge. As regards the doctrine of the unity of phthisis, we need not look beyond Laennec. But his conceptions as to the relation between that disease and inflammation may almost be summed up in the statement that inflammation might occasionally be excited by the irritation caused by a numerous crop of tubercles. Nor can we assign a large share of credit to Reinhardt. It is true that, in 1850, he identified grey tubercle and grey infiltration with inflammatory processes, but, on the other hand, he maintained that yellow or cheesy tuberculous matter never arose out of the grey, but was merely inspissated pus. Among English physicians, Thomas Addison taught that inflammation constitutes the great instrument of destruction in every form of phthisis, or, as he put it in 1841, that pneumonic phthisis and inflammatory tubercle are identical (Addison's works, pp. 40, 64). Prof. Alison, of Edinburgh, in his 'Outlines of Pathology,' published in 1844, stated that "in certain constitutions, tubercles and all their consequences are direct effects of inflammatory action." Dr C. J. B. Williams classified grey and yellow tubercles as cacoplastic and aplastic varieties of coagulable lymph, "differing from the normal plasma, not in kind, but in degree of vitality and capacity of organisation."

At present we should best perhaps put the case thus. Tuberculosis is a general infectious disease, derived, as a rule, but perhaps not invariably, from one or more local caseous masses the product of inflammation. The primary and the secondary products are both inflammatory, of the kind described as *granuloma* (p. 60), and both constantly contain a specific bacillus, which may be reasonably regarded as the contagium vivum to which the morbid process is due. But besides the granuloma-growth there is also ordinary secondary inflammation, purulent, ulcerative or fibroid, which in most cases follows as a complication.

AGUE*

Ye ben full cholerik of complexion;
Beware the sunne in his ascension
Ne find you not replete in humours hote;
An if he do, I dare well lay a grote,
That ye shall have a fever tercian,
Or an ague that may be your bane.

CHAUCER.

Intermittent Fever—Its pathology, history, and distribution—Incubation—cold, hot, and sweating stages—Regular and irregular varieties—Rem Fever—Malarial cachexia—The spleen, liver, and other organs in ag Melanæmia—Nature and laws of malarious contagion—Microphytes Microzoa—Diagnosis—Treatment.

WE have now to discuss a disease which differs from the specific fe in some important respects. In its most typical forms its course is i rupted by definite intervals of apyrexia, so that, instead of appearing continuous fever, it consists of a succession of independent paroxysm "fits," which recur with marvellous regularity. For such cases the nam *intermittent fever* is commonly employed. But they cannot be separ from others which are due to the same cause, although the subsidenc the pyrexia is only partial, so that they are said to be examples of *rem fever*. The English term "Ague" includes both of these as well as sub nate varieties.

The poison which excites ague has long been known as *malaria* (*i. e.* air). It enters the human body from without; but, unlike the virus of other specific diseases, it never passes directly from one person to anot In other words, ague, though in a broad sense one of the "infective" dise is not "contagious," nor even "infectious" in the narrow sense in which epithet is generally employed. Vaccinia and Syphilis are contagious person to person, *i. e.* inoculable by direct contact, but not infectious, *i. e* conveyed to persons at a distance. Typhus and Measles are contagious infectious, *i. e.* conveyed from person to person by direct contact and distance. Cholera probably, and Enterica almost certainly, is not conv through the air, nor yet by direct contact, but only by means of a viru contagium which is developed after its discharge from the body. Ague is conveyed from person to person at all, but from a place, probably from or water, to persons. According to the bacterial theory of specific disease proved in a few instances, more or less probable in several, and without evidence but analogy in the rest (cf. *supra*, pp. 12, 13)—the microphyt Vaccinia and Syphilis can only be transferred by direct inoculation into blood or lymph; that of Typhus, Plague, or Scarlatina, can be carried by air or on fomites, and introduced through the stomata of the air-vesicles

* *Synonyms.*—Intermittent Fever, Periodic, Malarial, Miasmatic, Paludal or M Fever.—*Fr.* Fièvre intermittente, palustre, paludéene; *Germ.* Wechselfieber, Sumpf The English word "Ague" is derived from Fièvre aigue, *Febris acuta.*

the lymphatics by means of the inspired air; that of Enteric Fever is harmless when first discharged, but after certain stages of growth outside the body of the host may enter the alimentary canal, and there reproduce its kind, like the ovum of a tapeworm or a trichina; and, lastly, the microphyte of Ague is not solely parasitic, but lives and grows outside the body, and possibly, like many parasitic worms, can only complete its cycle of development when it is at one stage parasitic and at another free.

Descriptions of the different varieties of ague are to be found in the writings of Celsus and other ancient writers; for various forms of intermittent fever were, and still are, the commonest disease in Greece and Italy. The first observer who took a clear view of malaria, as a noxious effluvium given off by marshes, appears to have been Lancisi, in a work published at Geneva in 1716.

Down to a very recent period intermittent fevers were common over the greater part of England, particularly in London and in the counties of Lincoln, Cambridge, Essex, and Kent. Drainage has much limited the extent and severity of the disease, but it is still met with along the banks of the Thames, and we see more or less marked cases of malarial cachexia among our out-patients from Southwark, Rotherhithe, and Deptford.

The *geographical distribution* of malarial disorders has, however, so important a bearing on their ætiology that it is best deferred till later.

It is a curious fact that malaria appears to be almost without influence upon domestic *animals*, at least in those countries where its effects have been most studied. In Italy, however, a few instances have been recorded in which horses or oxen have had intermittent attacks of fever, or have suffered from cachexia attended with enlargement of the spleen.

Incubation.—As a rule, those who are attacked by ague have lived for a time in a malarious region, and seem to succumb to repeated doses of the poison, rather than to take the disease on any particular occasion. When a single exposure has taken place, an interval of from six to twenty days generally elapses before the paroxysms begin. Dr Maclean, however, in 'Reynolds' System,' mentions the case of three German missionaries, fresh from Europe, who passed a night with an English officer in an unhealthy spot, at the foot of the Segoor Pass in India; next morning they pursued their journey, but within less than twenty-four hours three out of four of the party were stricken with fever. And Hertz, of Amsterdam, writing on this subject in 'Ziemssen's Cyclopædia,' declares that on several occasions, having purposely placed himself in a marshy ditch at a time when it was drying up, he has been attacked within half an hour by giddiness, shivering, nausea, and other symptoms, which ended in a slight paroxysm of fever a few hours afterwards. On the other hand, it is well known that the onset of the disease may be much longer delayed. Sailors have fallen ill upon the open sea, weeks or months after leaving a port infected with malaria. In London the Irish poor often apply for hospital relief in the spring, on account of ague which, although only just declared, must have been contracted during their autumnal migrations into Kent for hop-picking.

Premonitory symptoms.—Among strangers who have recently entered a malarial district, the occurrence of a regular paroxysm is in most cases the earliest symptom of ague. But, according to Hertz, when the patient has been living for a long time in the same place, a prodromal stage may sometimes be observed. It lasts a week or more, and consists of vague

malaise, fatigue, headache, and pains in the limbs, in association with nau
loss of appetite, and other symptoms of disordered digestion. There may
be occasional sensations of chilliness, alternating with slight flushes of h

The attack.—The ague-fit itself is divided into three periods, which h
long been known as the "cold," the "hot," and the "sweating" stages.

1. The *cold* stage begins with the patient's feeling tired, weak,
listless. He yawns and stretches out his limbs. He complains of an
comfortable sensation at the epigastrium, of headache, or of giddin
Before long, these symptoms merge into those of shivering or *rigor*.
experiences a chilly feeling along the spine, which quickly spreads all
him. He shudders, his teeth chatter, his knees knock together, his wh
frame may be so violently agitated that the bed into which he has c
shakes beneath him. His voice is feeble and his speech is interrupted
the quivering of his lips. At the same time his appearance undergo
remarkable change. He grows pale, his features shrink, his ears and
and finger-tips turn livid, his skin becomes dry and rough—*cutis anserin*
"goose-skin." Yet the thermometer, as De Haen discovered more th
century ago, shows that in the central parts of the body the temperatu
not lowered, but raised several degrees above normal. It must not, howe
be supposed that the patient's sensations deceive him as to the conditio
the surface, and especially of the more distal regions. One has only to
his nose or his fingers in order to assure oneself of the fact that they
really cold; and a surface-thermometer, or an ordinary thermometer hel
the patient's hand, will remain far below the natural standard, while in
axilla, the mouth, or the rectum it shows marked pyrexia. The tempera
begins to rise before any symptom of the onset of the ague-fit is experie
by the patient. At first its increase is gradual, but with the develop
of the rigor it makes a sudden leap upwards, perhaps passing through
or five degrees Fahrenheit in the course of an hour.

The patient during this stage feels dry and parched; his tongu
white; he often complains of nausea, and sometimes vomits. His puls
quick and small, and may be irregular. His respiration is short, hurr
and distressed. His urine, although pale, is often very irritating, so
Maclean has found it desirable to administer bicarbonate of potass
even tincture of opium for the relief of this symptom alone. Wa
spoke of the urine as scanty, although passed frequently; but the car
measurements and analyses of Redtenbacher and of Ringer have shown
it is in reality considerably increased in quantity, when compared with
amount passed each hour during the apyretic interval, and that there is
a marked increase of the urea and chloride of sodium. The excess, b
of the water and of these solid constituents, not only lasts throughout
whole length of the fit, but begins before the patient feels chilly.

2. The *hot* stage gradually succeeds. The patient ceases to shiver,
begins to experience flushes of heat about the face and neck, which presen
diffuse themselves over his body. But for a time, if he attempts
throw off the bedclothes he has piled over him, slight rigors reapp
At length, however, even the extremities become hot and glowing.
aspect is now again altered. His face is flushed, his eyes are injected,
skin becomes smooth and turgid. The temperature, as measured by
thermometer, still continues to rise for some time, and reaches 104°, so
times 106·5°, or even higher. The surface of his body, even of the di

parts, now feels pungently hot to the hand. Evidently, there is not only an increased generation of heat, but it is now freely carried to the periphery, and abundantly dissipated. The pulse is not only rapid, but full and throbbing; and the breathing is deep as well as frequent. The patient complains more than ever of headache, which is now of a throbbing character; and he is exceedingly restless and uncomfortable. The urine during this period is described as differing in appearance from that of the cold stage, being now high coloured and concentrated; and both Watson and Hertz speak of it as passed in but small quantity. There is often an eruption of herpetic vesicles upon the lips, or the nose, or the tongue.

Albumen in the urine, accompanied by casts and even by blood, has been observed by Griesinger and Hertz, but this is a very rare complication.

3. The *sweating* stage follows in its turn. The surface gradually becomes soft; then a little moisture breaks out upon the forehead and the face, and before long the whole skin is bathed in the most copious perspiration, which saturates the patient's linen and bedclothes. It is said that the thermometer sometimes continues to rise for a little while, so that the maximum temperature may occur during this, and not during the hot stage; but, as a rule, the pyrexia begins to decline from the moment when the skin ceases to be dry. The fall is at first slow, but after half an hour or an hour it becomes more rapid, and goes on until the normal temperature is reached. According to Wunderlich, this takes place by a series of steps, there being a drop of from one fifth to one third of a degree Fahr. about every fifteen to thirty minutes. All observers speak of the urine during this period as differing from that passed in the previous stages in showing a thick deposit of lithates. The breaking-out of perspiration is attended with complete relief to the patient; he loses his pains, the throbbing of his arteries ceases, he is no longer tormented with thirst, and his tongue becomes moist. After a time he perhaps falls asleep; and, when he wakes, he feels perfectly well, except that he may be somewhat exhausted.

Recurrence.—The subsidence of the ague-fit or paroxysm by no means brings the disease to an end. As we have already stated, its most remarkable feature is that precisely similar attacks are repeated again and again, recurring at perfectly definite intervals. Some patients have a fit every day; the ague is then said to be *quotidian*. In other cases it occurs every other day; and this form of intermittent fever is styled *tertian*, for according to the Latin way of counting it falls on every third day, or as we should say on every other day. If there are two entire days between the paroxysms, the disease is a *quartan* ague. In other words, an interval of about twenty-four hours characterises a quotidian, one of forty-eight hours a tertian, and one of seventy-two hours a quartan. Nor does this exhaust all the possible varieties. In some cases in which there is an attack every day, each paroxysm differs in severity or duration or in the hour of its occurrence from that of the preceding day, and exactly resembles that which occurred two days before. Thus a tertian type is clearly perceptible, and the affection is then called a *double tertian*. So, again, it is possible to have a *double*, or even a *treble quartan* ague. Or a patient may have two ague-fits one day, and a single fit on the following day, this series being regularly repeated; and one may then suppose that a quotidian and a tertian are combined. Such a case constitutes the ἡμιτριτᾶιος of Celsus, or, as subsequent Latin writers termed it, a *semitertian*.

The return of the paroxysms of ague is often so regular that it can foretold with absolute certainty; but even then the hour at which the begin is not always the same on each occasion. Sometimes it is earlier, e time by a constant difference; sometimes it is later. In the former case, disease is said to *anticipate;* in the latter case to *postpone.* Whereas one is a sign that the severity of the case is on the increase, the other i cates that it is becoming milder. Such variations may even bring abou change of type; thus an anticipating tertian may at last pass into a quotid and a postponing quotidian may be gradually converted into a tertian. So times the attacks of a quotidian fever are so prolonged that before the sw ing stage of one fit is over the cold stage of the next one begins; the dis is then said to be *subintrant.*

With regard to the relative severity of the three principal forms of a writers state that quartans are peculiarly obstinate.* This variety, whic comparatively rare, is most apt to occur in the autumn; and it has l been well known that autumnal agues are worse than those contracted the spring.† Hertz states that in the tropics the disease never assumes quartan type; and yet ague is certainly more severe there than in the t perate zone. In hot countries the quotidian appears to be the most comm form of ague, whereas in temperate climates the tertian is more frequen

There are some other differences in the course of the several varieti intermittent fever. Thus the fits of a quotidian are said generally to s during the morning, those of a tertian at about noon, those of a quartan later in the day. It is stated that the average duration of a paroxys quotidian ague is from ten to twelve hours, that of a tertian from six to e hours, that of a quartan from four to six hours, but that in the last-na variety the cold stage is more prolonged than in the others.

Subsequent course.—Intermittent fever appears to be never directly f Its duration is very variable. Sometimes, even though the patient rem in the place where he acquired it, it ceases after a few paroxysms. In o cases it comes to an end when a change of season reduces the malaria state of inactivity. In others, again, ague recurs for an indefinitely l period, until the victim is compelled to remove to some other dist There is always a strong tendency to relapse, not only under a f exposure to the ague poison, but even without it; and it is a remark fact that, whether the type be tertian or quartan, the paroxysms o relapse often occur on those very days on which they would have been if the disease had gone on without break from the first. Graves has rel an instance of quartan ague which he watched for twenty-seven months, in which the periodic rate was maintained through thirteen out of sixt intervals, some of which lasted more than two months. A similar obse tion had previously been made by Dr James Gregory in the case of a relati who had marked on an almanac the days on which the fits of a tertian a might be expected to recur, and who found that for a long while they so whenever the east wind blew. Since the introduction of the ther meter into clinical practice, however, it has been shown that there is so times a distinct rise of temperature at the proper time for the paroxy although the patient himself may be altogether unconscious that anythin

* Nevertheless the Latin couplet runs:

Pro febre quartana
Rara sonat campana.

† "An Ague in the spring is Physick for a King." Yet James I died of a tertian f in March.

the matter with him. Ringer states that in spite of the absence of all febrile disturbance the periodicity of the disease may be indicated by the voiding of urine undue in quantity, and containing an excess of urea.

Varieties.—Ague-fits are not always characterised by the whole series of phenomena described above. Sometimes the cold stage is absent, sometimes the sweating stage. It is said that sometimes the usual order is reversed, as in a case cited by Watson from Maugenet. The patient in question was always first attacked with profuse sweating; then he became dry and hot; finally he felt cold and had distinct rigors.*

In certain exceptional cases the paroxysms differ altogether from those which are ordinarily seen, and they may be attended with great danger to the patient's life, so that both Trousseau and Hertz classify them apart. The strangest form is that which has been termed *syncopal*, in which there is a condition of suspended animation, so that the patient runs a great risk of being buried alive. Trousseau relates two instances of this. One occurred in a station-master on the Avignon Railway. He had been subject for some time to paroxysms of intermittent fever, and had repeated fainting fits; once he became pulseless, was supposed to be dead, and was carried to the mortuary. After some hours a servant happened to enter the place, and found him groaning; he was therefore taken back to his bed, and under large doses of cinchona he regained his health. It is singular that Dr Chauffard, the physician who observed this case, also met with another one of the same kind. A man had fallen into a faint, was taken for dead, and his face was covered with a sheet; Dr Chauffard, however, detected slight movements of his heart, although the radial, axillary, and carotid arteries had ceased to beat; he immediately administered a quinine enema, and the man was saved.

Scarcely less remarkable is another variety, which is attended with *coma*. Hertz says that instances of it were from time to time sent into the hospital under his care for apoplexy, and Graves placed on record a case in which he made this mistake. A gentleman awoke at about 4 a.m. with sensations of malaise, chilliness, nausea, and headache. After an hour he became extremely hot, the pain in the head was intense, and he passed from a drowsy condition into one of complete coma, with deep snoring, so that he "appeared to be labouring under a severe apoplectic fit." He seemed to derive much advantage from bleeding and other remedies, and in the evening he was perfectly well. The day but one after the same symptoms returned, and were removed by the same treatment; but when a third attack came on Graves saw that it was an example of *tertiana soporosa*, and cut it short by large doses of quinine.

In other cases it is said that epileptiform convulsions or tetanic spasms have been present, or that the symptoms have simulated those of hydrophobia. It even seems that the paroxysms of ague may imitate various abdominal or thoracic diseases—cholera, dysentery, peritonitis, pneumonia, pleurisy, are among those enumerated by writers. Hertz tells us of a man who was attacked at two o'clock in the morning with shivering and pain in the left side; at eight there was a distinct pleural friction sound, but twenty-four hours later he was perfectly well. However, on the following night, at twelve o'clock, all the symptoms returned, and by nine the friction sound was more evident than before; at four in the afternoon he was sweating and

* "'Tis but an ague that's reversed,
Whose hot fit takes the patient first."—*Hudibras*, part iii, canto i, 653.

free from pain; full doses of quinine were therefore prescribed, and he no further attacks.

With regard to these cases, and also to those of malarial syncope coma, we must be cautious in accepting them as mere varieties of ague it is an established fact that in districts where ague prevails all kin diseases assume a more or less distinctly intermittent character.

The same hesitation seems to be necessary before deciding the natu what have been termed *masked agues*, in which the paroxysms are state be of the most diverse descriptions, and attended with scarcely any fe disturbance, or even with none at all. The most important of these is ralgia, occurring especially in the region supplied by the first division o fifth nerve (and thus often called "brow-ague"), but sometimes assumin sorts of other forms. Not only have cases of sciatica and of other neur affections of the limbs been attributed to ague, but the same view has taken of painful affections of the mammary gland or of the testicle, and of alarming seizures attributed to cardialgia or to neuralgia of the vagi it is hard to say what evidence in favour of such interpretation wou conclusive. Obviously it is not enough to show that the attacks recur a certain degree of regularity, and pass off under the influence of quinin as much as this may be said of almost every kind of neuralgia. Th servance of a tertian or quartan type would no doubt be very significant Hertz says that masked agues are usually quotidian. It must be bor mind that the more extensive a man's experience in regard to intermi fevers, the more likely is he to be led astray by tradition, or by per bias, until he has recourse to the same convenient hypothesis for the sol of every obscure and indeterminate affection, such as occur frequently en in clinical practice in districts where no ague is met with. What, for exa is to be said of the case of Dr Macmichael, cited by Sir Thomas W who "caught an ague many years before his death by sleeping on a somewhere in Greece, and was ever after subject to occasional attac periodic headache and other aguish symptoms, for which he was oblig have recourse to bark or arsenic"? And what value can be assign Trousseau's statement that there are cases in which insomnia, unaccomp by fever and not preceded by rigors, recurs every two or three nights these cases are of the nature of masked ague, and that they are cur similar treatment?

Remittent fever.—Malaria is not limited in its effects to the producti the different forms of intermittent fevers. In hot countries, and d the hot seasons of the year, even in temperate climates, it causes f which are either continuous or interrupted only by *remissions*, interv which, instead of there being complete apyrexia, there is only a p lowering of the temperature and of the pulse, with some degrees of ment of the other symptoms. For cases of this kind the technical na *remittent fever*, but in India a common term for them is *jungle feve* another is *bilious remittent fever*. They are, in fact, characterised by a of phenomena which do not belong to the milder effects of the marsh p as well as by the absence of some of the more distinctive features of ord ague. Thus the cold stage is slight and ill-defined, and the hot sta followed by little or no sweating. The remissions themselves, which begin about midnight or in the morning, and which last for twelve or longer, may be so slight as to require careful clinical observation for

detection. A feeling of oppression at the epigastrium is present from the commencement of the patient's illness, and vomiting is usually a marked symptom throughout its course. The vomited matters may at first consist of food, but afterwards a watery fluid is ejected, often in surprising quantity, and it may ultimately become greenish-yellow, brown, or even in rare cases black. Headache is generally a prominent symptom, but not delirium.

In the worst cases, in which the disease is said to be of an "adynamic" character, the patient rapidly becomes very prostrate and insensible, his skin is yellowish and covered with petechiæ, his tongue is black, his teeth are covered with sordes, and hæmorrhages may occur from the nose, the mouth, and the limbs; or hæmaturia may be a prominent symptom. French writers have described such cases under the title *fièvre bilieuse hématurique.* Maclean, from whose description of remittent fever in 'Reynolds' System' most of these details are taken, says that except in such cases the urine seldom contains albumen; it is often abundant, and sometimes pale.

The duration of remittent fever is said to be generally from five to fourteen days. Even its worst forms ought, according to Maclean, to do well in most cases, if seen early and skilfully treated. Sometimes it ends with a critical perspiration, sometimes it subsides gradually, sometimes it passes into one of the regular types of intermittent fever. Death seldom occurs before the seventh or the eighth day, a point which may distinguish this disease from yellow fever in countries where they both prevail.

Typho-malarial fever is a name used in the United States for cases which are probably not a separate form of disease, but enteric or other specific fevers occurring in persons subject to malaria. See Dr Johnson's paper ('Trans. Assoc. of American Phys.,' p. 40).

Malarial cachexia.—In persons who have suffered from protracted ague, or who have had repeated relapses, a chronic condition of ill-health is apt to develop itself which is known as malarial cachexia. A precisely similar condition is not infrequently seen in those who have lived for a long time where the disease is prevalent, even though they may have never had any febrile symptoms. One of its most characteristic signs is a peculiar earthy sallow complexion; another is a damp, clammy state of the skin, especially noticeable in the hands. The patient is depressed in spirits, and wakes unrefreshed by the night's sleep; he often suffers from giddiness, noises in the ears, tingling in the hands and feet, pains in the lower part of the back, palpitation, loss of appetite, a furred tongue, and digestive disorders.

In all cases of this kind enlargement of the *spleen* can be made out on percussion. Very often the organ is to be felt beneath the left costal cartilages, extending down to the umbilicus or even lower. Sometimes it forms a conspicuous tumour, which used to be called an "ague-cake." Although in some very rapidly fatal forms of malarial fever the spleen has been found of unusual size, the rule appears to be that it only swells with every paroxysm of the disease, and subsides more or less completely during the intervals. It is generally supposed that the yielding capsule of this viscus allows it to become the receptacle of an undue proportion of the blood which is driven inwards from the surface of the body in the cold stage.

In cases of death from malarious cachexia the state of the *spleen* is found to be very different under different circumstances. In acute cases it is soft or diffluent, and during life it may rupture from the slightest accident, pour

blood into the peritoneal cavity and bring the case directly to a fatal is
Hertz states that embolic masses are sometimes formed in it, which may
to suppuration, or to gangrene, followed by intense peritonitis. On the o
hand, in cases of chronic ague or of malarial cachexia, the spleen becomes
tremely indurated. Not only is its hardness discoverable by palpation du
life, but after death it is found to have little or no pulp, while the fibrous
ments of its substance have undergone an immense increase. Its capsule is o
very much thickened, and it may be fixed by adhesions to adjacent part

The *liver* is often found enlarged and indurated, and there appear
be some doubt whether it may not undergo a change identical with cirrh
Clinically this condition is further indicated by the presence of pain
tenderness in the hypochondriac regions, and by the occurrence of ab
ascites with which œdema of the legs may be associated.

Maclean cites Parkes as having observed chronic Bright's disease
those who had suffered from ague, and confirms the statement from
own experience. But can one be sure that sufficient care was taken
eliminate other possible causes?

There is, however, another and altogether peculiar result of severe
protracted ague. It is called *melanæmia*, and consists in the presence o
brown or black pigment, partly free, partly enclosed in leucocytes, whic
found not only circulating in the blood, but also lying in the substanc
the solid tissues. The pigment in question is no doubt derived from
colouring matter of the blood. Klebs states that it yields Prussian b
when tested with ferrocyanide of potassium, so that it must contain i
which no longer forms part of an organic compound. It occurs either
minute granules or in larger masses, irregular in shape, and sometimes s
crystalline in appearance. Where there is a large quantity of it, the org
are conspicuously discoloured. Thus the liver, the spleen and the kidn
assume a slaty-grey colour, the medulla of bones becomes chocolate-bro
and the cortex of the brain may appear as if rubbed over with blacklead

A most striking example of pigmentation of the brain, no doubt mala
in origin, occurred at Guy's Hospital in 1829 in the practice of Dr Brig
who figured it in his 'Medical Reports.' The case was that of a man w
with his wife, died of severe fever immediately after they had walked up
London from Horncastle in Lincolnshire. In Germany, as in Engla
melanæmia with abundant pigmentation of the organs appears to be very ra
all the well-known cases recorded by Frerichs belonged to an epidemic of a
which arose in Silesia after an inundation in 1854. But more than
observer has stated that he has constantly been able to detect pigment
the blood, by microscopical examination, in cases of "pernicious" ague,
of malarial cachexia. In 1877 Dr Stephen Mackenzie made a similar ob
vation several times in the case of a man who had brought ague with h
from India. The red discs were normal; but many leucocytes contain
granules which were arranged round their nuclei, or sometimes complet
filled them so as to give them a uniform brown or black colour. Af
treatment with quinine, the melanæmia was no longer discoverable. T
blood of the portal vein has been said to be specially affected with t
change; Dr Hammond has even punctured the spleen during life, and dra
off a few drops of blood from it for examination. The pigment is genera
supposed to be originally formed in the spleen, and to pass from t
organ into the blood, so as to be carried all over the body. It often blo
up the capillaries, but it is also found in the walls of larger blood-vessels, a

even outside them in their sheaths. These facts do not prove that it may not have come from elsewhere, for wandering leucocytes may have carried it with them; but many pathologists think that, in some cases at least, it is developed *in situ* from extravasated red corpuscles. Whether melanæmia directly gives rise to any symptoms appears to be doubtful. Dr Mackenzie's patient had hæmorrhages into each retina, and the same thing has been observed in some other cases of ague. It is natural to suggest that they might have resulted from plugging of minute vessels with pigment, but the anæmia which is so commonly present in such patients would equally account for them. In 1880 a man who had caught ague in the Black Sea was in Guy's Hospital; he had a large hæmorrhage in one retina; but no pigment could be found in the blood. Capillary hæmorrhages in the brain have been attributed to pigmentary embolism of this organ; and a variety of cerebral symptoms have been supposed to result; but Hertz asserts that there is, in individual cases, no close correspondence between the occurrence of such symptoms and the demonstrable presence of the pigment in the minute vessels of the brain. Perhaps the most interesting question of all is whether there may not be something in common between melanæmia and that remarkable affection, paroxysmal hæmoglobinuria.

Ætiology.—We have now to consider what is the nature of the *malaria* or exciting cause of ague, under what conditions it is developed, and how it gains access to the human body.

That ague is apt to prevail in marshy districts is universally admitted. Yet it is no less certain that something more is needed than mere saturation of the soil with water. For countries like Ireland, which are widely covered with peat bogs, scarcely breed malaria; nor, according to Parkes, are salt marshes infested with ague, when they are regularly overflowed by the tide; and this, notwithstanding that for a large part of each day their surface is exposed. Again, the most virulent forms of ague have sometimes been seen in dry and barren districts. Such, according to Hirsch, are the table-land of Castile, the plain of the Araxes, and the lofty plateaus of Northern India and Persia, all of which are highly aguish; he even says that careful inquiry has proved that the malarial regions of Italy are in large parts of their extent devoid of water and sterile. Again, instances in which British troops have been attacked with the disease while encamped upon dry sandy soils, both in Holland and in Spain, are cited by Watson from observations made by Dr William Ferguson nearly a century ago; and Maclean lays stress upon the development of malaria in Hong Kong and in other places, situated upon granite rocks which are undergoing disintegration. These exceptional cases are fatal to a hypothesis which at one time was generally upheld, namely, that the poison of ague is nothing more than a product of putrefying vegetable matters. But there is no real difficulty in bringing them into harmony with cases in which the disease is developed in marshes, or along the estuaries of rivers. For accurate investigations have shown that even there the malaria is developed, not in the wet part of the year, when the ground is flooded with water, but rather during those seasons at which large parts of it are exposed to the air, and become more or less dry. And as Maclean has pointed out, it is tolerably certain that, in every instance supposed to be of an opposite kind, water might very easily have been present, either in the form of subterranean streams, or upheld by a bed of clay or other impermeable material, or only

saturating the soil up to a certain level. So again, it has been noticed t in Italy an occasional overflowing of fresh-water marshes by the sea been followed by a great development of malaria; but this is not incon tent with the fact that marshes which are always saturated with salt w are healthy, since the conditions in the two cases are clearly different.

One way in which a hot sun probably favours the development of a is by cracking the dry surface of the ground and allowing emanations escape from the moist layers beneath. Again, it has repeatedly been fo that the disease has reappeared in places that had long been free from when the soil has been extensively disturbed for building purposes, during the construction of canals or of fortifications. Conversely, much ca done towards preventing the exhalation of malaria by draining the gro and spreading a layer of fresh soil over its surface, or, in the case of villa or towns, by paving the streets and courts.

Many districts are decidedly damp without being aguish; but merely proves that other conditions besides a wet state of the soil necessary for the production of malaria.

At certain times intermittent fevers spread to regions which lie far bey their usual limits, and assume somewhat of an *epidemic* character. The y 1558, 1678–79, 1718–22, 1807–12, 1824–27, 1845–48 are mentioned Hertz as having been characterised by such a wide diffusion of a Watson says that he never knew ague widely prevalent in London exc in 1827. From 1866 to 1868 there was an epidemic in Mauritius, altho the island had previously been so free from malaria that the sufferers f Indian fevers resorted thither. In 1869 it broke out for the first time Réunion.

Climatic conditions.—With regard to the nature of other conditi which favour the development of the ague-poison, very little is known. is curious that there has often been an antecedent or simultaneous epide of influenza, of typhus, cholera, or the plague.

One important condition is undoubtedly a high temperature. A prevails with far greater intensity in the tropics than in temperate clima while towards the poles it is unknown: its boundaries are about 63° and 57° S. of the equator. Hirsch has endeavoured, upon very defect data, to determine the corresponding limits of temperature, and finds t mean summer temperatures rather than mean annual temperatures must taken into account. In this hemisphere, he finds the highest range malaria toward the North at a line between the isothermal lines of 60° 61° Fahr. Again, in particular districts, the occurrence of malarial fe is governed in a very marked manner by variations of temperature. Dur the winter they disappear entirely; and, other things being equal, they always more severe in proportion as the season happens to be a hot o In the tropics the exact period of the year at which ague is most preval varies in different localities; generally speaking, it closely follows the ra season. Hertz lays down the rule that, in order to escape malaria, should arrive in the East Indies between November and January, in t West Indies between January and March.

In temperate climates malaria is usually more active in the spring a autumn than in the height of summer. Wenzel made the important obs vation that among the men employed upon the works at Wilhelmshafen t development of ague followed the summer heat by an interval of twenty twenty-five days, of which he supposed from six to eleven days to be occupi

by the generation of the poison in the soil, while the remaining fourteen days are the period of its incubation in the persons infected.

The liability to ague is greater near the sea-level than in cooler places higher up; but Parkes mentions some instances in which marshes at elevations of five thousand to six thousand feet have, in hot countries, been found to give off malaria. It is, of course, quite a different question to determine what is the lowest point on the hills above a marsh at which one may reasonably expect immunity from the disease. The fact is well ascertained that the wind sometimes carries the ague-poison to considerable distances from its source, not only on level ground, but also up the slope of a range of mountains. Parkes says that in temperate climates an elevation of at least five hundred feet above a malarial spot should be reached in order to escape its influence; in the tropics, one of a thousand to fifteen hundred feet, or even higher still. On the other hand, at the level of a marsh itself, the injurious emanations seem to have but little tendency to rise and diffuse themselves in the air. There is evidence that, both in barracks and in private houses, persons sleeping on the ground-floor are more apt to be attacked than those who occupy upper stories.

It is at night, and for a short time after sunrise, that malaria is most to be feared; probably the poison is harboured by the stagnant mists which are so apt to lie over the surface of damp ground until sunrise.

The presence of actively-growing vegetation appears to be adverse to the development of malaria; and there is reason to believe that the inhabitants of houses in the neighbourhood of a marsh are sometimes protected by the interposition of a belt of trees. The Eucalyptus is believed to be particularly useful in this respect. The poison also seems to be incapable of traversing a surface of water without undergoing absorption. When English troops occupied Walcheren and other parts of Holland, it was repeatedly noticed that only the soldiers who disembarked were attacked by ague; those who remained on board ship, even in narrow channels, escaped.

Chorography.—In England the chief seats of ague are along the eastern coast; Romney Marsh in Kent, the estuary of the Thames in Kent and Essex, the fens of Cambridgeshire and Lincolnshire, and the marshy lands of the East Riding of Yorkshire. In all these districts the disease has become far less frequent than formerly, since population has increased and improved drainage has brought more and more land under cultivation. Not many years ago cases were common in London. James I and Oliver Cromwell * died of ague contracted in this city. At present its occurrence is altogether exceptional, even in the low-lying parts of Southwark. In Scotland there is now but little malaria, and it is said to have begun to disappear before any drainage works were carried out. On the continent of Europe the regions in which the ague-poison is most prevalent are the following: the great plain of North Germany, with some of the Baltic provinces of Russia; Holland, with the adjacent parts of Germany and Belgium; the south-western departments of France; the western side of Spain and Portugal; almost the whole west coast of Italy; a great part of Greece and Turkey; the plains of Hungary; and the shores of the Black and of the Caspian Seas.

* 'The Court and Character of K. James,' by Sir A[nthony] W[eldon], 1651, p. 160. Dr Bates gives the following account of the spleen at the autopsy of the Protector:—"In naturalibus fons mali comparuit; lieue, licet ad conspectum sano, intus tamen tabe instar saurcæ referto." 'Elenchus motuum nuperorum in Anglia,' 1662, p. 417.

Nowhere is it so severe as in Italy, especially in the Maremma of Tusca the Campagna of Rome, and the Pontine Marshes. What is of especial terest is that in these districts it has greatly increased in extent wit the last four centuries. Places which were at one time thickly popula and well cultivated have become waste and deserted; and coincidently w these changes they have grown in the highest degree unhealthy.

In Asia malarial fevers abound, not only in many parts of India, but a in Persia and in China. In Africa the west coast is but too well known the malignant character of the agues which prevail there; and ague is frequent occurrence in Algeria and in Egypt, as well as in Mozambique a Zanguebar. On the American Continent, the States which surround Gulf of Mexico are those in which the effects of malaria are most frequ and severe; they are also seen in parts of Brazil and of Peru, as well as many of the West Indian islands. It is a remarkable fact that in Austr ague is everywhere of a very mild type, if it prevails at all; and it is s to be altogether absent from New Zealand and from Tasmania.

Infection by water.—Hitherto we have assumed that malaria enters the bo with the air which is breathed; and no doubt this is usually the case. I there is good evidence that the poison is sometimes introduced in drinki water. The most striking case of all seems to be that recorded by M. Boud of the one hundred and twenty soldiers conveyed by the French transp ship "Argo" from Algiers to Marseilles in 1834. In the hurry of embar tion the water which was to be supplied to these men was taken fro marshy place near Bona. Thereupon, all but nine of them became attac with various forms of ague; and it turned out that these nine, instead drinking the same water, had purchased wholesome water from the crew the vessel, all of whom remained well. In two more ships, which made voyage at the same time, there were six hundred and eighty other soldie and they too escaped. Another instance is that of a farmer's family Houghton, near Bedford, who drank well-water, and who were at one ti almost the only persons free from ague in the parish, the other inhabita of which had only ditch-water. In another village, close by, it was notic that the digging of wells, by which a supply of fresh water was yielded, w followed by a great diminution of the disease. It seems not unlikely th to the same cause may be attributed whatever part of the decline of ag during the last few years, both in England and in Scotland, cannot accounted for by the improved drainage of the soil. Statements, however, to the prevalence of malaria in former times, cannot be universally accept since it is certain that until recently its effects were frequently confound with those of enteric fever.

Predisposition.—Of the conditions that may render one person more s ceptible to malaria than another we at present know but little. That mo cases of ague are seen in men than in women or in children is doubtless d to the circumstance that men are more exposed to its exciting cause. similar explanation may be found for the fact, if it be a fact, that duri pregnancy women possess a certain degree of immunity.

It seems to be well ascertained that *negroes* are proof against ague; that black soldiers are invaluable for field service in certain parts of the We Indies. Even if this fact be due to a kind of acclimatisation, transmitte by inheritance, it is not the less remarkable. For, although ague oft attacks persons who had recently arrived in a malarious district, yet tho who have lived there for a longer time commonly suffer from malari

cachexia, being puny, sallow, and sickly. The negro, on the contrary, enjoys life and health in such regions.

It seems to be certain that over-exertion and fatigue render a man more susceptible to the action of malaria; and that those who are convalescent from other diseases, or recovering from the effects of wounds, are particularly apt to suffer. It would be worth while to investigate whether persons whose general state of circulation is such that they are prone to the disease known as paroxysmal hæmoglobinuria are more sensitive than others to the ague-poison.

Contagium vivum.—It is impossible to weigh the various conditions which favour or modify the development of malaria, without coming to the conclusion that no hypothesis accords so well with the facts as the presence of a living organism. In 1866 an American physician, Dr Salisbury, declared it to be an alga of the genus *Palmella*, which he had found in marshy soil in Ohio. But he did not attempt to show that the spores of this microscopic plant were really capable of generating the disease; and the same alga has since been discovered in regions where no ague exists. Nor were any direct proofs brought forward by other observers in Italy and elsewhere, who afterwards took up the subject, and by each of whom some different species was assumed to be the essential agent in propagating the disease. It was, therefore, a step in advance when in the spring of 1879 Prof. Klebs, of Prague, and Prof. Tommasi-Crudeli, of Rome, carried out together a series of experimental investigations which they believe have determined not only that what constitutes malaria is a particular kind of bacillus, but also that a fever of intermittent type can be generated in rabbits by subcutaneous injection of liquids containing this organism.* They constructed a machine by means of which a large quantity of air could be rapidly made to pass over the surfaces of glass slides moistened with glycerine jelly. This they set to work in the Pontine Marshes and in Rome, taking the air always from very near the surface of the ground. Direct microscopical examination of some of the glass slides afterwards revealed the presence of rod-shaped bodies and delicate threads; and by suitable methods of cultivation these were made to undergo further development into jointed filaments, with spores in their interior. Similar structures were found in mud taken from the borders of lakes in the same region. When this cultivation had been injected in rabbits the spleen, without being softened, was constantly found enlarged after death, and often contained black pigment. Finally, they cite Dr Marchiafava as having succeeded in discovering the spores and jointed filaments of the same parasite in the spleen, and in the blood of two persons who had succumbed to pernicious malarial fever in Rome.

Mr North has since with great perseverance and devotion followed the investigation of malaria at Rome, and his results differ in many particulars from those of Klebs and Crudeli ('Brit. Med. Journ.,' April 23rd, 1887, p. 865). He is inclined to regard ague as due to a primary derangement of the heat-controlling mechanism of the body, and not as the result of the invasion of microbes.

In the absence of satisfactory evidence of any pathogenic microphytes,

* I must confess that, after reading carefully the details of the experiments in question, and studying the temperature charts which Klebs and his Italian coadjutor give, I fail to see that there is anything definite or characteristic about the fever which occurred in their animals after injection of fluids containing the so-called "*Bacillus malariæ*."—C. H. F.

it was of great interest when the presence of a microscopic *animal* organ was announced as a constant occurrence in ague.

The Italian physicians, Crudeli, Marchiafava, and Celli had descri and figured remarkable pigment granules in the blood-corpuscles of mala cases.* Next, Laveran and Richard discovered pigmented amœboid bo and flagellate organisms within the blood-discs ('Comptes-rendus' for 18 Dr Osler, of Philadelphia, confirmed these observations, and in addi found, outside the blood-discs, crescentic bodies containing granules pigmented corpuscles furnished with one, two, or more flagella in ac motion. ("The Hæmatozoa of Malaria," with figs., 'Brit. Med. Jour March 12th, 1887.)

In Philadelphia, Baltimore, and Washington, American physicians the crescentic bodies so constant that they form a valuable means by wh the malarial nature of an obscure disorder may be practically ascertained

Independently of these observations in man, flagellate organisms h been described in the blood of mules in India suffering from a serious dis known in the Punjab as "Surra," by Dr Griffith Evans and Mr Steel Burma), and also by the late Dr Timothy Lewis in Indian rats ('Qu Journ. Micr. Sci.,' 1879 and 1884). Dr Crookshank has found the s organism in the blood of rats in England ('Journal Roy. Micr. Soc.,' 18 At present it is uncertain whether these apparently polymorphic bodies connected with malaria as cause or effect, or whether they are physiolog as Dr Lewis believed them to be in rats.‡

With regard to the succession of symptoms in the paroxysm of a no special explanation seems to be needed. The same order of eve more or less modified, is seen in the rigors of pyæmia. Like an epile seizure, the ague-fit has a physiological basis. Its recurrence at the s hour on successive days, or every second or third day, is doubtless, as Cu originally suggested, connected with the diurnal habit of body which i plainly manifested in other ways in both health and disease. One th fore need not wonder that Griesinger and Duchek failed to modify the ti at which ague-fits returned by changing the patient's hours of meals and other habits of life.

Diagnosis.—The recognition of ague is for the most part easy, but it m present difficulties. In places which are free from malaria, there is someti no little risk of the real nature of a patient's illness being overlooked, w he has acquired it elsewhere, perhaps while travelling in a country wh the existence of the poison was unknown to him. Again, it is an extrem difficult point to settle whether or not some of the milder and less cha teristic effects of malaria may still continue to appear from time to time places (such, for instance, as the Borough of Southwark) in which the reg forms of ague have long been extinguished. Many experienced physici have undoubtedly held this belief.

* See Professor Crudeli's paper, with drawings of blood-discs after Marchiafava Celli, in the 'Report of the International Congress at Copenhagen,' vol. ii, p. 23.

† During a visit to the United States in 1888 the writer saw marked specimens of characteristic discs in blood taken from patients suffering from ague.

‡ The Indian species were *Mus decumanus* and *M. rufescens*; the organisms in t blood (named Herpetomonas by Saville Kent in his 'Manual of the Infusoria') have l compared to the *Spirillum Obermeieri*, to spermatozoa, to Gaule's 'Cystozoa' in fr blood, previously described by Lankester (*Drepanidium ranarum*), and to stages of Greg nidæ or other forms of Leuckart's Sporozoa.

On the other hand, in regions notoriously infested with malaria, the physician has constantly to keep in mind not only that ague is apt to complicate other diseases, but that it is capable of simulating them. Further, there is always danger of his carelessly mistaking for results of malaria other affections which are attended with recurrent rigors or with paroxysms of febrile disturbance. Thus Graves relates an instance in which the intermittent hectic of *phthisis* was set down as ague. Another disease which is very apt to be overlooked in malarial districts is *pyæmia*. In reference to this, Hertz remarks that the pyæmic rigor generally sets in more gradually than that of ague; that its subsidence is more rapid than its onset, whereas in ague the reverse is the case; and, lastly, that in pyæmia there is very seldom an interval of complete apyrexia, lasting for twenty-four or even for twelve hours. But, as we have seen, ague itself may be merely remittent. The fever attending *internal suppuration* may also be attributed to malaria. The writer once saw a case in which a medical man was firmly convinced of the correctness of such a diagnosis, until his patient voided a quantity of horribly fœtid pus with his urine, after which a rapid recovery took place. Ulcerative *endocarditis*, too, must be thought of.

Finally, there are the continuous forms of ague, with which other diseases, and especially *enteric fever*, have often been confounded. Thus in Romney Marsh no kind of fever used to be recognised except that due to the endemic malaria; and in many parts of India it is only of late years that enteric fever has been recognised. The *fièvre bilieuse hématurique* is not always easily distinguished from yellow fever in countries where both diseases prevail.

Treatment.—We have so potent an antidote to malaria in Cinchona bark, and in the Quinine which is derived from it, that some writers have classed this remedy apart from all others, as the most signal instance of a specific. It is, perhaps, the only medicine of which the efficacy is never challenged by anyone, however rash and inexperienced. The bark itself is not now prescribed, on account of its causing nausea and sickness in the large doses required; quinine or one of the other alkaloids is substituted for it. As a *preventive* of ague, quinine has been found very efficacious for sailors exposed to malaria when sent ashore for a day, for travellers passing through, or for soldiers bivouacking in, a marshy district. From two to five grains are given twice or three times a day. It is less desirable for the permanent residents in an aguish region to take quinine constantly, since its action appears to become somewhat blunted in course of time; but they may take it at the seasons when the disease is most prevalent.

There is reason to believe that the paroxysm itself cannot be modified by quinine taken after its commencement, except perhaps when it is injected subcutaneously. Probably, therefore, if one is called to a patient in an ague-fit the best plan is to prescribe a full dose of some ten or fifteen grains, as soon as possible after its cessation, repeating the same dose more or less frequently during the interval, until an hour or so before the next fit is expected. Maclean advises that the patient should never have less than thirty grains during this period. It is best given in solution with a little sulphuric, hydrochloric, or hydrobromic acid. As is well known, pills containing quinine are exceedingly apt to become hardened by time, so as to pass through the stomach undissolved. If vomiting should occur, the alkaloid may be administered per rectum in a dose of fifteen grains suspended in broth or thin starch.

When the stomach is loaded an emetic is often a useful adjuvant, and if there is constipation a purgative; but it is a mistaken practice to put off the use of quinine until the tongue has become clean.

For the actual paroxysm of ague but little treatment is needed. The patient should go to bed, and have plenty of blankets and hot bottles or hot bricks wrapped in flannel. Baths are better avoided, and he should not be encouraged to drink warm fluids in any large quantity. Frictions with stimulating liniments, or with a mixture of soap liniment and laudanum, have been recommended. It is only when threatening symptoms of collapse are present that recourse should be had to coffee, wine, ammonia or ether. As the hot stage comes on, most of the bedclothes may be removed; the patient may have cooling drinks, or he may suck a little ice, and be sponged with vinegar and water. At the end of the sweating stage he should be carefully dried and have a change of linen. He may then get up, but if he afterwards goes out of doors he must be very careful not to take cold.

It very rarely happens in recent cases of ague, when quinine is properly given, that the patient has any subsequent attack of the same severity as before; and within a few days, or in a week or two at latest, they almost always cease. But in certain cases, especially such as are of long standing and of quartan type, the remedy seems altogether to fail. The drug which then proves most successful is Arsenic. The tincture of Eucalyptus may sometimes, it is said, be prescribed with advantage. The application of cold douches to the left hypochondrium is recommended by Hertz as a valuable accessory to other treatment.

For the dangerous *remittent* and continuous forms of ague, and for those cases in which the symptoms are those of apoplexy or of some other cerebral disease, quinine is still the remedy, but it must be employed much more boldly. Maclean insists on the importance of watching for even a slight remission, using it as an opportunity of giving fifteen or twenty grains of quinine by the mouth, and repeating the dose in two hours' time. If the stomach rejects it, a similar quantity may forthwith be administered as an enema, but in the most severe cases of all he does not wait a moment, and not only prescribes quinine energetically, but also gives stimulants and plenty of nourishment. This writer also speaks in high terms of Warburg's tincture, a remedy now known to contain, in addition to quinine, aloes, rhubarb, camphor, and opium. It is a powerful sudorific.

The *malarial cachexia* requires the administration of iron as well as quinine, but even these remedies are of secondary importance in comparison with the removal of the patient to a healthy locality. Maclean speaks strongly of the value of the ointment of red iodide of mercury in the treatment of chronic enlargements of the spleen and of the liver. He directs that a piece of the size of a nutmeg should be rubbed into the skin of the hypochondriac regions, and that these parts should be then exposed to the sun or to the heat of a fire.

YELLOW FEVER*

"I say, messmate, have you ever had the yellow fever, the *vomito prieto*, black vomit, as the Spaniards call it? No? Have you ever had a bad bilious fever, then? No bad bilious fever either? Why, then you are the most unfortunate creature, for you have never known what it is to be in heaven nor else in the other place. Oh the delight, the blessedness of the languor of recovery!"—*Tom Cringle's Log.*

History—Incubation, course, and events—Morbid anatomy—Ætiology and pathology—Question of its contagious or miasmatic nature—Diagnosis—Prognosis—Preventive and curative treatment.

MALARIAL diseases are, we have seen, as common in the New World as in the Old; they are typically endemic in character. But while the true Oriental plague has never visited the Western Hemisphere, it is there replaced by an indigenous if not an endemic disease, which from time to time visits the cities of tropical and subtropical America with terrible epidemics. This specific and infectious fever is almost limited to America, the West Indian Islands, and the West Coast of Africa. Although it has sometimes been imported into Europe, it has never maintained itself among us. In the East it is altogether unknown.

The first epidemic on record was in 1647, when it appeared in Barbadoes, and was called *nova pestis*. A destructive pestilence of the same kind appeared at Philadelphia in 1693, and again in 1762, 1793, and 1802. It visited Mauritius in 1815, and Gibraltar in 1804, 1814, and 1828. It is endemic in the island of Hispaniola (Haiti and San Domingo), and more or less frequent throughout the West Indies and the adjacent coasts of Mexico, Guiana, and the southern United States. It first appeared on the Brazilian seaboard in 1849, at Buenos Ayres in 1858, and at the port of Callao in 1853. Between 1780 and 1820 it repeatedly occurred in Cadiz and other Spanish ports, in 1821 at Barcelona, and afterwards at Marseilles and Leghorn. There was a terrible epidemic at New Orleans in 1878, and in Florida in 1888. Its range is certainly wider than it was in the last century. There was an epidemic in Lisbon in 1857, and another at Swansea in 1865, introduced from Cuba in the barque "Hecla;" but happily this did not spread.

Course.—The incubation of yellow fever is short, rarely above a week, but it may be considerably longer.

There are sometimes slight prodromal symptoms of malaise and headache; but the disorder often sets in suddenly with rigors and extreme depression. Sometimes it proves fatal in a few hours with collapse and lividity. More often the temperature rises for two or three days until it reaches 105° Fahr., or, as was once recorded, 107°; the face becomes flushed, the conjunctivæ are red and ferrety, and the eyes bright and staring. Frequently there is one-sided headache, or an agonising pain in

* *Synonyms.*—Typhus icterodes—Bilious Typhoid Fever—Black vomit—Yellow Jack—Specific diffuse Hepatitis—Icterus gravis with parenchymatous hepatitis—Calentura vomito-negro—Coup de barre—Mal de Siam is probably a bilious remittent, not Yellow Fever.

the back and sacrum, or there may be pains in all the joints. The stomach becomes very irritable, and rejects its contents; the epigastrium is tender to pressure. The palate is reddened and œdematous; the gums may be swollen and inclined to bleed; the tongue is furred at first, but afterwards clean, smooth, and raw-looking. Constipation is more frequent than diarrhœa. The urine is very scanty, with deficient urea, and it generally contains albumen.

About the third day, or a little later, the next stage succeeds—the "lull," or *stadium* of the fever. The skin becomes jaundiced; the urine is bile-stained; but the fæces are seldom clay-coloured. Hæmorrhage now occurs from various surfaces; epistaxis is frequent, and sometimes there is cutaneous purpura; the vomited matters, from being "white" and consisting of an acid watery liquid, become "black," containing dark brown flocculi or larger masses made up of altered blood-corpuscles. It is to be observed, however, that this symptom of *black vomit* occurs only in the more severe forms, and chiefly in such as end fatally. Thus, during an epidemic at Gibraltar in 1828, Louis found that of the patients who died, all but one had it, and of those who recovered very few. According to Alvarenga, however, at Lisbon, in 1857, there were no fewer than forty recoveries among cases in which black vomit was present.

Haenisch, the writer on this subject in 'Ziemssen's Cyclopædia,' who has himself seen the disease in the West Indies, states that there is usually on the fourth day a remission of the pyrexia, so that the temperature in twelve hours may fall nearly to normal. At the same time the patient loses his headache, and experiences so much relief that he fancies himself out of all danger. The stomach, however, still remains irritable; and the urine, if not previously albuminous, now becomes so. In some cases the improvement is permanent, and goes on to complete convalescence. But more often, after a few hours or within two days at the longest, matters change again for the worse. The temperature may now rise until it reaches 104°; but sometimes remains normal, as, for instance, in a patient of Mr Leggatt who died of yellow fever in London in 1878 ('Clin. Trans.,' vol. xi).

The symptoms at this period seem mainly to depend upon disturbance of the renal secretion. The urine is often entirely suppressed for several days together; it always contains albumen, and frequently blood-casts. The patient sometimes falls into an apathetic condition, but is more often restless and talkative, and sometimes he becomes violently delirious. The pulse is small and thread-like, sometimes rapid, sometimes slow. The jaundice increases until the skin acquires a dark mahogany colour. Hæmorrhage continues, blood being passed from the whole length of the intestine, from the female genitalia, from the external ear, or from the respiratory surface. Death is usually preceded by coma, but sometimes it occurs suddenly, the patient falling backwards upon his bed in syncope, after a state of violent excitement. But recovery may take place even when the case has appeared most threatening—usually by crisis, with a rapid fall of temperature and profuse sweating.

Among the *sequelæ* observed are suppurative parotitis, sloughing of the scrotum and penis, and the formation of boils containing blood mixed with pus. Convalescence is always slow, and several weeks elapse before the patient regains his strength.

During an epidemic of yellow fever cases occur, in which the symptoms are but slightly pronounced. The pyrexia is moderate in degree; there is

but slight jaundice, or it may be altogether absent, or may appear only when the attack is passing off. In this respect yellow fever resembles typhus, scarlatina, and plague.

The *protection* afforded by this disease against second attacks is said to be very efficient. Even relapses are rare.

Morbid anatomy.—Rigor mortis is early and well marked; and, according to Nielly, putrefaction is retarded, which is not what one would have anticipated. The body is deeply jaundiced, more deeply sometimes, says Dr Macdonald, than it was during life. The heart is often pale and soft, and the fibres in a state of fatty degeneration. The pleuræ are ecchymosed, the lungs purpuric, and hæmorrhage is found between the muscles and under the serous membranes or the meninges of the brain. There is usually acute catarrh of the whole length of the alimentary canal, and the stomach sometimes shows hæmorrhagic erosions; but in Mr Leggatt's case the stomach was pale, and its mucous membrane was not swollen. According to Macdonald, the stomach appears perfectly normal when the digested black blood is washed off.

The *liver* may present patches of a bright yellow colour, or its tint may be that of coffee; and all observers agree that in Mr Leggatt's case, Dr Greenfield, who made the autopsy, found that the portal canals were crowded with leucocytes, that many of the bile-ducts were filled with swollen epithelium, and that the greater part of the hepatic cells were undergoing disintegration, being swollen and fused together, or broken up into irregular fragments. It may, however, be a question whether in this case the state of the portal canals was not an accidental cirrhotic change, due to the known habits of the patient. If not, Dr Greenfield's observations tend to support a doctrine, upheld by Liebermeister in his work on 'Hepatic Diseases,' that yellow fever is nothing else than a "parenchymatous hepatitis" dependent upon a specific infection. This view is supported by Dr Wickham Legg in his work on the 'Bile and Jaundice,' where he cites the description of microscopical appearances by Dr Alonzo Clark, of New York, and several other observers. Acute fatty degeneration with atrophy is constant, and all observers agree that the hepatic cells are loaded with fat globules. The biliary passages are found free from obstruction, and the gall-bladder contains sometimes tarry bile, sometimes colourless, thin mucus.

The spleen is but slightly if at all enlarged; its tissue is often firm and healthy-looking, and it is not infrequently shrunken—probably as the result of gastric hæmorrhage.

The kidneys are enlarged and may show points of suppuration; in the case already referred to, Dr Greenfield found the epithelium of the convoluted tubes swollen and proliferating, and the straight tubes contained hyaline casts; but the kidneys were in this instance granular, so that these changes may have been unconnected with the yellow fever. According to Dr Legg, however, a condition of cloudy swelling like that found in the early stage of parenchymatous nephritis is the rule.

Ætiology.—With regard to the origin of yellow fever there have been great differences of opinion. One of the most striking features of the disease is that its infectious principle is often transported by ships. Of this a few instances have become historical. In 1823 H.M.S. "Bann"

carried yellow fever from Sierra Leone to the island of Ascension. In 1845 the "Eclair" steamer brought it from the African coast to Boà Vista, one of the Cape de Verde Islands. In 1861 it was conveyed by a wooden sailing ship, the "Anne Marie," from Havannah to St Nazaire, in France; and in 1865 by a similar vessel, the "Hecla," from Cuba to Swansea. Moreover, the disease frequently passes from one ship to another, not only where they have been lying side by side in the same port, but also on the high seas. The "Anne Marie," for instance, infected seven other vessels which happened to be brought near her; and during the local epidemic at Swansea a smack, the "Elinor," which took in cargo near the "Hecla," had her crew attacked after they had left that port and had reached Llanelly. So, in 1795, the "Hussar" frigate captured a French ship, the "Raison," on which yellow fever was prevailing; only those prisoners who were believed to be in perfect health were transferred to the English vessel, but notwithstanding this precaution the disease soon broke out on board the prize.

These facts are best explained by supposing that yellow fever, like typhus and the exanthemata, is propagated by a contagious emanation from the bodies of the sick; the infection, like that of plague, has often been found to cling to the hull, or perhaps to the cargo, of a particular vessel, after the crew have been paid off. It is believed in the West Indies that a cargo of hides or of sugar is favourable, and one of salt unfavourable, to the development of yellow fever on board, or to its transfer from one port to another, even when the crew escape.

In the 'Med.-Chir. Review' for 1848 and subsequent years there appeared a series of able articles, which are now known to have been written by the late Dr Parkes, in which evidence was offered that yellow fever is contagious. He relates in full detail the circumstances which attended its diffusion in Boà Vista from two soldiers, belonging to the fort, who were lodged while ill in the chief town of the island, Porto dal Rey, as well as from a labourer, who brought it direct from the "Eclair" into another town, Robil; and in each case he shows that the next persons to be attacked were those who lived close to the patients and visited them.

On the other hand, many of those who have had the largest acquaintance with yellow fever have disbelieved in its contagiousness, in the ordinary sense of that term, and many facts have been adduced in support of their view. One is that, as Griesinger has pointed out, the disease often remains localised upon the sea-shore, or in close proximity to the banks of a navigable river; it may even confine itself to a small part of a seaport town in the immediate neighbourhood of the harbour. Thus, when it prevailed at Lisbon in 1857, one hundred and eighty-two persons are said to have left the city for different places in Portugal, carrying with them the disease, and eighty-six died, but in no instance was it communicated to other persons in the places whither they went. In 1865 Dr Buchanan, having investigated with great care the local epidemic at Swansea, came to the conclusion that "the evidence tending to negative personal contagion was about as strong as such evidence can by its nature ever be."

Climate.—Yellow fever only flourishes in hot climates—a further reason, perhaps, for supposing that its infective principle multiplies outside the human body and subject to atmospheric influences. The regions in which the disease commonly prevails are all situated near the equator; and the occurrence of a local epidemic within the temperate zone seems constantly to be associated with an exceptionally sultry state of the weather at the time.

This was the case at St Nazaire, in France, when it developed itself there in 1861; and also at Swansea, in 1865. The latitude of Swansea is 51° 37′, which is beyond the geographical limit usually laid down for the disease. So, again, upon low coasts and near the mouths of rivers, the worst months are generally July, August, and September; although in small rocky islands within the tropics the period from October to February is stated to be the most dangerous. Some exceptions, however, to the rule that the contagion of yellow fever cannot resist cold have been recorded. Thus Dr Archibald Smith recorded, in the first volume of the 'Transactions of the Epidemiological Society,' the fact that in 1855 it prevailed at Cuzco, in the Peruvian Andes, where (as he was informed) the temperature of summer rarely, if ever, reaches 65° Fahr. in the shade; and also at Cerro Pasco, with a mean temperature of 44° by day. So, again, Mr Leggatt's patient, who died in London, was taken ill on March 21st, 1878, the mean temperature of the four previous days having been 54°. He had arrived at Southampton from Rio Janeiro (where the disease was epidemic) on March 17th, and there had been three cases of yellow fever on board the ship.

Many physicians believe that the disease is not contagious from the sick to the healthy, but that its germs are carried on the clothes of healthy persons, who thus spread the infection in the same way as other "fomites," merchandise, or, above all, ships. It is quite in accordance with the analogy of enteric fever, of plague, and of cholera, that though infective, and derived only from a previous case of the disease, yellow fever should not usually be propagated by direct contact, but by less obvious transference of a virus.

It is remarkable that yellow fever does not occur among negroes, and less, it is said, among mulattoes or quadroons than in those of pure European blood. According to Humboldt, the Indians of South America are also exempt.

Men are more often attacked than women, and adults than children and old people—facts which probably depend on the usual spread of the disease in epidemics being by commercial intercourse.

Pathology.—Yellow fever is certainly a specific disease in its origin as in its course, and the above ætiological facts point to its being miasmatic-contagious (cf. p. 12), although of this there is at present no proof. Dr Parkes, indeed, in the articles above referred to, maintained that yellow fever is, or may be, developed out of ague, which, he supposed, undergoes conversion from a non-contagious into a contagious disease. He considered it to have been proved, for example, by Dr Bryson that when the "Eclair" left Sierra Leone on her way to Boà Vista, there was no yellow fever in the former place, so that the crew of that vessel must be supposed to have derived the fever from "endemic sources" in the soil water or vegetation, to which they were exposed both in their boat-services and in unhealthy anchorages.

It is now, however, universally admitted that yellow fever is quite distinct from any form of intermittent or bilious remittent fever of malarial origin; for its geographical range is quite different, it is epidemic, it is transmitted from place to place, it consists of a single attack and protects against future invasion, albuminuria is constant, the spleen is not enlarged, and quinine, so far from being a specific remedy, is believed to be injurious; and no pathologist would now maintain that a malady so distinct in its origin, symptoms, anatomy, and natural history, can be developed out of an admittedly different

group of disorders. Moreover, it is obvious that the facts cited by Dr Parkes with regard to the spread of the disease at Boà Vista are quite consistent with the hypothesis that the infective principle of the disease—having once been introduced into the island from the "Eclair" upon clothing or upon the persons of those who landed from the ship, or having even been carried into Porto dal Rey by the soldiers who were taken ill in the fort—afterwards established itself in the soil, and flourished as a miasm. Moreover, it seems probable that when infection clings to the hold of a ship, the requisite nidus for a specific microphyte may be afforded by the bilge-water.

No microbe has yet been discovered, though it was carefully searched for with all the modern methods of detection during the epidemic in Florida and Cuba in the year 1888.

A question remains as to the relation of yellow fever to other forms of malignant jaundice (*icterus gravis*). Grisolle, Garnier, Liebermeister, and Wickham Legg, maintain that the two are identical, and that the cases of acute yellow atrophy of the liver which occur in Europe are sporadic cases of yellow fever.

This can scarcely be. The absence of fever in the former, and its presence and height in the latter; the atrophy as well as softening and degeneration of the liver, the swollen spleen, and the entirely non-contagious character of the former; to say nothing of the absence of hæmorrhagic vomiting and the presence of leucin and tyrosin in the urine—all these facts prevent our identifying the two diseases. But the icterus, the hæmorrhages, the rapid course, and the histological change in the liver, with the affection of the kidneys, show a close relation between them.

Diagnosis.—This is not always easy except when yellow fever is known to be prevalent. The affection most apt to be mistaken for it is the bilious form of remittent fever. Other diseases that must be borne in mind are relapsing fever and the various local forms of jaundice attended with pyrexia, acute yellow atrophy, and also poisoning by phosphorus. The distinction from remittent malarial fever has already been indicated.

The "bilious typhoid" described by Griesinger in Egypt and the Siamese fever of French writers were both probably bilious remittent fevers, and not, as has been supposed, yellow fever.

Prognosis.—Yellow fever is a dangerous pestilence—very different in this as in other particulars from malarial fevers. The mortality seems to vary widely in different epidemics, being sometimes as low as 15 per cent., sometimes as high as 75 per cent.

Even in the comparatively mild visitation of New Orleans in 1878 more than 4000 people died, and there were about 4000 deaths at Barcelona in the year 1821.

The general indications are that the greatest danger is in the case of drunkards, and of those who go about while the fever is already upon them. In children the prognosis is favourable.

In a given case, the severity of the initial pyrexia is not of bad omen; but much hæmorrhage, and particularly hæmatemesis, in the second stage, and the presence of petechiæ, severe cerebral symptoms, convulsions, and stupor, with dilated pupils and suppression of urine, are very unfavourable and often fatal symptoms.

The symptom of most value as regards prognosis at an advanced stage

is said to be albuminuria; if the amount of albumen in the urine diminishes as the case goes on, the patient is likely to do well; if it increases, a fatal termination is to be apprehended. Ordinary bilious vomit is accounted a good sign, hæmatemesis and black vomit a bad one.

Quarantine.—If yellow fever is a contagious-miasmatic disease, the precautions which should be adopted in order to prevent its transport from one country to another may be greatly simplified, in comparison with the regulations laid down until recently by the best authorities, as, for instance, by M. Mélier in France after the St Nazaire epidemic in 1861. As Sir John Simon remarks in his 'Eighth Report,' the segregation of persons arriving from an infected town, and their confinement in a lazaretto for a definite period of time, is, on this view, superfluous, and in a trading country like England, all but impracticable.* The points to which the whole energies of a port sanitary officer should be directed are the isolation and disinfection of the vessel which is known or suspected to contain the virus of the disease. It should be compelled to anchor at a distance from all other vessels; and every part of the hold should be thoroughly cleansed. It would now be advisable to use carbolic acid for this purpose, rather than the chloride of lime, which M. Mélier recommended. The cargo, and the clothes and other effects of the sailors and passengers, should be disinfected at the same time.

The necessity for stringent precautionary measures against the development of yellow fever in England and in other temperate climates is very much diminished by the fact that a high external temperature is believed to be a very important, if not essential, factor in its ætiology.

Treatment.—The treatment of yellow fever is mainly symptomatic. Quinine is useless or injurious. At the beginning, some experienced physicians give a purge of calomel, jalap, and ginger. The cold pack has proved useful in many cases, but mustard applied to the legs, or a hot footbath, appear to be preferable at the onset. For the relief of sacral pains dry cupping may be used. The irritability of the stomach may be checked by the administration of creasote or a few drops of hydrocyanic acid, chloroform, or chlorodyne. The patient should be fed with bland nutriment, such as thin arrowroot, barley-water, or chicken broth. Ice is used, if it can be obtained; and it seems to be the usual practice to allow the patient champagne or brandy, well diluted with water, during the second stage. In one case recorded by Dr Macdonald, the cook of H.M.S. "Icarus" very nearly succumbed in the second stage, "but he rallied immediately on the administration of a stout glass of rum and water and recovered steadily." Notwithstanding the almost constant presence of albuminuria and the not infrequent supervention of uræmic symptoms, physicians experienced in the treatment of this disorder use subcutaneous injections of morphia to relieve vomiting.

* Quarantine (Fr. *quarantaine*, a period of forty days) was introduced by the Republic of Venice in 1348, to prevent the invasion of the Oriental Plague, or Black Death as it was then called. In England it was enforced by law under Queen Anne (1710) in order to prevent the return of plague, and a fresh Act was passed in 1727 by the advice of Mead, after the terrible pestilence of measles in the preceding year. No plague appeared, but the quarantine laws became absolute until the beginning of the present century, when their uselessness was seen. Since 1831, the same precautions have been in force in many parts of Europe against Cholera. In the New World, and also in Spain, quarantine regulations have also been adopted to prevent the invasion of Yellow Fever.

DENGUE*

"Nova febrium
Terris incubuit cohors."
HORACE.

History—Course and symptoms—The fever and the interval—The exanthem—Sequelæ—Pathology—Contagion—Diagnosis—Prognosis—Treatment.

AMONG the new epidemic fevers of the present century is one which, like yellow fever, is unknown in England: but it is common in India and some of the colonies. It clearly belongs to the group of specific diseases, and is perhaps most like influenza.

In 1824 this disease, hitherto unknown to Indian surgeons, broke out at Rangoon in Burma, and quickly spread to Calcutta and to various places in Bengal or in Madras. In 1827 the same malady appeared in the West Indies in the Isle of St Thomas, and a few weeks later in the adjacent Isle of Santa Cruz. Here also it was at first regarded as a new disease; but it has since been found that Dr Rush, of Philadelphia, described it as prevailing in that city in 1780. During the last fifty years it has from time to time attracted attention both in the Eastern and the Western tropics. There was an outbreak in India in 1871–72, when it is said to have been derived from Zanzibar, passing to Bombay by way of Aden; in 1873 it spread to China and to Cochin China, and also to the islands of Mauritius and Réunion. In the West Indies it was last seen at Martinique in 1874 and in 1875. The only spot in Europe in which this malady has hitherto appeared is Cadiz, where it was observed by Poggio in 1867; and an earlier epidemic is said to have occurred in the same town as far back as 1784. Recent accounts tell of the same disorder as an epidemic in the Levant (1889).

In St Thomas the negroes called the new disorder "dandy fever," apparently in ridicule of the attitude and gait of the patient; and it is now universally known as Dengue—a Spanish word, of similar meaning.

Course.—Dengue sometimes sets in with lassitude, drowsiness, vertigo, a sensation of chilliness down the back, and other febrile symptoms; but more often it begins suddenly with pain in some particular part of the body, coming on while the patient is walking about, or in the night, waking him from sleep. Dr Stedman, who, in the 'Edinburgh Med. Journ.' for 1828, gave an excellent account of the disease in St Thomas, says that the first thing noticed was often a stiffness in one finger, especially the little finger; this would increase and be accompanied by intense pain, which spread over the whole hand and up to the shoulder; and in a few hours the fingers of both hands would be swollen, stiff, and painful, and their joints incapable of being bent. It does not

* *Synonyms.*—Dandy Fever—Breakbone Fever—Three-day Fever—Scarlatina Rheumatica—Febris exanthematica articulosa. Other absurd names are "broken-wing fever," and "giraffe," the latter because the neck is held in a stiff position. The word *Dengue* has been coined as a Latin name of the disease, but *Dengue* is the form used by French and German as well as English writers.

appear that effusion can be detected in the joints, as in rheumatic fever; but Hirsch says that, in the rare cases of death, serous infiltration has been found in the connective tissue round certain joints. Cotholendy, in describing dengue in the Isle of Réunion, suggests that there is an exudation into the sheaths of the tendons, and especially of the extensor tendons, and that it is this which renders movements of the joints so exquisitely painful; and he also speaks of "a slight fulness, a sort of œdema," of the hands and feet. There is often violent pain in the eyeballs, which feel too large for their sockets and as though they would start from the head. Before long every part of the body becomes the seat of extreme suffering, aggravated by restlessness, which compels the patient to be constantly changing his position. He also feels extremely prostrate and distressed. Sleeplessness is a marked symptom, and children are sometimes delirious.

In the meantime the temperature rises until it reaches 102°, 103°, 104°, or even (though very rarely) a higher point still. Observers who have employed the thermometer during some of the more recent epidemics have attached some importance to remissions, which occur three or four times in the twenty-four hours. Most writers have described the pulse as very rapid, 120 or even 140 in the minute, but a writer in the 'Arch. de Méd. Navale' for 1874, says that he often found it not much over 80, far lower than might have been expected from the temperature. Twining long ago noticed that the countenance was flushed and of a scarlet hue, and recent observers describe an exceedingly fugacious *initial rash*, consisting of bright red patches upon the face, the chest, the palms of the hands, and elsewhere, which, however, subsides after the lapse of a few hours. There is anorexia; the tongue is thickly furred, with bright red edges. The stomach is extremely irritable, and often rejects everything that is swallowed. The bowels are constipated.

The condition of the patient at this period might well cause considerable anxiety to anyone unacquainted with the disease; but at the end of twenty-four or thirty-six hours the pyrexia begins definitely to subside, without any marked critical sweat, and the temperature soon falls to normal or slightly below it. At the same time the pains in the limbs cease, and soon there is nothing for the patient to complain of except a sense of general weakness, loss of appetite, and backache. At this time it is said that the glands in the neck, the axillæ, and the groins may be felt to be slightly enlarged. The parotid is often swollen and salivation is frequent.

The duration of the interval is about three days. At the end of this time an exanthem—the *second rash*, according to recent observers of the disease—appears upon the skin. It is first seen upon the palms of the hands, next on the feet and the knees, and may in exceptional cases spread all over the body. In appearance it is usually intermediate between the rash of scarlet fever and that of measles, and it has also been compared with the erythema that sometimes accompanies rheumatic fever. In some cases it is attended with the formation of bullæ or of wheals like those of urticaria. It gives rise to a distressing sensation of tingling, which presently passes into still more intolerable itching. Its development is sometimes associated with a return of pyrexia, but recent observations seem to have shown that, as a rule, the temperature remains normal at this period of the disease. Consequently the fact that an eruption has made its appearance is not seldom overlooked; whether it is ever really absent is doubtful. After a few hours, or two or three days, it subsides and disappears, and with it the

fever, if any. Afterwards the cuticle begins to desquamate, usually as a branny powder, but sometimes in large flakes, and this may be attended with considerable discomfort and soreness, especially of the feet.

The second eruption is often associated with a repetition of the articular pains, which are, however, less severe than at the beginning of the disease. Or there may be a respite of three or four weeks, and at the end of that time the pains may return, compelling the patient to take to his bed again and be fed like a child. Dr Stedman speaks of these pains as always most severe in the morning and as wearing off in some measure towards evening. They are felt chiefly in the fingers and toes, in the wrists, the ankles, and the knees. The affected joints may be so stiff and swollen as to produce deformity. After a few days the secondary pains in their turn begin to subside, and one limb after another becomes free.

Bronchitis may occur as a complication, and Nielly has occasionally seen pericarditis in severe cases. Months may elapse before the patient is entirely exempt from return of pain, and the weakness is long-continued and distressing.

Pathology and causation.—The true place of dengue among infective diseases has yet to be accurately determined. In the rapidity with which it spreads over a population it resembles influenza more than any other malady.

A remarkable point is that scarcely anyone escapes, even among the inhabitants of a large city, as, for example, among the half million of residents in Calcutta in 1824. It attacks persons of all ages, including even infants a few days old, and the coloured races are as liable to it as whites. When it has lasted for a little time, one might think that there were none but cripples in the place, so many are seen limping about the streets on crutches, with bodies half-bent or with arms in slings. The duration of an epidemic is from two to seven months, according to Hirsch.

The disease is said to prevail chiefly with sultry, cloudy weather or at the time of heavy rains. In temperate climates it occurs only during summer and autumn, and disappears when frosts set in; but in the West Indies vicissitudes of weather seem to have no way interfered with its course. Its diffusion from one country seems plainly to be effected by human intercourse. Whether it is contagious in a restricted sense, passing from the sick directly to the healthy, is uncertain. Dr Stedman says that in 1827 it was introduced from St Thomas into Frederickstadt, a town of Santa Cruz, by some young ladies who went to stay at a house where all the members of his family were attacked, and a few days later it appeared in the next house and affected everyone there. Cotholendy relates that an infant, which had taken the disease while with the family of its nurse, was brought home to its mother; four days afterwards she fell ill; the grandmother and the aunt did not see the child until the day after its arrival, and they were attacked a day later than the mother.

He gives several other instances which indicate that the *incubation* of dengue lasts four days.

No distinctive morbid anatomy is known, and nothing has yet been ascertained as to the nature of the infective virus.

Diagnosis.—The epidemic character and the peculiar rash, with the immunity of the heart, distinguish this singular disease from rheumatism,

the articular pains and absence of sore-throat from scarlatina or measles. It is not malarial, for it affects the whole population equally; there is no splenic enlargement, and quinine is useless.

Prognosis.—Dengue is scarcely ever fatal, but infants sometimes die with convulsions during the primary pyrexia, and old people may succumb to exhaustion towards the end of the disease, or to bronchitis. When untreated, recovery is said to be very slow.

Treatment.—It is recommended to give an emetic at the onset of the malady, and then a purge of calomel and scammony or jalap, repeated each day. After the bowels have been freely opened, a dose of laudanum, or Dover's powder, completes the cure. Liniments of chloroform, belladonna, or cajeput oil are often useful, and Dr Stedman found that the application of mustard plasters or blisters to the neck or to the loins gave great relief. When the joints remain stiff and painful after the subsidence of the disease, sulphur baths are said to be very efficacious.*

* For valuable observations on the clinical characters and the treatment of dengue, as well as of yellow fever, the reader is referred to Dr Shattuck's notes to the American translation of Strumpell's 'Handbook of Medicine.'

EPIZOOTIC DISEASES

OCCASIONALLY TRANSFERRED TO MAN

"Nec tondere quidem morbo illuvieque peresa
Vellera, nec telas possunt attingere putres.
Verum etiam invisos si quis tentarat amictus,
Ardentes papulæ atque immundus olentia sudor
Membra sequebatur; nec longo deinde moranti
Tempore contactos artus sacer ignis edebat."

VIRGIL (of Cattle Plague).

Οἱ δὲ ὄνοι μάλιστα νοσοῦσι νόσον μίαν ἣν καλοῦσι μηλίδα· γίνεται δὲ περὶ τὴν κεφαλὴν πρῶτον, καὶ ῥεῖ φλέγμα κατὰ τοὺς μυκτῆρας παχὺ καὶ πυῤῥόν.—Aristotle, 'Hist. Animal.,' lib. viii, cap. 25 (of Glanders).

"Ὑδροφοβίαν Græci appellant: miserrimum genus morbi."—CELSUS (of Hydrophobia).

ANTHRAX.—*Nomenclature—Distribution—Varieties in animals—The Bacillus—Modes of infection—Varieties in man: Charbon, Intestinal and Thoracic Anthrax, their anatomy, prognosis, and treatment—Pathology—Prophylaxis.*

GLANDERS.—*History and nomenclature—Origin in horses—The Bacillus—Transfer of contagium—Acute form—eruption, ozæna, pyrexia, fatality—Chronic form—its treatment—Anatomy.*

FOOT-AND-MOUTH DISEASE.—*An epidemic disorder among cattle—Bacillus—Its conveyance to man—Its symptoms, diagnosis, and treatment.*

ACTINOMYCOSIS.—*Its occurrence in cattle—in the human subject—The microphyte—its transference—Pathology—Varieties and course—Treatment.*

HYDROPHOBIA.—*Incubation, symptoms, course, and event—The disease in animals—Its ætiology, anatomy, and pathology—Diagnosis—Treatment—Prophylaxis by Pasteur's method of inoculation.*

THERE are certain epizootic maladies which sometimes pass from animals to man. These may conveniently be classed together in this last chapter on the specific contagious and epidemic maladies.

ANTHRAX.*—This name, formerly used as a synonym for carbuncle, is now applied to a very different disease which human beings derive by infection from sheep, horses, and oxen.

With regard to the *geographical distribution* of anthrax as a disease of cattle, the parts of Europe in which it is most common are Poland, Hungary, the countries of the Lower Danube, Prussian Saxony, and certain departments of France. It is endemic in Catalonia, in the Romagna, and in Courland, and often prevails in Siberia and in Lapland, in Western Asia, Australia, Mexico and South America, in India, and in South Africa. It is not endemic in this country.

Anthrax occurs not only in domesticated animals, but also in deer,

* *Synonyms.*—Splenic Fever—Splenic Apoplexy—Malignant Pustule.—Siberian Cattle-plague ("Jaswa"). In India it is known as "the Loodiana plague," and at the Cape as "Horse sickness." *Old English* Blackbain, *Fr.* Charbon, Mal de Chabert, *Germ.* Milzbrand.

reindeer, buffaloes, and elephants. It also affects swine, and is easily inoculable upon rabbits and guinea-pigs. Carnivorous animals are much less susceptible; but cats are more so than dogs.

Varieties in cattle.—In different kinds of animals the symptoms of infection by the specific virus of anthrax differ considerably. But, according to Bollinger, three principal varieties may be recognised:

1. Sometimes the animal (generally an ox or a sheep) becomes convulsed and insensible, with rapid breathing, and dies after a few hours.

2. Other cases are characterised by pyrexia, which is often remarkably remittent in type. Clonic spasms of the limbs are also observed. The fæces may contain a large quantity of blood.

3. In yet other cases the most marked feature is the formation of brawny inflammatory swellings in and beneath the skin of the neck, chest, abdomen, or any other part, which may lead to extensive sloughing and ulceration.

Obviously only the last of these varieties would naturally be termed anthrax. But it is on every ground desirable that a single name should be given to them all. Indeed, although veterinary pathologists until lately regarded the more rapidly fatal cases as examples of a specific fever without localisation, it is now known that there always is a local lesion in some part of the body. Very often this consists of an infiltration of gelatinous exudation and blood into the connective tissue of the abdomen and of the chest. Or there may be an acute inflammatory swelling of the tongue, or of the submucous tissue of the pharynx, or of the lining membrane of some part of the intestine. We shall presently see that all these forms of the disease are also met with in men. Anthrax is by no means always fatal to animals; the average mortality in horned cattle and in horses is said to be 70 per cent. The characteristic lesion, found after death, is enlargement of the spleen, which is from two to five times its normal size, softened, and of a black colour.

The Bacillus.—The proof of the identity of the various forms of anthrax lies in the fact that all are caused by a single microphyte, which is now commonly known as the *Bacillus anthracis.*

This organism was discovered independently by two German observers—by Pollender in 1849, by Bräuell of Dorpat in 1857. But Davaine was the first who, in 1863, maintained that the contagion of the disease lay in these bodies, which on account of their being motionless he distinguished from the common mobile bacteria of putrefaction by the name of *bacteridia.* They consist of straight or slightly-bent rods, measuring 0·007—0·002 mm. in length. Dr Frisch and Dr Cossar Ewart have under certain circumstances observed movements in them, but as a rule they are motionless. By Cohn they have been classified as a bacillus which he termed *Bacillus anthracis,* though he observes that it is almost exactly like the *Bacillus subtilis* which constitutes the butyric acid ferment, except that the latter is motile.

The rods sometimes cohere together at their extremities, and Koch and Ewart have described them, when cultivated in aqueous humour as growing into long filaments, in the interior of which bright granules appear—the spores of the bacillus, far more capable of resisting heat and desiccation than the adult microphyte, and retaining for an indefinite length of time the power of development into the mature form. Nägeli maintained that the bacilli of anthrax, like putrefactive bacteria, multiply only by fission; and there is no doubt that *Bacillus anthracis* does multiply by fission under the

surface of an infusion, but by spores when exposed to air, and also, under certain conditions, by assuming a Torula form. These facts have been ascertained by Klein and other independent observers, so that Koch's original statement is abundantly confirmed. The bacilli are found in the blood, spleen, and many other organs (see Dr Crookshank's 'Bacteriology,' pl. xiv, fig. 1; xvi and xvii, fig. 1).

Infection.—The origin of anthrax, as it occurs in cattle, in horses, and sheep, is of great interest, and throws light upon the class of diseases known as *miasmatic-contagious* (p. 12), to which it belongs.

In the first place, it is readily transmissible by means of the blood from a diseased to a healthy animal. Inoculations are frequently and successfully practised by veterinary surgeons and by pathologists for diagnostic purposes.

There is reason to believe that the virus is not infrequently introduced by the stings of insects: Bollinger took gadflies from the body of an ox that had died of anthrax, and found by direct experiment that the contents of the stomach and intestines of these flies were capable of conveying the disease to rabbits. Another way in which it is sometimes given to sheep is by the bite of a dog that has just been feeding upon infected flesh.

There is not sufficient proof that secretions from the bodies of living animals affected with anthrax pass into the air and infect other animals through the lungs. Like enteric fever, yellow fever and cholera in man, this disorder is not directly contagious; yet the virus sometimes clings to stable utensils, harness, straw, or hay. Bollinger mentions a local outbreak which he carefully investigated at Weriken, and in which for four years the cattle in two sheds of the same proprietor were decimated by anthrax, while it did not affect those in other buildings, closely adjacent. An analogy for this occurrence may be found in those local house-epidemics of enteric fever in which the virus seems to go on multiplying itself in or near a particular water-closet, so as to affect a number of persons in succession, sometimes at long intervals of time.

Anthrax also resembles the miasmatic-contagious diseases of man in being often spread in ways still more indirect. It prevails among animals pastured upon damp soils containing much humus, as, for instance, upon peat-bogs, and near the borders of lakes or rivers that have overflowed. And it is most frequent during the hot months of the year, particularly in August and September.

Buhl has included anthrax among the diseases to which he applies his "ground-water" theory (cf. pp. 240–1), and he states that among the horses belonging to a large stud kept near Donauwörth the disease, which had been raging for a long time, ceased as soon as a system of drainage was carried out. But Bollinger argues that the reason why its prevalence is affected by dampness of soil is, in reality, that ground containing moisture affords conditions favourable for the multiplication of the *Bacillus anthracis*, which he supposes to be capable of maintaining an independent existence under such circumstances.*

* Some observers have thought that this organism may sometimes appear without having been derived from a previous case of anthrax. Buchner asserted that, by a series of experiments, he converted a bacillus which is found in infusion of hay into an organism capable of producing in animals an infective disease identical with anthrax, and he asserts that to turn the anthrax-bacillus back into a hay-bacillus is comparatively easy. There is, however, good reason to disbelieve this alleged transformation. See Professor Klein's Report to the Local Government Board, "On the Relation of Pathogenic to Septic Bacteria, as illustrated by Anthrax Cultivations" (Blue-book for 1882).

Anthrax is never purely *miasmatic*. Its specific bacilli exist in the soil, or in water, only when they have been introduced from a previous case. They may be either derived from the excreta of diseased animals, or from their dead bodies, buried as they often are in the fields, or left to rot among the brambles and nettles. Instances are recorded in which "enzootic" anthrax has ceased so soon as stringent rules for the disposal of all dead bodies were enforced. Probably some of the bacilli from the infected carcases become free, undergo desiccation, are suspended in the air, and inhaled into the lungs; others pass into drinking-water, and thus reach the stomach, while others, again, may gain access to the lymph-spaces and blood by some breach of surface in the skin.

Conveyance to man.—Shepherds, farm-labourers, and other persons who come into contact with living animals affected with anthrax seem seldom or never to take the disease by mere *contagion*. But veterinary surgeons may be infected in performing venæsection, and slaughterers in killing or in skinning; the probability is that a slight abrasion or scratch upon the skin then becomes directly inoculated with the virus.

Another way in which anthrax may arise in man is as the result of *eating the flesh* of an infected animal. Leube, of Jena, has recorded an instance in which the imperfectly cooked liver of a diseased goat conveyed it. Thus the human gastric juice seems not necessarily to destroy the virus, as has been found to be the case with the gastric juice of carnivorous animals such as the dog. Thorough cooking probably removes all danger, but there still remains the risk of injury to the butcher, and to those who are concerned in preparing such meat for the table; and this is sufficient reason why the use of it, as food, should be prohibited. According to Heusinger the disease may even be conveyed by milk or by butter.

But by far the most important cause of anthrax in man is infection *from the dried skins or hair* of diseased animals. In this way it is brought straight from Asia or South America to English workmen.

At Guy's Hospital cases are of not infrequent occurrence among the men engaged in the Bermondsey leather trade, and especially among those employed at a particular wharf on the river-side where foreign hides are unshipped. Broca years ago remarked how often anthrax was set up by carrying skins upon the shoulders. It is remarkable that the danger appears to be greater when the hides are first brought ashore than afterwards.

The same thing has been observed at Bradford, where a peculiar *Woolsorters' Disease* has for many years prevailed, and this has recently been shown to be identical with anthrax. The source of infection in that case is chiefly "Van mohair," a material which contains much putrid matter and many "fallen fleeces," torn off the bodies of dead animals. Dr Bell, who gave an excellent account of the affection in the 'Lancet' for 1880, remarked that the men who are attacked are chiefly the "bagmen"—that is, those who open the bags in which the hair is packed, shake it out, and sort it into different qualities. The reason is, no doubt, that the virus is in a dried state, and that as soon as it is disturbed it diffuses itself in the air and is inhaled into the lungs. Perhaps it may actually have multiplied itself during the transmission of the bags from Asia Minor; for, according to Dr Bell, the hairs are not known to communicate disease to persons who handle them in the country where they are grown. Indeed, no form of anthrax so severe as the Woolsorters' Disease had hitherto been described as occurring in man. The work of sorting is said to be free from danger

if the contents of the bags are first washed or even moistened with water.*

Other occupations which sometimes lead to infection with anthrax are the picking of Russian horsehair (as reported by Dr Russell, of Glasgow, in 1879), paper-making, and the manufacture of coarse woollen hats.

Varieties of human anthrax.—There are several forms of this disease in man, and they are not identical with those met with in animals.

1. *Malignant pustule* or *charbon* is the name by which the most common form has long been known. This consists in the formation of a vesicle, seated upon a more or less brawny base. Being usually produced by the inoculation of a scratch or slight sore, it occurs chiefly on the face, especially the cheek, and also on the neck, the forearm, or some other exposed part.

There is first a period of incubation, lasting generally several days, often ten, but sometimes only a few hours. A slight pricking or burning sensation is then felt, which often leads the patient to think that he has just been stung by an insect. A papule quickly appears, and soon passes into a transparent vesicle, which may reach a considerable size. This ruptures and dries up into a dark-coloured scab. Round it there sometimes arise smaller vesicles arranged in a ring. Meanwhile the base of the vesicle becomes indurated, and a red or purple areola is developed round it. A brawny œdema quickly spreads over the adjacent parts, affecting perhaps the whole of one arm, or of one side of the neck. Sometimes inflammation of lymphatic vessels occurs, and the corresponding glands become swollen.

During the early part of this morbid process the health remains unaffected. The patient may continue at his work, and at most complains of slight malaise and of febrile disturbance. But after about forty-eight hours severe pyrexia sets in, often attended by delirium, prostration, diarrhœa, sweating, and acute pains in the limbs; and death, preceded by a state of collapse, may occur between the fifth and the eighth days. In one case which occurred at Guy's Hospital, the original vesicle had been situated an inch and a half behind the left angle of the lower jaw, and respiration became so difficult towards the last that recourse was had to tracheotomy. We found after death that brawny infiltration had extended deeply to the fauces, so that the entrance of the larynx and its interior were greatly swollen. In other instances the immediate cause of death is septicæmia or coexistent anthrax of the intestine or lungs.

Diagnosis.—The only disease liable to be mistaken for anthrax affecting the skin is that which has been particularly noticed by Sir James Paget under the name of *facial carbuncle*. It presents no definite vesicle or central scab; and in most instances we have found the veins of the face obviously plugged with puriform thrombi, which has not been the case in anthrax.

Bollinger, however, and other writers describe, under the name of *anthrax-œdema*, a modification of the true charbon, in which there is neither vesicle nor eschar, but only a pale yellowish swelling of the subcutaneous tissues. The eyelids are the most frequent seat of this variety.

* See Reports to the Local Government Board by Mr John Spear: 'On Woolsorters' Disease,' 1880, and 'On Anthrax among persons engaged in the London Hide and Skin Trades,' 1883. Also papers by Sanderson ('Journ. R. Agric. Soc.,' vol. xvi, p. 267) and Greenfield (ibid., xvii, 30). That the infection of anthrax is conveyed by the hides is implied by Virgil in the concluding lines of the third Georgic, which stand at the head of this chapter.

One way of arriving at a diagnosis of a suspected case of anthrax is to inoculate a rabbit or a guinea-pig, or even a mouse, with fluid from the part primarily affected, or with the blood. Such small animals commonly die in two or three days, or still more rapidly, with dyspnœa, dilatation of the pupils, and perhaps convulsions. Their blood may then be seen to swarm with bacilli. However, if the experiment of inoculation is followed by a negative result, a negative conclusion is not warranted.

On examination after death the naked-eye appearances are those of an infective fever:—congested lungs, flaccid heart, and more or less ecchymosis or hæmorrhage. As a rule, the spleen is much enlarged and softened, but in a case under the writer's care in August, 1889, which proved fatal after operation on the primary pustule, the spleen was not swollen; but the distinctive microscopic evidence of anthrax-bacilli was present.

Treatment.—The recognition of the ordinary external form of anthrax is of extreme importance, because surgical treatment at an early stage is capable of arresting its progress. The disease is not, indeed, necessarily fatal, even when left to itself. Sometimes the central part of the swelling sloughs out, the surrounding induration subsides, and an ulcer is left which more or less quickly heals. Such a spontaneous cure is very rare. Among nine instances recorded in 1863 by Dr William Budd eight ended fatally. On the Continent it has long been the practice to destroy the local lesion as soon as it is brought under the eye of a surgeon. Bollinger cites the experience of two observers who lost only thirteen cases out of one hundred and forty-two; another had still greater success, since among two hundred and nine patients all but eleven recovered. In 1878 Mr Davies-Colley recorded in the 'British Medical Journal' two cases, in each of which recovery took place after excision of the entire mass of indurated tissue, with the application of chloride of zinc paste to the wound. Contrasting with these is a case which was left alone, and which ended fatally. Several other successful cases at Guy's Hospital have been recorded by the same writer in the 'Med.-Chir. Trans.,' vol. lxv, 1882. Since that date (June, 1882—December, 1889) forty-eight more cases have been admitted to Guy's Hospital; and of the total sixty-five cases of external anthrax forty-three recovered after operation. Two typical cases in butchers during an epidemic of anthrax among cattle were operated on by Dr Pitts, of Chelmsford, and recovered perfectly ('Brit. Med. Journ.,' March, 1887, p. 616).

The rapidity with which the surrounding œdema subsides after the operation is often very striking. It may be effectual even when the case is at an advanced stage, and when severe general symptoms are present. But under such circumstances quinine should be given in large doses, and the patient's strength must be kept up with nourishing food and with stimulants. Carbolic acid should also be administered internally, since its therapeutical value in the lower animals when affected with anthrax appears to be generally admitted.

2. *Intestinal anthrax*, another form of splenic fever, affects the gastro-intestinal mucous membrane. This has been recognised only within the last few years, and chiefly by German pathologists. It is sometimes associated with an external malignant pustule. A case in point, in which Dr Goodhart made an autopsy, occurred at Guy's Hospital in 1877. Otherwise its symptoms seem to be obscure, so that a correct diagnosis is not likely to be made

during life unless the patient is known to have been exposed in some way to the virus of the disease. However, Bollinger gives the following account of the symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in with diarrhœa, and the evacuations often contain blood. There may be pain in the abdomen, which becomes somewhat tumid. Dyspnœa and lividity appear, with restlessness and excitement, or with stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is but slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

On *post-mortem* examination the abdominal cavity is found to contain a moderate quantity of serous fluid which is often blood-stained. The lining membrane of the stomach and intestines shows patches of swelling, generally of the size of lentils or coffee beans, but sometimes one or two inches in diameter. These on section are seen to consist of a pink fleshy infiltration of the mucous and submucous tissues, so that the valvulæ conniventes appear firm and prominent; the surface of the affected parts is more or less excoriated and discoloured or covered with an adherent layer of extravasated blood. There are also spots of ecchymosis on both the serous and the mucous aspect of the gastro-intestinal tract. The mesenteric and the lumbar lymph-glands are often greatly enlarged, and of a dark red colour. The mesentery may form a large brawny swelling, and the connective tissue in front of the spine may be infiltrated in a similar manner. The spleen is softened to a pulp, but it is not generally much enlarged. A nodule in the stomach may slough out and repair begin, as in a case recorded by the late Dr Mahomed, in the 'Path. Trans.' for 1883.

Fatality.—Hitherto no instance of recovery from intestinal anthrax has been recorded. Leube has related a case in which the lower lip, the inside of the cheek, and the hard palate presented indurated patches of a bluish-red colour: epistaxis and hæmaturia appeared, but the malady ended favourably in about a fortnight. The patient took fifteen grains of carbolic acid and thirty grains of quinine each day, and the patches in the mouth were cauterised three times daily with carbolic acid. But the diagnosis of anthrax was not established beyond question; for only spores, no bacilli, were found in the blood. (Compare Mr John Poland's case: 'Path. Trans.,' 1886, p. 553.)

3. *Pulmonary anthrax.*—A third form of anthrax affects mainly the thoracic viscera. It has hitherto been recognised chiefly among the wool-sorters of Bradford. After Dr Bell, of that town, had drawn attention to it, Mr Spear and Dr Greenfield investigated it for the Local Government Board. Dr Greenfield's description of it is briefly as follows:—The earliest symptoms are great prostration and a sense of oppression of breathing. Shivering seldom occurs. The respiration is not much accelerated, but it is laboured and difficult, with a feeling of pressure or constriction. There may be more or less abundant bloody expectoration, or none at all. Auscultation seldom reveals anything more than slight rhonchus. The face is sometimes congested, sometimes pale, with a slight cyanotic tint. The extremities are cold and bluish; even in the axilla the temperature may be subnormal; but in the rectum the thermometer may rise to 102° or 103°.

The pulse is rapid and weak and sometimes irregular. There may be nausea and vomiting, but not generally diarrhœa. In many cases the appetite remains good, and digestion seems to be unimpaired. There is sometimes a sort of hysterical condition, or a state of mental anxiety and depression; but other patients have been so unconscious of danger as to refuse to send for medical advice until the last few hours of life. Death may be preceded by delirium, convulsions or coma, or the mind may be clear to the last, and the end come suddenly and unexpectedly. One of Dr Bell's patients lived only seventeen hours after he was first taken ill, and many other cases have terminated fatally in from three to five days. Dr Bell says that those who survive for a week generally recover.

The *post-mortem* appearances vary considerably. Decomposition appears to be somewhat rapid. The lung tissue may either be congested or quite natural. There may be blood-stained secretion in the bronchial tubes, and their mucous membrane may be ecchymosed. Sometimes the pleuræ contain a pint or two of fluid, and the lungs are partially collapsed in consequence. There is often blood-stained serous or gelatinous infiltration of the mediastinal tissues, and the bronchial glands are swollen, softened, and ecchymosed. The pericardium may be marked with petechiæ, and may contain a blood-stained fluid; or a large quantity of blood may be poured out between it and the sternum. Sometimes the pharynx and the adjacent tissues are infiltrated with blood or serum. There may also be ecchymoses in the pia mater, in the kidneys, and elsewhere. It is remarkable that the spleen is hardly, if at all, softened. In prolonged cases hæmorrhagic infarctions may be found in the lungs, attended with slight pleurisy; or there may be patches of broncho-pneumonia.

Obviously there may sometimes be considerable difficulty in distinguishing, by clinical evidence alone, this form of anthrax from a severe attack of ordinary pulmonary inflammation. Indeed, until recently, fatal cases of the Woolsorters' Disease at Bradford were commonly registered under the head of pneumonia, bronchitis, or congestion of the lungs. It can hardly be doubted, as Dr Bell suggests, that a similar affection will hereafter be found to occur in many towns where carpet and blanket wools, dry hairs or furs, are used for manufacturing purposes.

General pathology of anthrax.—Certain points in regard to the distribution of the bacilli in animals or men affected with anthrax have still to be mentioned. They have an important bearing upon the theory of infective maladies in general, because they illustrate what is perhaps a universal law, namely, that the microzymes of contagion multiply locally before they infect the blood to any considerable extent. In the ordinary form of the disease affecting the skin, the bacilli may be found, according to Davaine, about the second or the third day, in clusters embedded in the rete mucosum at the centre of the vesicle. E. Wagner has since found that they are at this time so closely packed in the papillæ of the cutis as to conceal all the tissue elements. Thence they spread both laterally and towards the deeper structures, enter the vessels, and are carried all over the body with the blood. In the gastro-intestinal variety the bacilli infiltrate all the swollen and œdematous tissues, so that Buhl and others described this affection under the name of *mycosis intestinalis* before they recognised that it had any relation to anthrax.

At an early stage of the disease the blood may contain no bacilli.

Therefore, if in a doubtful case one fails to discover them in this fluid, one must not attach much importance to this result. On the other hand, the presence of well-marked rods is conclusive; but it does not seem to be sufficient to find spore-like cocci only, as Leube did in the case referred to above (p. 356). In man, even when a fatal termination is approaching, the bacilli seem to be seldom found in such immense numbers, and so generally distributed in the blood, as they are in animals. Davaine calculated that from eight to ten millions are present in a single drop of a diseased beast's blood. In Buhl's first case of intestinal anthrax they are said to have been present in the blood of the portal vein, but not in that of other vessels. They may often be detected in the substance of the spleen and of the solid tissues generally, and also in the fluid poured out into the serous cavities.

Mr Barker, in a case published in the 'Med.-Chir. Trans.,' vol. lxix, p. 127, found the bacilli most abundant in the rete and the papillary layer of the cutis, and they appear to be long confined to that part.

The best account of the histology of the disease in this country is given by Dr F. C. Turner, in the sixty-fifth volume of the same 'Transactions.'

In anthrax we have a typical example of a specific contagious malady, which is due to the presence of microphytes. All the conditions stated on p. 13 are fulfilled. For the disease is recognisable from its clinical features, and breeds true. The organism is well marked in its microscopical characters, size, and shape, in its reproduction and its cultivation. It exists in the blood and tissues of every case of the disease. It exists nowhere else, for its non-mobility distinguishes it from *Bacillus subtilis*. Lastly, when a "pure cultivation" has been obtained, it can be inoculated, and will reproduce the disease, with fresh broods of organisms in the blood.

Prophylaxis by inoculation of the attenuated virus, though inapplicable as a practical measure for human beings, is of too great theoretic importance to be wholly passed over.

Pasteur ascertained that by repeated cultivations of *Bacillus anthracis* in mutton broth, at a temperature above 104°—110° Fahr., he obtained a modified virus which when inoculated into an animal produced a mild attack of anthrax which protected from future attacks. These results were tried on a large scale at Milan and afterwards at Chartres, and proved brilliantly successful (see a paper by Mr George Fleming, in the 'Nineteenth Century' Magazine for March, 1882). Unfortunately, the result is not always so complete, for even the attenuated virus is occasionally fatal. Moreover, the Algerian breed of sheep appears to be insusceptible of the same protection, and in Hungary the results were much less successful than in France. See an excellent abstract by Dr Dawson Williams of Pasteur's and other methods of attenuation, and of Koch's criticism thereon ('Microparasites in Disease,' N. Syd. Soc., p. 560). Dr Roy found in Buenos Ayres that the Viscacha (*Lagostomus trichodactylus*), a rodent allied to the chinchilla, is capable of receiving the disease, and that anthrax thence transferred by inoculation to cattle is both mild and protective, at least for a considerable time—so that the analogy to smallpox and cow-pox is very close.

GLANDERS.*—From an early period in the present century it has been known that those whose occupation brings them into contact with horses sometimes become affected with a disease to which these animals are liable. The disease in question was described as far back as the fourth century B.C. under the names μᾶλις and *malleus*. In England, writers on veterinary medicine have been accustomed to distinguish two varieties, "glanders" and "farcy," the former of which is characterised by a morbid state of the nasal mucous membrane, the latter by the formation of nodules and abscesses in and beneath the skin, and by indolent inflammation of the lymph-glands and lymphatics, attended with swelling (or "farcy buds") at the valves. The first case in the human subject which was correctly interpreted in this country was recorded in 1821 by Mr Muscroft, of Pontefract. In 1830, Dr Elliotson drew the attention of the Royal Medical and Chirurgical Society to the disease, and afterwards proposed to call it *Equinia*. According to Virchow, the derivation of "glanders" is from *morbus glandulosus*, and the equivalent German word *Drüse* is still in use for one of its forms.

Conveyance of contagion.—As a rule, the virus of glanders is directly introduced into a wound or sore, or fissure in the skin, especially of the face or of one of the hands. A horse-slaughterer who died in Guy's Hospital in 1866, appeared to have poisoned a slight cut on the lip which had been made by a barber in shaving him. Injuries to the hand in skinning dead horses, or in dissecting them, have sometimes been the starting-point of the disease. It has also been transmitted by a bite from a glandered animal, probably through the saliva. Or a diseased horse in sneezing may propel a drop of muco-purulent secretion from its nose directly into the eye, or the nose, or the open mouth of anyone standing near it. The contagion remains active in a dried state for a long time, for horses in a particular stable have been attacked many months after the occurrence of a case. Probably it is sometimes deposited upon the straw in a stall, and a groom may disturb the dried particles and cause them to float off into the air so as to infect him with the disease. However, the Messrs Gamgee, in Dr Reynolds' 'System,' express doubts on this point.

According to Bollinger, who describes glanders in 'Ziemssen's Handbuch,' it may be communicated by eating the flesh of a glandered animal, at least in a raw state; he says that lions in menageries often become diseased in this way. Decroix, indeed, is stated to have repeatedly eaten such flesh uncooked, without injury; but it must be borne in mind that in all probability comparatively few persons are susceptible of the virus of glanders. At any rate the disease in man has always been exceedingly rare, even in countries where (as in France during the first half of this century) it has committed terrible ravages among horses. When it affects human beings, it is capable of spreading from one individual to another. Dr Elliotson mentions in the 'Lancet' for 1838 that a laundress who washed the clothes of one of his patients contracted it. In at least one case, infection has taken place through a cut received in examining the body of a person who had died of the disease. It is also inoculable from man to the lower animals; goats and rabbits are said by Bollinger to be the most suitable subjects for experiment. Oxen are stated not to be susceptible, but, next to horses, it is especially apt to affect asses and mules. It is seen in hot as well as in cold climates.

* *Synonyms.*—Equinia—Farcy—μᾶλις, Malleus *v.* Maliasmus.—*Fr.* La Morve, Le Farcin *Germ.* Rotzkrankheit, Wurmkrankheit.

A point of some importance is that grooms are sometimes attacked with glanders when they are not aware that any of their horses have been diseased. Bollinger seems to have satisfactorily explained this, by showing that in the horse glanders sometimes fails to present its more characteristic symptoms and attacks the lungs and the air-passages only.

Bacillus.—Bollinger failed to find bacteria in careful examinations of the fresh blood of glandered animals and of fresh nodules from their bodies. Gerlach stated that the virus is not destroyed by putrefaction. Löffler has since ascertained the constant presence of minute rod-shaped organisms in the "farcy buds," lungs, and spleen of animals dying of glanders. He has succeeded in growing this bacillus, and inoculation with the pure cultivation has reproduced the disease in horses or asses, and also in guinea-pigs, rabbits, and mice. His results agree with those of Schütz, Bouchard, and other observers, and are now generally accepted.*

Glanders in man.—The disease occurs in two forms in the human subject, of which one is termed acute and the other chronic.

1. In *acute glanders* the patient is first attacked with malaise, headache, and pains in the limbs. There is often no initial rigor, and for a time there may be no marked pyrexia. At this stage the disease is generally mistaken for acute rheumatism or for enteric fever. But if a wound or scratch has been infected with the virus, the part commonly becomes red, swollen, and very painful; and an erysipelatoid inflammation may diffuse itself over a wide area, affecting, for example, the whole side of the face, or the hands, and part of the forearm. In such cases the diagnosis generally inclines towards septicæmia or pyæmia from a poisoned wound. In other instances, as in that of a man who died in Guy's Hospital in 1863, the first complaint is of pain in the side with dyspnœa, so that acute pleuro-pneumonia is suspected.

Presently symptoms appear which to an experienced eye reveal the nature of the disease. One of these is an *eruption*. Upon the limbs and the body there arise papules, which rapidly pass into flat vesicles and then into bullæ or pustules, attaining the size of peas or even of sixpenny-pieces. They become depressed in the centre, and rupture, allowing a thin purulent fluid to escape, which is often blood-stained. With regard to their histology, von Wyss has ascertained that the papulation begins as a local inflammatory change in the papillary layer of the cutis. When an incision is made through a pustule after the patient's death, its floor is found to consist of an ashen-grey, infiltrated layer. The eruption sometimes appears within twenty-four or forty-eight hours after the patient is first taken ill, sometimes not for a week, or even longer. When present it is characteristic of glanders, although it might be mistaken at an early stage for small-pox or chicken-pox, and perhaps later on for pemphigus. Generally there is mixed with it an affection which may be taken as the representative of "farcy" in horses. This consists in the formation of hard painful lumps or swellings in the subcutaneous tissues and muscles; they more or less rapidly suppurate, and if they are incised they may ulcerate, so as to expose tendons or bones beneath. It is curious, however, that in most cases the lymph-glands fail to become enlarged. A marked instance to the contrary is recorded by Travers in his work on 'Constitutional Irritation.' Not only

* 'Deutsche med. Wochensch.,' 1882 and 1883; 'Revue Médicale Française,' Dec., 1883; 'Microparasites in Disease,' New Syd. Soc., p. 387, and pl. vi. Klein ('Micro-organisms and Disease,' 3rd ed., p. 128), and Payne ('Pathology,' p. 667, with fig. after Flügge).

was there suppuration of the glands of the arm originally affected, but those at each angle of the lower jaw and in the groin are also said to have been swollen. The joints in glanders not infrequently become inflamed, and pus is effused into them.

The other characteristic symptom is an affection of the mucous membrane of the nose and of adjacent parts (*oxæna*), representing what in horses is termed "glanders" in the narrower meaning of the term. First a thin whitish mucus runs from one or both nostrils; afterwards it becomes purulent, blood-stained, and fœtid. The nose itself now becomes swollen, red, and very painful, and inflammation may spread from it towards the forehead or over the cheeks. If an opportunity arises of examining the parts after death, the lining of the nasal passages is found to be ulcerated, and the septum may even be necrosed. It is to be observed that in the human subject a discharge from the nose is by no means always present in glanders; Hauff observed it in only thirty out of seventy cases which he collected. It is often not an early symptom, but appears in the second or the third week. Thus, during the first days of a doubtful case, the fact that the nose is healthy must never exclude the diagnosis of glanders.

Other mucous membranes are also affected. The conjunctivæ may be severely inflamed, and the eyelids may become greatly swollen. Virchow relates a case which came under von Graefe as one of acute exophthalmos, and its real nature was not suspected until after the autopsy. Sores may form in the mouth, and the gums may become spongy. The pharynx and the palate may ulcerate or become covered with a kind of false membrane. Sometimes ulcers form in the larynx, producing hoarseness, and an œdematous laryngitis may set in, so that tracheotomy may be required. Symptoms of bronchial or of intestinal catarrh may be present. Pustules and sores, which might easily be supposed to be chancrous, are said by Virchow to occur on the glans penis.

The general condition of the patient becomes worse from day to day. The pulse is generally much accelerated, but sometimes it remains slow, the temperature rises irregularly until it may reach 104°; the tongue becomes dry and brown; albumen appears in the urine, and sometimes leucin and tyrosin. Delirium and sleeplessness pass into stupor and coma, and finally collapse, with involuntary escape of the urine and fæces. Death usually occurs towards the end of the second, or in the third week. Sometimes the disease has ended fatally within a week, or even in three or four days, sometimes not for four weeks. Bollinger gives one case of recovery among thirty-eight which he collected.

All that can be done in the way of *treatment* is to sustain the patient's strength with quinine, tincture of iron, nourishing food, and stimulants. The pustules should be opened and the nostrils cleansed by syringing them out with some antiseptic solution.

2. *Chronic glanders* is much more insidious. It is often characterised by the formation of intractable ulcers with thick livid edges. In the museum of Guy's Hospital we have two models of such ulcers, the part affected being in one case the back of the hand, while in the other case there are separate sores upon the forehead, the lobule of the ear, and the side of the face. In other instances, abscesses form about the joints and give rise to fistulous sores; or inflammatory swellings appear on the limbs, beneath the skin, or in the muscles. Or, again, there may be an eruption of pustules like those which are seen in acute glanders, but more slowly developed.

The nose often remains free, but sometimes there is an erysipelatoid redness of the skin or a fœtid purulent discharge from the mucous surface, beginning perhaps after two or three months have passed. Bollinger says that the root of the nose may even become gangrenous. The nostrils are often blocked with dark-coloured crusts. Such an affection is very apt to be regarded as syphilitic, or to be classed under the vague title of ozæna. It is of great importance to remember that the diagnosis may be cleared up by inoculating a goat or a rabbit with some of the discharge. This practice was adopted in some of the earliest cases of glanders that are known to have been observed in this country, those which were recorded by Travers. Bollinger gives details of two such experiments which he performed with matter taken from horses; the inoculated animals, which lived two or three months, showed characteristic symptoms and lesions. In a case at Guy's Hospital in 1866, the late Mr Poland introduced pus from the patient's face beneath the skin of the shoulder and set up pustules there, but probably this result was not of diagnostic significance. Virchow mentions that at Würzburg a man was for six months in the hospital on account of refractory sores upon the limbs. At the autopsy lesions characteristic of glanders were discovered. It was not then known that the disease existed in the neighbourhood, but inquiries were set on foot and led to the discovery of an epizootic prevailing over a wide area among the horses used for towing barges on the River Main.

In some cases the chief symptoms of chronic glanders are cough with sanguineous expectoration, hoarseness, pyrexia, and emaciation, so that phthisis may be suspected.

The average duration of chronic glanders was found by Bollinger to be four months; sometimes it was not more than two or three months. It may end by passing into the acute form of the disease. Among thirty-four cases which he collected from various sources, including many of slight severity, there were seventeen recoveries. As might have been anticipated, the convalescence is generally very slow and is sometimes incomplete. He was himself acquainted with a veterinary surgeon who, at the end of eleven years, was still cachectic, and was troubled with cough and other symptoms due to the presence of cicatrices in the larynx and in the nose. But he says that some patients, after a severe illness lasting for months, get quickly well and regain the flesh that they have lost.

The means of *diagnosis* now adopted in cavalry stables is to inoculate an ass with the mucus from the nostrils of a horse suspected to be glandered.

As to *treatment* of chronic glanders, Bollinger thinks that carbolic acid and iodide of potassium are the internal medicines most likely to be useful; the Messrs Gamgee speak of arsenic in combination with strychnia as having sometimes exerted a remarkable influence. Ulceration of the interior of the nose should be treated with injections of creasote water, or a solution of carbolic acid, and the application of nitrate of silver or tincture of iodine may be of service.

Morbid anatomy of glanders.—Many of the *internal lesions* which are found in cases of glanders in man appear to be the result of pyæmia or septicæmia, which often arises secondarily, just as it does in erysipelas; but nodules and more or less extensive patches of catarrhal pneumonia, which have no pyæmic characters, are also seen. Virchow placed glanders, in association with syphilis and lupus, among the "granulation tumours." But he himself states that in the human subject the process concerned in the formation of the pus-

tules and abscesses has little to distinguish it from a simple inflammation. In animals both he and Bollinger describe the affection of the nasal mucous membrane as beginning with the development of minute miliary papules, and in the lungs they speak of nodules of various sizes, some almost exactly like tubercles, others as large as peas, or even as walnuts. In man, Virchow says that on careful examination, characteristic small yellow bodies may be seen in the mucous membrane of the nose, of the frontal sinuses, and even of the larynx and of the trachea. In von Graefe's case he detected them in the choroid of the eye. Von Wyss has since observed them in the gastric mucous membrane. It is interesting to find in one of Elliotson's cases, recorded in the 'Med.-Chir. Trans.' for 1833, a description of similar appearances. "On cutting into the various tumefactions on the head, trunk, and extremities, they were found to be full of pus, underneath which, in many, a number of small white granules were seen, and others, in several instances, were closely attached to the periosteum and perichondrium. The frontal sinuses contained a jelly-like mucus, and a number of similar granules, and on the septum narium was one ulcer exactly like those I have seen in the nostrils of glandered horses, and upon it lay a cluster of granules. Two of these large white circular elevations were found immediately below the sacculi laryngis; Mr Youatt, who was present, called them true glandrous chancres. About an inch below the valve of the colon, for three inches in extent, upon the whole of the surface, were white granules exactly like those in other parts."

In a case which occurred at the Middlesex Hospital in 1872, and which is recorded in the 'Med. Times and Gaz.' for that year, Dr Coupland discovered an acute inflammation of the spinal cord.

FOOT-AND-MOUTH DISEASE.*—In striking contrast with the dangerous epizootic diseases hitherto described is one which in cattle seldom causes more than a transitory illness, and which when communicated to man generally produces but trifling effects. It is termed the "Foot-and-Mouth Disease." Some writers have unwisely appropriated to it the mediæval name of *murrain*, which is familiar from its being used in the English translation of the Bible. On the Continent the complaint in question has been known for centuries, but it is said to have been first introduced into England in 1839. In 1869 it prevailed here extensively, and in 1871 it is believed to have affected as many as 700,000 animals. It occurs chiefly in cattle and in sheep; pigs are also liable to it, but probably only as the result of infection by feeding upon the milk of diseased cows. It is very rarely seen in horses.

In cattle, the foot-and-mouth disease is attended with a moderate degree of pyrexia. One of the most marked symptoms is the formation of vesicles and bullæ upon the mucous membrane of the mouth, including the lips and the tongue; these parts also become swollen, and there is a dribbling of saliva. The vesicles soon break, and their floors become covered with a thick grey layer. A somewhat similar eruption appears upon the feet, round the borders of the hoofs, and in their clefts. The vesicles then pass into pustules and dry up into crusts. They give the animal pain in walking or standing, so that it generally remains lying down. Another seat for the vesicles is upon the udder and the teats, which may in consequence become raw and excoriated. But within twelve or fourteen days all the

* *Synonyms.*—Aphthous Fever—Aphtha Epizootica—Glossanthrax—Eczema Epizooticum.

effects of the complaint pass off. It almost always ends in recovery, except in calves. Of these from 50 to 75 per cent. die, probably because the milk yielded by their mothers (when they also are affected) has an irritant action upon the alimentary canal, besides conveying the specific virus.

The *contagium* appears to reside in the fluid contained in the vesicles. By cultivating in agar-agar, in broth, and in milk (which it turns acid, but does not curdle), Dr Klein has succeeded in isolating an apparently distinct micrococcus.

The disease in man.—One may well be surprised that, if foot-and-mouth disease is capable of transmission to man at all, it should not be frequently transmitted. Yet in the 'Brit. Med. Journal' for 1867 will be found a list of only twenty-two notices of such an occurrence, which were all that Prof. McBride could find recorded during the previous thirty years; and most of these were taken, not from medical journals, but from the 'Veterinary Review.' It might therefore be supposed that human beings are but little susceptible to it. But, if so, it is curious that two foreign observers, Hertwig and Jacob, should each have readily succeeded in infecting himself experimentally by drinking the milk of diseased animals.

Conveyance.—There are two ways in which the complaint may be communicated to man. One is by the direct *inoculation* of the specific virus into a crack or sore place, generally on the hand or on a finger. This is especially apt to happen to a person engaged in milking a cow with the eruption upon the teats, but sometimes saliva or fluid from the mouth conveys the contagion to a veterinary surgeon who is attending to the sores there; and a butcher is said to have taken it by holding his knife between his lips while dressing an infected carcass.

The other way is by *drinking the milk* of a diseased cow. This is far more important, not only because it concerns children, who suffer much more than adults, but also because it is very likely to be overlooked, at least by persons who are not conversant with the symptoms observed under such circumstances. As to the characters of the milk which is yielded by cows suffering from the foot-and-mouth disease, there have been some discrepancies of statement. Sometimes it is offensive to the smell and the taste, and it may even be mixed with blood or pus in considerable quantity; but this seems to be only when the teats are severely excoriated, or when the mammary glands themselves have become inflamed as the result of an undue retention of their secretion. In other cases it is less obviously altered; and then one of its most marked peculiarities is a tendency to coagulate when boiled, or even at a much lower temperature, forming innumerable little flocculent masses, which float in a bluish whey. Sometimes it seems to have a yellowish tint, and when examined microscopically displays granular cells like those of the colostrum. But sometimes it is said to have a perfectly natural appearance. It seems generally to be diminished in quantity by about one half. There is no doubt that it is very often drunk with impunity, not only by adults, but even by children. In any case boiling may be assumed to render it innocuous.

Course.—The *incubation* of the "foot-and-mouth" disease in man is said to be from three to five days. At the end of this time slight pyrexia arises, with loss of appetite. A sensation of dryness and heat in the mouth is then noticed, and vesicles quickly appear upon the inside of the lips, on the tongue, and sometimes upon the fauces and hard palate. They reach the size of peas, their contents become opaque and yellowish, and in from

one to three days they break, forming shallow, dark-red ulcers. There is some pain, which is increased by mastication, by swallowing, and by talking. The lips become swollen, and mucus and saliva are poured out in excess. In a case related by Mr Briscoe, of Chippenham, in the 'Brit. Med. Jour.' for 1872, the tongue swelled until it protruded for two inches and a half outside the mouth, and it was so firmly wedged between the teeth that for thirty-six days the patient took no food except milk; there was great dyspnœa, and suffocation appeared at one time imminent; sloughs peeled off the tongue, and the discharge became very fœtid. Such a condition, however, is altogether exceptional. There is often slight disorder of the digestive organs, indicated by diarrhœa and abdominal pain.

In some patients an eruption appears upon the fingers, especially round the nails. It consists of small clear vesicles, which pass into pustules, and sometimes run together. It very rarely happens that the inflammation is sufficiently intense to cause shedding of the nails. The feet are seldom affected, but sometimes vesicles have been observed between the toes. Mr Amyot ('Med. Times and Gaz.,' 1871) mentions the case of a woman in whom the feet "became hot and covered with painful tubercles." It is said that the female breasts have occasionally presented tubercles, and even the face and other parts of the body. In Mr Hislop's case ('Ed. Med. Jour.,' 1863), in which there was a red scaly rash upon a farmer's forehead, and upon the body and lower limbs of his wife, cured by bichloride of mercury and iodide of potassium, one may venture to doubt whether foot-and-mouth disease was really present, or syphilis.

Diagnosis.—The eruptions, however, for which the foot-and-mouth disease seems most likely to be mistaken are varicella and slight smallpox. The affection of the lips and tongue might be set down as a simple aphthous stomatitis. Indeed, it is not impossible that a slight catarrhal inflammation of the mouth, with a little soreness of throat, may be a far more common result of infection by diseased milk in infants than has hitherto been suspected. A doubtful case might always be cleared up by the inoculation of a sheep which has not had the disorder already.

Prognosis.—The duration of the foot-and-mouth disease in man is usually from ten to fourteen days, but sometimes not more than a week. A case fatal by septicæmia has been recorded, but with that exception death has occurred only in very young children.

The sole *treatment* required is the application of a solution of borax to the mouth, or of the solid nitrate of silver to any painful ulcers; the eruption on the fingers may be dealt with like a mild eczema.

ACTINOMYCOSIS.—Of late years a fourth contagious disease of the lower animals has been recognised in man.

The malady was long known among cattle as a suppurative inflammation, usually affecting the tongue or jaw, occasionally the skin or the lungs (cf. Rivolta, of Pisa, in 'Virchow's Archiv,' 1875, vol. 88, p. 309). It was called "wooden-tongue" by the herdsmen, scrofula and osteosarcoma by veterinary surgeons. The vegetable parasite which gives rise to it was first described in cattle by Bollinger in 1877, and named Actinomyces (*i. e.* ray-fungus).

In man it has probably been often mistaken for tubercular nodules and caseous inflammation or sarcomata, in the liver or lungs.

The first two undoubted cases published were by Dr James Israel, of Berlin,

in 1878,* one a man of thirty-nine, with multiple abscesses and serous inflammations, and the other a man of thirty-six, with a submaxillary abscess, who recovered. In both the characteristic nodules and the "club-shaped or pear-shaped bodies" are unmistakable (pl. i, fig. 2; iii, fig. 5). In his article he publishes notes of a third case observed by Professor von Langenbeck at Kiel in 1845 of vertebral caries in a man; and the drawings then made and reproduced by Israel (pl. iii, fig. 9, *a* and *c*), prove that it was Actinomycosis. Ponfick first recognised these *Mycoses* to be the Actinomyces discovered in cattle by Bollinger. He has since published a monograph on the subject ('Die Actinomycose des Menschen,' 1882).

A remarkable case was brought before the Hunterian Society by Dr F. C. Turner (March, 1887). Here it was combined with true tubercular disease, probably of independent origin.

Numerous cases in the jaws and tongue of cattle are in the Museum of the Royal College of Surgeons (Nos. 2254, *b*, *c*, &c., and 2274, *b*, &c.).

The remarkable peculiarity of the disease is that each inflammatory nodule or abscess has in its centre a parasitic fungous growth, which varies from the 100th to the 10th of an inch in diameter. In one of the earlier cases observed in this country a section from the morbid tissues of the tongue of an ox was shown the writer by Professor Roy at the Brown Institute. The large size, glittering aspect, globular shape, and surrounding mass of radiating club-shaped structures distinguished the organism at once from any known morbid product, and suggested the presence of lime salts or some other crystalline material. This, however, is not the case.†

Actinomyces, as the fungus was named by the botanist Hartz in allusion to its radiating gonidia, is not one of the group of Schizomycetes to which the micrococci, sarcinæ, bacteria, bacilli, and vibriones of anthrax, glanders, and other specific contagious febrile diseases belong. It is apparently one of the Hyphomycetes, the group to which moulds like *Penicillium glaucum* and *Mucor* belong. The centre of each minute yellowish nodule consists of an immense number of interlacing threads, apparently mycelium.

The mode of invasion of Actinomycosis is unknown. It is remarkable that it has never been traced to direct contagion from cattle, and it is possible that both cattle and man derive it from some vegetable source, and that it is introduced through carious teeth, or directly from the mouth, intestines, or bronchi.

When established in a living tissue each mass reproduces by gemmation, and so spreads in its neighbourhood. As it grows, a zone forms around it of large nucleated cells, not unlike giant-cells in appearance. They are surrounded by leucocytes (inflammatory or exudation corpuscles), and these again, in the larger and older specimens, by a capsule of spindle-cells or fibrous tissue. The inflammatory nodules thus formed coalesce and gradually form large tumours. While still scattered and small in the lungs they much resemble tubercles. The characteristic rosettes of club-

* 'Virchow's Archiv,' 1878, vol. lxxiv, p. 15, "Neue Beobachtungen auf dem Gebiete der Mykosen des Menschen"—'Klin. Beitr. zur Kenntniss der Actinomycose des Menschen,' Berlin, 1885. Translated in abstract for the New Sydenham Soc. in their vol. on 'Microparasites in Disease,' by Dr Hime, of Bradford, 1886.

† See Israel's original paper above quoted, where it is stated that Dr Ferd. Cohn, who recognised the fungus, at first thought them to be fat crystals; also the discussion on Dr John Harley's case of so-called "Actinomycosis of the Liver," 'Med.-Chir. Trans.,' 1886, p. 135, and 'Proc.,' N.S. vol. ii, p. 20.

shaped glistening cylinders stain strongly with eosin, or magenta and picric acid.

As we should expect from the botanical character of the parasite, it does not infect the blood, lymph, and tissues like bacteria and their allies, but acts as a local irritant, spreading slowly, and limited in its action to the tissues and organs successively affected. Pathologically, therefore, actinomycosis does not belong to the same group of acute febrile infective diseases as anthrax and glanders. It belongs to a group well represented in the pathology of the lower animals, of which the *pébrine* of silkworms is a well-known example. The *Saprolegnia*, which causes salmon disease, is another. Grobe, Loch, and other pathologists have produced artificial "mycosis" of the kidneys and other viscera in the rabbit by injection of the spores of *Aspergillus*. In Favus and Ringworm, as in Thrush, the parasite is a fungus; but, owing to its not penetrating the skin or mucous membrane, its effects are only superficial. It is possible, however, that cases may hereafter be recognised in man of other internal diseases due to invasion of parasitic fungi, and therefore analogous to actinomycosis.*

The fungus is difficult to cultivate out of the body. Recently, however, Boström is said to have succeeded, and his results throw doubt on its position among Hyphomycetes.† Inoculation has generally failed, but Ponfick has succeeded in reproducing the disease in calves.

There are nearly a hundred cases (some not certainly genuine‡) now recorded of this disease in man. Usually, as in cattle, it affects the mouth and jaw, sometimes the bronchi and lungs or the intestines and peritoneum, and sometimes the liver only. Two of the last group were described by Mr Shattock ('Path. Trans.,' 1885), one of them an old specimen from the Museum of St. Thomas's Hospital, and Dr John Harley's case is a third.

The fungus differs considerably in some recorded cases in man from the characters described in cattle. Whether these are specific differences or depend on a stage of development or some other modifying cause remains to be seen. (Compare Dr Theodore Acland's figures, 'Path. Trans.,' 1886, pl. xxv, with those in Dr Harley's case, and both with Israel's, Ponfick's, and Crookshank's drawings.)

When the tumours or abscesses are accessible to surgical treatment, actinomycosis may be cured both in animals and in man. Hence the best prognosis is when the disease affects the tongue, jaws, or neck, and the worst when it affects the lungs or the liver.

In a remarkable case, seen by the writer with Dr Ransom, of Nottingham, the patient, a strong and healthy man, of about fifty, was attacked with what appeared to be typhlitis followed by extensive suppuration in the subperitoneal tissues, and at last by abscess of the liver. The correct diagnosis was made by Dr Ransom discovering in the pus discharged after incision minute brown granules, just visible to the naked eye, which collected at the bottom of a glass, and showed the characteristic club-shaped structures above described.

* In Dr Paltauf's fatal case of *Mycosis mucorina* in man, the fungus invaded the body through the intestines, and led to abscesses in the lungs, brain, and other organs (see 'Virchow's Archiv,' vol. cii, with references to fungi in the air-passages, &c., p. 543).

† 'Trans. German Med. Congress of 1885' (Crookshank's "Bacteriology," p. 217).

‡ One, for instance, tabulated by Israel, is Mr Treves's "Case of Supposed Actinomycosis" (as he rightly called it), which proved to be large-celled alveolar sarcoma, 'Path. Trans.,' 1884, p. 356.

HYDROPHOBIA.*—From the time of Aristotle it has been known that dogs are liable to a fatal disease which they transmit by their bite; and this disease, when occurring in man, was called "Hydrophobia," from the dread of water, which is one of its chief symptoms. In the lower animals, however, that very symptom is absent. Although the symptoms of this terrible disease are those of paroxysmal nervous convulsions, its natural place is in association with Glanders, Anthrax, and other specific diseases, transferred from animals to man.

Incubation.—Judging from what we know of the inoculation of poisons in general, we should have expected that the introduction of the virus of rabies through a bite would have produced its effect after a definite interval, and without much delay. The contrary, however, is the case. After the healing of the wound, which takes place naturally and quickly, there occurs a period of incubation, which is often prolonged beyond that of all other infective diseases, and which is of uncertain duration, both in the lower animals and in man. Bollinger, in Ziemssen's 'Handbuch,' says that in 60 per cent. of all cases in the human subject it is between eighteen and sixty days, in 6 per cent. between three and eighteen days, and in 34 per cent. longer than sixty days. How protracted it may be we cannot yet positively tell. Instances have been recorded in which three years, five years, even twelve years, were supposed to have elapsed. In 1854 there occurred at Guy's Hospital, in the practice of Dr Hughes, a case in which it was said that five or seven years had passed since the bite. It is possible, however, that in these cases a subsequent infection took place without the patient's knowing it. That this is possible must be admitted; for, on the one hand, the origin of hydrophobia cannot always be traced at all, although it is most unlikely that it ever arises *de novo;* and, on the other hand, Mr Youatt succeeded in tracing certain cases to sources that might very easily have been overlooked; one, in a man, to his having tried to untie with his teeth a knot in a cord by which a rabid dog had been confined; and another, in a woman, to her having used her teeth to press down the seam in mending a tear in her dress caused by the bite of a rabid animal.

The following are the results of inquiries as to the period between the bite and the appearance of symptoms in cases at Guy's Hospital. Six were published by Dr Bright (1820–30). Seven more were reported by Mr Cooper Forster in the 'Guy's Hospital Reports' for 1866 (Third Series, vol. xii, p. 1). The incubation in these thirteen cases varied between four weeks and eleven months, except one, in which it was supposed to be from five to seven years. In ten cases which have occurred since the publication of that report and the present year (1867–89), collected for this edition by Dr E. W. Goodall, the periods of incubation were nineteen weeks, thirty-five days, thirty-eight days, forty days, nine weeks, nearly twelve weeks, thirteen weeks, and about twelve weeks respectively, and, more doubtfully, four weeks, while in the tenth case (a man aged thirty-six, who died in 1878) no certain evidence could be obtained, either from the patient or his friends, that he had ever been bitten by a dog. No doubt the period of incubation varies more than in other specific diseases, but in the most authentic and well-observed cases it is rarely more than twelve months.

In 132 cases of hydrophobia, selected by the Registrar-General (1886) on account of the circumstances being accurately known, the shortest incuba-

* *Synonyms.*—Rabies canina—Lyssa (λύσσα, raging madness).—Fr. La rage—Germ. Hundswuth, Wasserscheu.

tion was eleven days, in a child bitten by a rabid cat. In 23 cases it was under a month, in 64 between one and two months, in 21 between two and three months, in 124 it was under five months, in 127 under ten months, and in 130 under two years. In one case it was between three and four years and in one other above four years.

The most frequent incubation period is about six weeks. When the infecting wound is on the face the incubation is probably shorter. In children also it is usually shorter than in adults.

Experimental inoculation in dogs, rabbits, and other animals shows, on the whole, shorter periods than when the disease dates from the infliction of a bite by a rabid dog; and when the virus is introduced, not subcutaneously but beneath the dura mater after trephining the skull, the period of incubation is measured by days, a week being a very frequent time.*

Prodroma.—In some cases the earliest indication of the onset of hydrophobia is afforded by an uneasy sensation in the seat of the bite, which becomes painful, or tingles, or itches, or feels cold; sometimes the cicatrix itself may be seen to be reddened, livid, or swollen; perhaps it may open afresh, or a papular eruption may appear around it. The pain or other morbid sensation extends upwards along the nerves; from the hand, for example, into the arm and up to the shoulder, as in a coachman whose case is recorded by Sir Thomas Watson. In a gentleman who came under the care of Mr Cooper Forster in 1866 the pain was of extraordinary severity; it came on in paroxysms, obliging him to stop suddenly in the street, and to cry out; it was not referred to the course of any particular nerve. There was in that instance no redness or tenderness of the cicatrix. The arm, however, felt much colder than the other one. He had been bitten eleven months previously; in the intervening period of time he had three or four times complained of pain up the arm and twitchings in the hand.

There is probably no tendency to lymphatic inflammation beyond what might arise after any other injury of equal severity. Watson cites two instances of such an occurrence; but in one of them, at any rate, the red lines which extended up the patient's arm were probably due to the caustic which had been applied.

Other early symptoms are a peculiar restlessness, irritability, and depression of spirits. The patient suffers from nausea and loss of appetite; he complains of headache, is sleepless, and has a distressing sense of apprehension. It is curious that he often says nothing about having been bitten, and will even vehemently deny it. And yet he may make other remarks which show that his mind is dwelling on the subject, and that he is trying to persuade himself that he need not be afraid. The most characteristic symptom is a repugnance to fluid, which may show itself in various ways. Sometimes there is a little difficulty in swallowing, from a feeling of tightness about the throat. Sir Thomas Watson's patient, the coachman, refrained on account of a similar sensation from sponging himself as usual with cold water, though he remarked that he "could not think how he could be so silly." Often the breathing is interrupted by frequent sighs, which may stop the man in the middle of a sentence.

Symptoms when developed.—The above prodromal or early stage generally lasts from one to three days, but sometimes it is altogether absent. In

* See "Horsley's Lecture before the Epidemiological Society," Feb. 13th, 1889 ('Brit. Med. Journ.,' p. 342).

either case, the full development of the disease is ushered in by the sudden occurrence of violent convulsive paroxysms, affecting the muscles of deglutition and those of respiration, and repeated at more or less frequent intervals. They are brought on by a variety of causes. The attempt to drink almost always instantly precipitates one; the sight of fluid in a basin often has a similar effect. Watson mentions a case in which the patient was thrown into a violent state of agitation by hearing a dresser, who was sitting up with him, make water. The least draught of air, the waving of a mirror before the eyes, the opening or shutting of a door, the slightest touch upon the skin, the attempt to comb the hair, may each excite an attack. Sometimes the patient succeeds in gulping down a little fluid by carrying it to his mouth with the eyes shut, or he may insist on the withdrawal of the bystanders as enabling him to swallow better. Watson saw one man who so dreaded anyone's breath on his face that he would not converse with the apothecary of the hospital except in such a position that the chin of each of them rested on the other's shoulder. The seizures often consist of a series of shuddering or sobbing movements, more or less like those which occur when one steps into a cold bath. Sometimes, however, they are attended with tonic spasms of the cervical muscles, the sterno-mastoidei (for example) starting forwards strongly.

The following description of the more violent paroxysms was drawn up by Dr Bushell, now Physician to the British Legation at Pekin, from a case which occurred while he was dresser to Mr Cooper Forster. "At the onset of a severe spasm the patient springs up in bed, and puts his hands furiously to his throat, as if to tear something away; the head is thrown violently back, the mouth is opened, and the eyeballs are protruded; then he makes several expiratory efforts, sometimes with a shrill screaming cry; the head is thrown violently from side to side; the hands are tossed wildly about, beating his chest, and striking anything that is near." Sometimes the jaws are sharply brought together, so that one can hardly tell whether the patient is not trying to bite those about him. The production of a barking noise, like that of a dog, appears to be fabulous. Bollinger denies that trismus ever occurs, or a general tonic spasm like that which characterises tetanus. Nor is there an authentic case in which complete opisthotonos was present, although Dr. John Ogle described one in which there was said to be emprosthotonos ('Med.-Chir. Rev.,' 1868). Sometimes these convulsions seem to be spontaneous, but often they can be recognised as reflex spasms from a sensory excitation. The skin is in a state of hyperæsthesia, particularly to effects of temperature.

The duration of the paroxysms is variable. According to Bollinger, they occasionally last from thirty to forty-five minutes. When they are at all protracted, the patient's face and limbs become pale and livid and covered with sweat. The countenance assumes an aspect of terror, and the pupils are widely dilated. The pulse is rapid, the respiration also quickened, irregular, and more or less forced.

The temperature varies greatly. It is usually moderately raised, sometimes normal, and occasionally rises to 103° or even 105°, as in a case noted by Mr Southam, of Manchester ('Brit. Med. Journ.,' 1881, vol. ii, p. 814).

The urine appears to be normal as a rule; sometimes febrile, rarely albuminous. The occasional presence of glycose is perhaps due to inhalation of chloroform.

In women and young children the course of hydrophobia is often com-

paratively mild, although it is even more quickly fatal; this was so in two of Sir Thomas Watson's cases, one in a lady aged thirty-two, the other in a girl only five years old, and also in a little girl who died in Guy's Hospital in 1875. She was so slightly affected that Mr Stocker supposed the case to be one of hysteria. In other instances, however, children affected by hydrophobia exhibit the most frantic mania.

In some very rare instances the paroxysms are said to be altogether absent, so that the patient is able to drink, though with difficulty, throughout the disease. But, as a rule, there is an almost absolute inability to swallow, even during the intervals. The patient, who is much distressed by thirst, may resolutely carry the cup or the glass to his lips, but as soon as a little fluid has entered his mouth it is forcibly ejected, and all the rest is spilt over his clothes. The tongue is said to be generally clean and moist; but the fauces and the palate may be seen to be injected, and their glands are swollen. A viscid saliva which collects in the mouth is a source of great annoyance; it is hawked up with noisy effort, and spat out upon the floor in all directions, or upon the faces and clothes of the nurses.

Another symptom, which is present in certain cases, is priapism, with frequent involuntary emissions. Trousseau observed painful hyperæsthesia of the genital organs.

The mental state, even between the convulsive attacks, is almost always one of great agitation. As the disease advances, the patient often raves at those around him, accusing them of being the cause of his illness, and complaining of their gazing upon his misery. Even if what he says is not nonsense, he generally talks hastily and excitedly. Sometimes he is wildly maniacal, so that he has to be confined in a strait jacket, or to be held down by several powerful men.

Not infrequently death occurs by suffocation in the middle of a paroxysm, and more or less suddenly. The whole *duration of the disease* is seldom a week, and sometimes it terminates within from twelve to forty-eight hours. In eight cases death occurred in twenty-four hours, in eighty-seven between the first and the seventh day, in five on the eighth, and in one case on the ninth, tenth, and twelfth days respectively (Registrar-General's 'Report,' 1886).

Cases which last a week are exceptional; Sir Thomas Watson's coachman did not die until the middle of the seventh day. In that instance the end was very gradual; the pulse grew rapid and weak, the mental powers failed, he lay moaning and tossing from side to side, frothy saliva ran from his mouth, he lost the power of moving his left arm, fluid stools were passed involuntarily, the lower extremities grew cold, and the coldness extended up to the chest. Some patients have towards the last become paraplegic. In the more protracted cases the spasms may cease for some hours before death, the patient be quite calm and able to talk or drink or wash his hands without discomfort, but nevertheless the coldness of his surface, and the absence of pulse at the wrist, show that there is no real improvement.

Contagion from rabies.—Hydrophobia is doubtless caused in all cases by the transference to the patient of the specific virus of rabies; but as to the conditions under which this may occur, many interesting questions arise. Thus it has not yet been finally determined whether the disease is under any conditions communicable from one human being to another. No case is known of the kind, and medical men and nurses always escape, although the poisonous secretion is often ejected upon their clothes and hands and

faces. In former times the dread of catching the disease was so great that the patient was often smothered between feather beds by his relations, or was allowed to bleed to death through an opened vein. Bollinger says that on the military frontier of Austria persons labouring under hydrophobia, or suspected of it, are even now liable to be shot by their neighbours, and that those who have been bitten by rabid dogs sometimes commit suicide.

There is no doubt that the virus resides in the saliva and salivary glands. Magendie long ago produced rabies in dogs by inoculating them with the saliva of hydrophobic patients. Pasteur has now proved that the spinal cord is also the seat of the virus; and that inoculations from it, especially if introduced under the dura mater of a dog, after trephining the skull, will reproduce the disease in dogs and rabbits.

The disease in animals.—Rabies may occur in many kinds of animals besides dogs. It is common in wolves, jackals, and foxes, and the bite of a rabid wolf is notoriously the most dangerous of all. Cats are sometimes affected by it, but far less frequently than dogs. A scratch from a cat is believed to have conveyed it to a child, no doubt from the claws having been impregnated with poisonous saliva. Among herbivora, horses, oxen, goats, sheep, pigs, rabbits, and guinea-pigs are capable of being infected experimentally by inoculation, or if they are bitten by rabid dogs. Mr Youatt recorded a case in which a groom took hydrophobia through a scratch which he received from the tooth of a rabid horse.

Rabies broke out as a destructive epidemic among the fallow-deer at Richmond in 1889, and soon afterwards in the Marquis of Bristol's park. More than 450 died out of a herd of between 600 and 700 in the course of three months.*

A most important question is what indications should lead one to decide, when a person is bitten by a dog, that the animal is rabid. Mr Youatt met with cases in which a dog exhibited no symptom of rabies when it inflicted the fatal bite, though it was soon afterwards attacked. No fewer than eighteen or nineteen instances of this kind have been collected. The simplest way of accounting for them is to suppose that the disease is infective even during its period of incubation, which in dogs is believed to be generally of from three to five weeks' duration, but occasionally to be prolonged over as many months. It is evident, therefore, that when a dog is killed soon after having bitten a human being, one can never be quite sure that hydrophobia may not supervene. The proper course is to keep the animal securely confined for some weeks.

There are two varieties of the disease in dogs; one characterised by maniacal excitement, the other by paralysis of the jaw, so that it hangs down and allows a frothy saliva to run out of the mouth. In each form the bark is altered into what is described as a "hoarse inward sound, dissimilar from its usual tone, and generally terminating with a peculiar howl." Towards the last the hind legs and the loins become paralysed, so that the dog staggers about and falls. The popular distinction between ordinary rabies and "dumb rabies" with paralysis is not without foundation.

One of the earlier symptoms is an extraordinary perversion of the appetite, the animal eating hair, straw, sticks, bits of leather, earth, stones, and other substances, which remain undigested and may be discovered in the stomach after death, mixed with a dirty-brown pulpy fluid. Their pre-

* See Mr Adami's interesting account in the 'Brit. Med. Journ.' for Oct. 12th, 1889.

sence often affords a valuable indirect proof that a dog which has been killed under the suspicion of rabies was really affected with that disease. It has been said that a verdict of acquittal may be given whenever the digested remains of the animal's natural food are found in its stomach and chyme in its small intestine; but of course the disease might even then have been incubating. Change of disposition, snapping, hiding, wandering, are all indications of rabies in a dog.

As already remarked, a dread of water is not a symptom of rabies in any animal except man. "Mad dogs," as they are commonly called, plunge their muzzles into water and lap it up eagerly (for they are very thirsty) although they may not be able to swallow.

Rabies is always fatal in dogs—usually in a week after the symptoms have appeared, occasionally after nine or ten days.

In *rabbits* the symptoms of rabies (transmitted from dogs) are like those of dumb madness, in the absence of excitement and the development of paraplegia, which, as in dogs, takes the form of "acute ascending paralysis." A man who was bitten by a rabid cat in 1886 died under Dr Bristowe in St Thomas's Hospital with symptoms of this kind.

The study of the disease when reproduced by inoculation in animals shows a very similar series of symptoms to those which are characteristic in man. There is first a stage of excitement with visual delusions; then hyperæsthesia with reflex spasms; next the stage of mania, and (particularly in rabbits) paraplegia, corresponding to the "dumb rabies" of dogs; and lastly, death, often by sudden failure of the heart.

Distribution and frequency in man.—At one time hydrophobia was supposed to occur chiefly in temperate climates, but this is not the case.* The only part of the world in which it is as yet unknown is Australia. Like other specific diseases it is often absent from a town for several years together, until some accident introduces it and it becomes epidemic. It has recently been much more common in London than for many years before.

Between 1820 and 1830 six cases of hydrophobia occurred in Guy's Hospital. Two occurred in 1831, one in 1837, and then none for nearly twenty years. In 1856 there was a single case admitted, in 1865 two, and in 1866 the case of a private patient of Mr Forster's was alone recorded. Then again there was a pause for several years, the next case occurring in 1874. In 1875 another was admitted, three in 1877, and one in 1878. After five years' interval there was one case in 1883, two in 1885, and one in 1886. None have been admitted during the last three years (1887–8–9).

For London, the Registrar-General's returns show twelve deaths from hydrophobia in 1838, and four in 1839. Then only one, three, four, two, three, two, in the successive years to 1845, none in 1847, '49, and '52, and only one in 1846, '48, '50, '51, and '53. Seven were returned in 1854, and two in 1855 and '57. None in 1856 and none in 1858, '59, '60, '61, and '62. Two in 1863, and none again in 1864, but nine in 1865, and six in 1866. There were three in 1867 and '69, none in 1868 and '70; one in 1871, '72, and '73. Then there were nine in 1874, six in 1875, six in 1876, and sixteen in 1877; five in 1878, two in 1879, three in 1880, five in 1881, four in 1882, eight in 1883, nine in 1884, and in 1885 no less

* The following statement of the deaths from hydrophobia which occurred in the Punjab is taken from a report by Brigade-Surgeon Bellew:—Population of the province, 17,514,978. Deaths from hydrophobia: in 1879, 69; 1880, 107; 1881, 139; 1882, 128 1883, 117; 1884, 158; 1885, 146.

than twenty-seven. In 1886 the number suddenly fell to nine—after muzzling was enforced—and in 1887 and 1888 there was not a single death. In twenty years (1869–88) there were 780 deaths in England and Wales.

In England, the greatest number of cases of hydrophobia occur in Lancashire, Cheshire, and the West Riding of Yorkshire, and the next greatest number in London and the Home counties.

According to the popular belief, the disease is more frequent in the hot season than during winter and spring. Of 132 cases, throughout England and Wales, fifty-one occurred in July, August, and September.

Rivers sometimes seem to limit the diffusion of hydrophobia in a remarkable manner. Schrader has stated that in 1852 and 1853, when it was raging in Hamburg and in the neighbourhood on both sides of the Elbe, no cases occurred on the islands in that stream.

Only some of those who are bitten by rabid animals are attacked by hydrophobia; several writers agree in fixing the proportion at about 50 per cent., but this is certainly too high. Inasmuch as the disease if left to itself is, so far as is known, always fatal, we may take the mortality among persons bitten by rabid dogs as indicative of the frequency with which the disease is developed. The statistics in the department of the Seine for 1887, reported by M. Dujardin Beaumetz, and those of the county of Lancaster for the same year very nearly agree: in and near Paris there were 7 deaths out of 44 bitten; in Lancashire 6 deaths among 36 bitten, or about 16 per cent. Some of those who escape perhaps owe their immunity to an idiosyncrasy which renders them insusceptible of the virus; at least this appears to be the case in the dog. Sir Thomas Watson mentions that a dog was caused to be bitten repeatedly, but without any result.

Another circumstance affecting the transmission of the disease is the presence of clothes or other coverings, by which the teeth are wiped clean of the virus before they penetrate the skin. The undoubted fact that rabid wolves are more dangerous than dogs may be due to their flying straight at the naked throat. Youatt and all modern writers are agreed that a breach of the cutaneous surface is necessary to allow of the entrance of the poison; in Sir Thomas Watson's oft-quoted case, however, both the coachman and his fellow-servants declared that the terrier dog by which he was inoculated had drawn no blood, but merely indented the skin of the hand: the saliva had (if the observation was accurate) entered the lymph channels by the *rete mucosum*. Mucous membranes may perhaps be capable of infection without any previous lesion. A case is recorded in which a rabid dog licked the face of a sleeping man near his mouth, and communicated the disease, although not the slightest scratch or abrasion could be found after a strict search.

The large experience recently obtained at the Pasteur Institute in Paris has amply confirmed Mr Forster's belief that bites on the face are the most fatal, and probably they have the shortest incubation. Next come the hands, and lastly, the parts habitually covered.

A question of some consequence to pathologists is whether the virus retains its powers after the death of a rabid animal. Mr Youatt thought that this was not the case; but Bollinger cites the case of a student at the Veterinary College at Copenhagen, who opened the body of a dog that had died of the disease the night before; his finger was slightly fissured at the time, and about six weeks afterwards he died of hydrophobia. The poison, however, appears to have little power of resisting decomposition, for no case

appears to be recorded in which it has been transmitted by fomites, or in any other way than directly from the saliva, or (experimentally) from the nervous centres.

Seat.—Until lately it was supposed that hydrophobia, like other neuroses, has no morbid anatomy, at least so far as the nervous centres are concerned. But Benedikt, as early as 1875 ('Virchow's Archiv' for that year, p. 537), and Dr Gowers ('Path. Trans.,' 1877), Dr Coats ('Med.-Chir. Trans.,' 1878), and Dr Ross ('Path. Trans.,' 1879) have found that the vessels in the bulb towards the floor of the fourth ventricle are surrounded by masses of leucocytes within their sheaths, and that collections of these exuded cells, forming miliary abscesses, may occur among the nervous elements. Similar changes, but less marked, were also found in the spinal cord. They do not, however, occur in all cases, for in some beautiful preparations made by Dr Frederick Taylor no deviations from the normal appearances are to be recognised. On the other hand, preparations made from the patient who died in our wards in 1885, by Dr Hale White, showed unmistakable extravasation of leucocytes, and here and there of blood-discs, in the sheaths around the vessels in the bulb. In the spinal cord and in the brain the lesions were more scanty and doubtful.

Experimental evidence confirms the belief that the cord and bulb is the seat of the disease.

Other conditions, more or less constantly found after death, are congestion of the mucous membrane of the pharynx and epiglottis, of the stomach with hæmorrhagic erosion, and of the lungs. They are more uniform in rabbits than in human beings.

Pathology.—Hydrophobia offers some of the most difficult problems in the science of medicine. What becomes of the virus during the prolonged and indefinite stage of incubation? What new process gives rise to the first characteristic symptoms? Some writers suppose that a "recrudescence" takes place, the poison having hitherto been imprisoned in the wound, but being at this time absorbed in the blood. One cannot imagine that hydrophobia is due merely to a transference of morbid action along the nerves (a peripheral ascending *neuritis migrans*, such as perhaps occurs in tetanus), for fresh poison is generated, so that the saliva and even the blood become infective. Indeed, if it is the fact that a dog can communicate the disease during the period of incubation, the whole theory of recrudescence must be given up.

There can, however, be no reasonable doubt since Pasteur's experiments, to be presently described, have been made, that hydrophobia is a specific contagious disease, a "zymotic" "blood-poison." The microbe on which it probably depends has not yet been discovered.

Diagnosis.—Hydrophobia is not generally difficult to recognise. There has been no doubt as to the nature of any of the cases, all of them fatal, that have been observed at Guy's Hospital within the last few years. One of the oddest of all vagaries of medical opinion is the notion that there is really no such disease, and that all the persons who are supposed to have died of hydrophobia have really succumbed either to traumatic tetanus or to fright. This idea seems to have been started early in the century by Bocquillon, and it was upheld by Sir Isaac Pennington, Regius Professor of Physic at Cambridge, and more recently by Prof. Maschka, of Prague. But as Sir Thomas Watson long ago remarked, young children and idiots, who could not have understood anything about the disease, have died of it, and many

of the adults who have been attacked have been men of strong mind who refused to believe that they were seriously ill. As for tetanus, the symptoms in most cases are altogether different.

It must, however, be admitted that medical literature contains a considerable number of well-recorded cases, concerning which it is perhaps impossible to say positively whether they were instances of hydrophobia or not. The difficulty is with regard to those cases in which recovery is supposed to have taken place. For all experience tends to show that whenever the clinical characters of hydrophobia are present in a typical form the patient invariably dies, or, in other words, nearly every case in which the fatal issue has been arrested seems to have presented some feature or other which casts a doubt upon its genuineness. And yet one cannot deny that, taken as a whole, the symptoms have often resembled those of hydrophobia more closely than those of any other known disease.*

In some cases of epilepsy, hysteria, or mania, there is spasmodic difficulty in swallowing liquids which may be more or less like that which occurs in hydrophobia. But the doubt, if any, is soon dispelled by the progress of the disease.

Prophylaxis.—If all rabid animals could be alone destroyed it would be possible to "stamp" out the disease, but this is impossible, for beside domestic dogs we have to account for wolves and other animals.

A more hopeful plan is to prevent dogs from biting by enforcing the use of a properly constructed muzzle. By this means hydrophobia has been banished from Berlin; and its success when used in London has been remarkable. In 1885 twenty-seven persons died of hydrophobia in London, beside twelve in the suburbs. In December of that year dogs were muzzled. In 1886 only nine persons died of the disease, and those persons were bitten before the muzzling order was issued. In December, 1886, the order lapsed, and was not renewed. In 1887 and 1888 there were no deaths from hydrophobia in or around London. Between February and October, 1889, ten deaths have occurred.

Treatment.—The treatment of hydrophobia by drugs is hopeless. Morphia,

* Here, for example, is a case recorded by Dr Nicholls, of Chelmsford, in the 'Lancet' for 1878. The patient, a carter, aged twenty-five, was bitten by a stray dog in a neighbourhood where there had recently been a case of hydrophobia, and where several dogs known to have been bitten were still at large. Some weeks afterwards, having in the meantime thought little about the matter, he became weary, his legs ached, and he had loss of appetite. Two days later he refused a glass of ale at a customer's house, saying he could not drink it. On the fourth day he drank a quantity of coffee, but said that he "gulped it down," and complained at intervals of his throat. He was also much annoyed by trifling noises, and particularly by a toy windmill outside his house. On the fifth day, having delivered a load of coals in the country, he called at a public-house, and after three efforts got down a pint of ale. He then muttered that he was as thirsty as ever, wherefore another cup was passed to him, but on attempting to sip from it he failed. After this he drove into the town, dashed through the streets at a gallop, and became maniacal and unconscious. Dr Nicholls was called to him and found him with a rope tied round his legs, struggling furiously, and beating his arms and head. He was uttering a peculiar noise, between a howl and a scream. His face was livid and covered with a cold sweat; his jaws were clenched; he was foaming at the mouth. Convulsions like those of tetanus then came on and continued at intervals for about twenty-four hours, being, however, controlled to some extent by inhalations of chloroform. The opisthotonos was extreme, the body resting on the head and heels for a minute at a time. On the evening of the sixth day he partially regained consciousness, and asked for drink, which he gulped down in small quantities, the greater portion being expelled from the mouth. Two days later he displayed a remarkable horror of anything white, such as a bandage, a basin, a white glove, &c., turning aside and becoming convulsed as soon as he saw them. By about a week from this time he was well. If the disease was not hydrophobia, what was it?

chloral hydrate, or inhalation of chloroform, relieves the distressing spasms for a time at least, if employed boldly, but do not even postpone the fatal issue. Curare is probably equally useless, and the same must be said of cannabis indica, atropine, cocaine, salol, and methane, as well as of quinine, mercury, and the other drugs employed by Morgan and the older physicians (see Mr Dowdeswell's paper in the 'Proc. Roy. Soc.' for 1887). Darkened rooms, hot baths, electricity, and every other appliance that reason or credulity could suggest have been fully tried with equally negative results.

Promoting free bleeding from the wound, sucking and washing it, and applying nitrate of silver or other suitable caustic,* are rational methods of treatment, and, if adopted early, may undoubtedly prevent the virus from reaching the general circulation, and thus save the patient's life.

The Buisson or sweating treatment, still used in Russia, has been tried and found utterly useless.

Since, however, the first edition of this chapter was written, everyone has heard of the researches of M. Pasteur and of his method of preventing or even curing hydrophobia by inoculation of an attenuated virus. The following is a brief account of these remarkable investigations.

Starting from the belief that hydrophobia in man and rabies in dogs and other animals is a contagious disease, Pasteur ascertained that the fresh cord and bulb of a rabid dog, when pounded up with sterilized broth makes a highly contagious materies morbi; and this, injected under the skin or into the veins, or, most certainly and rapidly of all, under the dura mater, will reproduce the disease in a dog, cat, rabbit, or any other susceptible animal. As above stated, the symptoms vary for different species and even for different individuals; but after death the cord will again furnish the contagium, so that the identity of the disease is proved.

Next he set to work to attenuate the virus, as he had that of anthrax (cf. *supra* p. 358); and devised a plan of drying the cords in a warm, dry, sterilized atmosphere (20° C. is the temperature chosen, or about 68° F.); by this means the virus is rendered less powerful, and becomes progressively weaker day by day. When broth, charged with these dried cords, is introduced into a dog, he undergoes the disease in a mild form, and can then without danger be inoculated with a stronger virus, until at last he is rendered "refractory" to the most recent and intense contagion, or to the effects of direct inoculation from a rabid dog by biting.

The efficacy of this prophylactic or "preventive" treatment (called "vaccination" by M. Pasteur) is not a matter of question. If protected and unprotected dogs or rabbits are bitten by the same rabid dog or cat, the former escape and the latter die.

It would therefore be theoretically possible by inoculating all living dogs to "stamp out" hydrophobia for ever; just as, if vaccination were made compulsory everywhere for a generation or two, smallpox would become extinct and vaccination itself no longer needful. But this is as impracticable for hydrophobia as it is at present hopeless for variola. All that can be done by way of prophylaxis is to diminish the number of dogs by taxation of the owners, to destroy masterless dogs, and to enforce the use of muzzles.

Pasteur next tried whether it is not possible to anticipate the march

* Mr Youatt did not practise excision, but trusted entirely to cauterisation with nitrate of silver. He himself was bitten seven times, and operated on 400 persons besides, among whom he had only one death, which he ascribed to fright.

of the disease, by taking advantage of its undoubtedly long incubation-period and using attenuated virus with a shorter period, so as to overtake the original virus and fortify the organism against it, before its natural effects appear. Trials of this "protective" method on dogs and rabbits already bitten proved encouraging; and at last, when urged to do something to save a child named Joseph Meister, who had been severely bitten by a mad dog, Pasteur, as he tells us, with great anxiety, inoculated him with attenuated virus on July 6th, 1885. The result proved satisfactory; the boy recovered, and many other persons bitten by rabid (or often, in all probability, by non-rabid) dogs applied to Pasteur, and received "protective' inoculation. The numbers increased enormously, patients arriving from all parts of France, from Italy, Russia, England, and even from America and India. A certain number of deaths with undoubted hydrophobic symptoms occurred, particularly among several Russians who had been bitten by rabid wolves. Pasteur's results were not completely confirmed by independent investigators, *e. g.* Professor von Frisch, of Vienna, and he was violently attacked, not only by fanatical opponents of experimental pathology, but by certain French physicians. The difficulties in the way of a sound conclusion are serious. For, first, no one knows how many of the people who have been inoculated had really been bitten by a rabid dog at all. Secondly, when a bite is inflicted on a part covered by clothing, the venomous saliva is generally wiped off, and so the person bitten escapes. Thirdly, many who were bitten had been well treated by caustics, and thus the virus may have been, and no doubt frequently was, destroyed before they were inoculated. Lastly, the proportion of men or animals who, from some "insusceptibility" (*i. e.* some individual unknown cause), escape the disease even when the virus is fairly injected is unknown, though, judging by analogy from syphilis and from vaccinia, probably a small number by comparison.*

The serious question has also to be considered whether the intended protective inoculation may not, if unwittingly employed on persons who have not really been infected before, produce a fatal form of the very disease against which it is supposed to protect.

It cannot be said that these questions are fully answered, or that all the difficulties have been surmounted. But a report, presented to the President of the Local Government Board by a Committee appointed for the purpose of investigating this subject,† goes far to prove to unprejudiced critics that not only has Pasteur discovered an efficient "preventive," *i. e.* strictly prophylactic treatment of hydrophobia—a fact of great scientific importance—but that he has also succeeded in applying the same method to the "protective" or therapeutical treatment of the same dreadful malady when it has already been contracted.

Among a total of 2682 persons inoculated at the Pasteur Institute, forty are said to have died of hydrophobia. Nine of these deaths occurred among forty-eight persons who had been bitten by wolves—a peculiarly dangerous kind of rabies, and met by a more rapid and probably more hazardous process of inoculation.‡

* See M. Roux's "Croonian Lecture before the Royal Society," reported in the 'Brit. Med. Journ.' for 1889, vol. i, p. 1269

† The Committee consisted of Sir James Paget, chairman, Sir Joseph Lister, Sir Henry Roscoe, Professor Burdon Sanderson, Dr Quain, Dr Brunton, and Dr Fleming, with Mr Victor Horsley as secretary.

‡ The ordinary method was to inject on the first day of treatment virus derived from

Excluding these, of 2634 persons bitten (or supposed to be bitten) by mad dogs, only thirty-three died, a percentage under 1·5, and far less than that assigned by any computation to cases treated in other ways or not treated at all.

Of 233 persons bitten by undoubtedly rabid animals (as proved by inoculation from the spinal cord or by rabies appearing in other animals bitten by them) only four died, instead of perhaps fifty and almost certainly twenty.

Of 186 persons bitten on the face (the most dangerous part) by undoubtedly rabid dogs, only one twentieth died, instead of the previously probable proportion of at least a fourth.

Ninety cases were personally investigated by Dr Burdon Sanderson, Dr Brunton, and Mr Horsley, who went to Paris for the purpose. Among them no death had occurred. In thirty-one there was no clear evidence that the dog which had bitten the patient was rabid; but in twenty-four the bite had been inflicted on an uncovered part of the skin by undoubtedly rabid dogs.

Lastly it appears certain that the ordinary inoculation as now practised by M. Pasteur is perfectly safe, although there is room for fear that the *méthode intensive* may be attended with a certain risk.

Whether the protection will last a lifetime, or longer than the two years or more which have now elapsed since it began to be practised, cannot of course be at present known. It is possible that further experience may modify the conclusion above stated; but no dispassionate critic can withhold his admiration from the insight and ingenuity, the zeal and perseverance, which M. Pasteur has displayed, or can doubt that humanity is indebted to him for what, on the lowest estimate, is a more than promising method of treating a frightful and otherwise hopeless disease.

a cord dried for fourteen days; on the second, that of a cord dried for thirteen days; on the third, that of a cord dried twelve days; on the fourth, that of an eleven days' cord; fifth, ten days; sixth, nine; seventh, eight; eighth, seven; ninth, six; and tenth, five days. These injections were afterwards made more frequent, and the increase of strength more rapid. But for very severe cases, like those of wolf-bites, Pasteur now uses a *méthode intensive*, injecting on the first day of treatment virus derived from cords dried for fourteen and thirteen days; on the second, for twelve and eleven; on the third, for eleven and ten; on the fourth, ten days twice; on the fifth, nine days twice; on the sixth, nine days again; on the seventh, eighth, and ninth, eight days; on the tenth, eleventh, and twelfth, seven days; on the thirteenth and fourteenth, six days; and on the fifteenth and last day of treatment virus of maximum intensity derived from a cord of only five days' drying.

DISEASES OF THE NERVOUS SYSTEM

Local or regional diseases compared with general and specific diseases—Principles of their classification and nomenclature—Order to be followed in this work. Nervous Diseases—Peculiarities of this group—Arrangement adopted.

THE group of specific fevers of which we have now concluded the survey is the only one which can be called "natural." They agree in ætiology, and in the nature of the disturbance set up by the exciting cause, *i. e.* in their pathology; they agree in being infectious; in their general symptoms and course, and particularly in their relations to time; in their protective power; and, lastly, they are usefully studied together from the practical points of view of diagnosis, prognosis, and treatment.

Even in this group, however, we have seen that some members are more closely related than others—the eruptive fevers, for instance; and some have been put together from such accidental characters as their being derived from the lower animals, or being confined to tropical climates. Moreover, as in all natural classifications, certain members of the group, as Tuberculosis, Ague, Actinomycosis, are wanting in some of its characters, and might, not without reason, have been excluded, while other specific febrile diseases have been postponed to later chapters, as Mumps and Whooping-cough, for practical convenience, or, as Rheumatic Fever, in order to group it with other diseases, different in pathology, but closely connected clinically. Lastly, some general diseases are in important respects local—witness Enterica and Diphtheria—while others, counted among local disorders, undoubtedly affect the whole organism, as Gout and Purpura, Leprosy and Pneumonia.

It is desirable to dwell on these considerations in order that the student may clearly understand the futility of attempts at a scientific nosology. Diseases are not natural objects. They are structural or functional disturbances, viewed from the human standpoint of life and death, comfort and pain. They are dissimilar in origin and nature, and therefore incapable of uniform classification. It follows that no one system of Nosology can produce a useful, a scientific, or even a symmetrical result.

If we classify diseases by their *origin* we can form certain large classes which would express true relations, but would be impracticable for adoption in a systematic treatise. For instance, we should collect together—*parasitic* and *contagious* disorders; *malarial* diseases; those directly the result of mechanical *injury*, of chemical irritants, or of heat—"traumatic" as they are sometimes called; those produced by exposure to *chills*, the "catarrhal," or, as some German writers unfortunately call them, "rheumatic" diseases; those produced by chemical *poisons* ingested, as lead colic, lead palsy, and saturnine gout, the "zinc-ague" of brassfounders, mercurial tremors and stomatitis; those produced by *drugs*, as urticaria from copaiba;

those produced by unwholesome *food*, as alcoholic delirium and cirrhosis, gout, and the long list of dietetic disorders.

Then would come the diseases of imperfect *development*, in intra-uterine life or in infancy, at dentition or at puberty, and the large and important series of morbid changes which are the result of premature or normally supervening *senile* changes—atheroma, fatty and fibrous degeneration, involution of the uterus or the ovaries or the breast; atrophy of certain organs and hypertrophy of others. These, when delayed to the full term, are the "natural" diseases which lead to a kindly death.

If we attempted a *pathological* classification we should place next to the specific fevers those maladies in which the same process of pyrexia is excited by local inflammation, and make a class of inflammatory fevers, as was done by Cullen and by Hildenbrand.

A nosology based on *morbid anatomy* would follow the lines traced by Rokitansky and his successors, but we have only to turn to the best systematic works on the subject to see how unsuitable such a plan would be for a treatise of medicine. Of many diseases we know neither the seat nor the cause; some whose cause we know have no anatomy, and many anatomical changes have no clinical history.

A *histological* classification would be impracticable as the basis of a complete system; although it is instructive to remember that serous membranes, lymphatic organs, mucous surfaces, bones, and secreting glands have points in common under morbid as well as under healthy conditions. We shall, indeed, find it convenient to group together primary diseases of the blood, diseases of the skin, and diseases of the joints.

The only remaining course open to one who plans a systematic treatise like the present is, after setting aside general specific diseases, to put those together which affect the several organs—the brain and spinal cord, the larynx and lungs, the heart, the digestive apparatus, and so on; and this plan is followed here, as it has been by Dr Bristowe and by other recent writers. Functional diseases are classed with those of the structures which subserve the disordered functions—neuralgia with neuritis, epilepsy with cerebral diseases, asthma with bronchitis. Diseases of uncertain seat are arranged according to their most important symptom, or according to custom and convenience. Thus, exophthalmic goitre goes with disorders of the circulation, and diabetes with those of the urine; for we have no satisfactory grounds for putting them anywhere else, and at least readers know where to find them.

Nomenclature.—Closely allied to classification is terminology. In the case of specific diseases with a constant origin and a definite course, the best names are those which, first, are meaningless; secondly, consist of a single word, and that short, distinctive, and euphonious; and, thirdly, can form adjectives. Syphilis and Typhus (not typhus fever) are two of the best names we have. When, however, the disease is less definite and certain, names otherwise good become ambiguous, as Erysipelas, Diphtheria, and Pneumonia.

In choosing names we must in the end be guided by usage—

Quem penes arbitrium est et jus et norma loquendi;

but we should follow the best usage; and in this country it is in every way desirable to keep, as nearly as may be, to the official nomenclature of the Royal College of Physicians and the Registrar-General.

Some names are vernacular, like Measles, Mumps, Smallpox, Shingles, Dengue, Glanders, Ague, Gout, and these are excellent

Others are Greek or Latin names, classical or barbarous, as Morbilli, Variola, Zona, Psoriasis, Lupus, Scabies, and these are as good or better. Similar names may be more or less successfully coined, as Diphtheria, Pertussis, Enterica, Equinia, Purpura, Leuchæmia.

Personal names are undesirable. They are seldom generally accepted; they are cumbersome, they are often ambiguous, and they are seldom historically accurate. Such are Pott's disease, Morbus Maculosus Werlhofii, Bell's palsy, Maladie de Raynaud, Graves' or Basedow's disease, Cruveilhier's paralysis, Parkinson's disease, Hodgkin's disease, Maladie de Charcot.

When the anatomical lesion which causes the symptoms is known, it furnishes the best designation of the whole train of structural and functional disturbance, as abscess of the liver, annular cylindrical carcinoma of the descending colon, lithic-acid renal calculus, cirrhosis of the liver, stenosis of the mitral valve, glioma of the middle lobe of the cerebellum, and so on. These, however, are rather diagnoses than names.

Better, perhaps, are the names which express the pathological processes which lead to the structural results, *e. g.* suppurative hepatitis, anterior polio-myelitis, tubercular peritonitis, acute yellow atrophy of the liver.

Often, when neither the structural change nor the morbid process is certainly known, we must be content with clinical names, denoting a recurrent group of physiological events—a "complex of symptoms" the German writers call it—as Epilepsy, Chorea, Asthma, Chlorosis, Scurvy. Many such terms—Dropsy, Jaundice, Hemiplegia—have now become symptoms rather than "diseases," steps to a further diagnosis. But often no further step is at present possible; the symptom is the disease (see p. 4).

The order in which we take the various local diseases is of little consequence. We shall here follow the traditional order of taking the most noble cavity, the Head, first; after the diseases of the brain and nervous system, will follow chapters on the diseases of the Chest; then will come diseases of the Abdomen and Digestive organs generally, of the Liver, the Kidneys, and the Spleen, with its lymphatic allies; then diseases of the Bones and Joints, diseases of the Blood, and, lastly, those of the Skin.

In entering upon the study of Nervous Diseases we begin the most difficult department of medicine. Unlike the lungs and heart, the brain works silently, so that no auscultatory phenomena accompany its disorders. From the abdominal viscera it differs in being beyond the reach of palpation and percussion, and in yielding no secretions for chemical examination. Hence *the symptoms of nervous disease are, almost without exception, those of perverted nervous function.*

We cannot absolutely separate the diseases of the nervous system into those which affect respectively the brain, the spinal cord, and the nerves; for the precise seat of many of these disorders is still obscure or doubtful; and there are affections which, although they begin in one of these great divisions of the nervous apparatus, afterwards involve the rest. Still, the arrangement adopted here will, as far as possible, be to take first the diseases of the Peripheral Nerves, next those of the Spinal Cord, then those of the Cerebral Centres, and lastly the functional disorders, which are conveniently grouped together as *Neuroses.*

AFFECTIONS OF THE NERVE-TRUNKS

> But pain is perfect misery, the worst
> Of evils, and, excessive, overturns
> All patience. MILTON.

NEURALGIA.—*Definition—Pathology and general characters—Trifacial neuralgia—Neuralgia of arm and trunk—Sciatica—Treatment of neuralgia.*

PERIPHERAL PARALYSIS.—*Causes—Motor palsies of vertebral nerves in particular—The reaction of degeneration—Sensory paralysis—Pain—Causalgia—Glossy skin and other trophic results—Diagnosis, prognosis, treatment—Paralysis of cranial nerves—Motor, of the portio dura, hypoglossal, and oculo-motor—Sensory, of the trifacial and olfactory—Treatment.*

PERIPHERAL NEURITIS.—*Its history and symptoms—Histology—Causes—Prognosis and treatment.*

THE affections of the nerve-trunks may be broadly divided into two clinical groups. In one *pain* is the principal symptom; these affections constitute the *Neuralgiæ*. In the other group the chief symptoms are *loss of muscular power and of cutaneous sensibility;* these are commonly called the *Peripheral Paralyses*, to distinguish them from similar conditions due to diseases of the brain or spinal cord.

NEURALGIA.—In the former of these two groups the principal, and sometimes the only symptom is *pain*. Pain, however, is a symptom common to many, indeed to most, diseases, and since it is a function of the nervous system, all pains are in one sense neuralgia.

Now, pain itself, although it is so familiar to everyone, seems nevertheless, to be an ultimate fact of sensation, incapable of definition or of explanation. It is not enough to say with Erb, that pain is "the reaction of the sensorium to a certain degree of excitation beyond that which would cause common sensation," for hyperæsthesia may exist without pain, and pain is often accompanied by anæsthesia. So different, indeed, are the two conditions of the sensory apparatus that some physiologists have been driven to the supposition that there are afferent nerves and centres for painful impressions, distinct from those for common sensation and for temperature.

Physiologically, pain may be excited by injury to one of the sensitive nerves in its course or at its periphery, or possibly by injury to its centre. The pain is in either case referred by consciousness to the peripheral distribution of the nerve. The most common cause of pain is increased pressure on the nerves, but mechanical injury, and chemical or electrical disturbance, may produce the same effect. Pressure on the papillæ and their touch-corpuscles or other tactile end-orders produces the sense of touch; when these are destroyed, as on the surface of an ulcer, no true sensation of touch is produced: but pain may still be excited. If, however, the pressure or injury passes a certain limit, pain disappears again and numbness or paralysis ensues.

Under ordinary circumstances, the impressions which give rise to pain are made upon the *terminal* filaments of a nerve; and the sufferer recognises as the seat of the pain the part to which the filaments in question are distributed. In neuralgia, on the other hand, the pain is said not to be excited by direct irritation of the distal extremities of the nerve to which it is referred.

If we accept this distinction, we must deny the pains of toothache when directly referred to the aching tooth to be true neuralgia, and we must admit as true neuralgia the pain produced by an aneurysm or other tumour pressing on the branches of nerves.

Practically, however, our knowledge of the special physiology of pain is not enough for us to be sure that the above distinction is valid in all cases, still less to apply it. We must therefore be content to call neuralgia that kind of pain which is so far as we know primary or idiopathic. When pain is not produced by pressure due to inflammatory engorgement of vessels (the most frequent cause), when it is not caused by injury or other direct cause, then we may call it neuralgia, at least provisionally. This is like the way in which we shall find that epilepsy and some other functional disorders of the nervous system are defined. Its justification lies in the empirical fact that the pains which on this diagnosis are called "neuralgia," have certain clinical characters in common which separate them from secondary pains. Before enumerating them, however, it should be mentioned that in two groups of cases we can give some explanation of neuralgia, which puts it on a separate pathological basis. In one, the neuralgia is really due to peripheral irritation, but this is not applied to the painful nerve, so that the patient is mistaken in his interpretation of the local sign. This is sometimes called "reflex neuralgia." A good instance is the trifacial neuralgia which is often excited by disease of a tooth. Such an affection is most readily explained by supposing that the irritation is directly transferred from one nerve-nucleus to another within the cerebro-spinal centres. In the other form of neuralgia, of which Sciatica may be taken as an example, there is some reason to believe that the morbid process begins in the trunk of the affected nerve, and that it is a form of local "neuritis" or perineuritis. If we were sure of this, it would be well to exclude sciatica from the category of neuralgia, and to class it with other inflammations of the peripheral nerves: the functional disorder would be recognised as organic, and the "disease" which was a symptom would become a local inflammation (cf. p. 4).

The clinical characters of neuralgia are as follows:

(1) The pain, instead of being only referred to the peripheral extremities of the nerves, is felt to shoot or dart along their course.

(2) The pain comes and goes; it rises rapidly to severe intensity and as rapidly subsides again.

(3) The pain is stabbing or piercing in character; not throbbing like that of inflammation, nor grinding like that of toothache, nor increased with movement like that of lumbago. It has not the disabling, ineffectual, baffled character of the pain which accompanies stricture, dyspnœa, or the efforts of defæcation or of childbirth, nor the sickening, paralysing pain of orchitis; but it is clear, sharp, and thin, high-pitched in quality, coming in bouts which force tears or cries from the sufferer, and leave behind them a massive, wearying after-swell of exhaustion.

(4) Certain "tender points" are developed, pressure upon which causes

increase of the patient's sufferings, while they are often, though not always, the seat of spontaneous shooting pains. It was Valleix who first drew attention to these *points douloureux*, as he termed them; and he maintained that they are of great importance in the diagnosis of neuralgia. He further showed that they are constant in position for each of the principal cutaneous nerves, corresponding generally with the spots at which they emerge from bony canals or from fasciæ. Trousseau afterwards laid stress upon the presence of an additional tender point at the spinous process of the vertebra, beneath which the affected nerve escapes from the spinal canal; this he terms the apophysial point. With regard to the frequency of the occurrence of "tender points" in cases of neuralgia there has been much diversity of opinion, dependent perhaps on the varied scope of the term neuralgia as used by different writers. Thus Eulenburg, who includes migraine, found tender points in only about half the cases which he examined. Anstie insisted on the fact that it is only when the pain reaches a certain degree of severity and of persistence that they develop themselves. It seems probable that they are, after all, merely spots at which nervous filaments happen to be so placed that they can be readily compressed. In cases of sciatica the external popliteal nerve is much more sensitive to pressure on the affected than on the healthy side, in that part of its course in which one can without difficulty feel it—namely, when it is running under cover of the biceps tendon.

(5) Another character of neuralgia, which is remarkably constant, and is often useful for diagnosis, is its limitation to the nerves of one half of the body. Even when it attacks a large number of nerve-trunks on the same side it seldom extends across to those of the opposite side; and it is never absolutely symmetrical, presenting the same degree of severity, and reaching the same extent of distribution, both to the right and to the left of the spinal column.

(6) A sixth characteristic of neuralgia is, that after its subsidence it leaves the affected parts tender to the touch.

Even with the help of these characters, it is often a question whether a given pain should be rightly termed neuralgia or not. The pain in the shoulder which attends abscess of the liver, the pain in the arm which is experienced in aortic aneurysm, the pain in the testicle which is caused by a renal calculus, are each typically neuralgic, because they are not due to lesions of the shoulder, the arm, or the testis; and so also are gastrodynia and angina pectoris, when they are not due directly to irritation of the terminal filaments of the gastric or the cardiac nerves. Even the pain in the side which accompanies hepatitis or pleurisy is in part neuralgic, for it often extends along the cutaneous branches of the affected nerves far beyond the area of inflammation, and the skin itself may be tender.

But it is doubtful whether the pain, "neuralgic" as it is in character, which accompanies the passage of a renal calculus should be described as neuralgia; and *migraine*, which was regarded as a form of neuralgia by all the older writers, and even by Eulenburg and Anstie, will be described in this volume, according to modern practice, as a separate neurosis. It is, indeed, certain, for reasons that will be given hereafter, that the seat of migraine is in the nervous centres themselves; but it is equally certain that the pain is referred to the cutaneous nerves of the scalp, and shoots and darts along in the course of their fibres. Moreover, in persons who have suffered from this disease for some time, true "tender points" are developed, exactly as in an ordinary neu-

ralgia; and other trophic changes occur, which show that a morbid influence has been reflected outwards upon the hair and other superficial tissues. Thus it does not appear easy to exclude migraine from the definition of neuralgia, while the common "reflex" forms are allowed to retain their places. Perhaps the difficulty may be removed by the consideration that the nerves of the scalp may fairly be supposed to bear the same relation to the brain beneath, which has been shown by Hilton and by van der Kolk to exist between the superficial nerves generally and the organs which lie beneath.* Just as almost any disease of the lung may be accompanied by pains referred to the nerves which are distributed to the skin over the chest, so we shall find that various affections of the brain and its membranes may be attended with pains running in the course of the frontal, the temporal, and the occipital nerves. The pains themselves are "neuralgic," but the presence of other symptoms show that the disease, as a whole, is something more than neuralgia.

However this may be, it is certain that neuralgia bears a close ætiological relation to the neuroses in general, as we shall find in studying the different local varieties of the disease.

Neuralgia of fifth nerve. Trifacial neuralgia. Prosopalgia. Tic douloureux.—Most cases in which pain is referred to the branches of the first division of the fifth nerve belong to Migraine; but there remain numerous instances in which the lower or middle branches, or all three at once, are affected; and among them is included the most severe of all neuralgiæ—a terrible malady, for which the most convenient name is, perhaps, *tic douloureux*. By Trousseau it is called "epileptiform neuralgia," apparently because it occurs in paroxysms with sudden onset, which so far resemble the attacks of epilepsy. It was described by Fothergill, in 1773, as "a painful affection of the face, distinct from the rheumatism and the ague in the face." True tic douloureux is happily very rare.

The absolute suddenness with which the pain of tic douloureux comes on is one of its most remarkable characters. The patient is perhaps sitting quietly reading, when he jumps up from his seat, and rushes up and down the room, with his hand forcibly pressed against his cheek; or he rocks himself backwards and forwards in the chair, crying out or uttering deep groans. In ten or twenty seconds, or a minute at the longest, the paroxysm is over. It ceases as abruptly as it began. The pain sometimes affects all the branches of the fifth nerve; sometimes only those of the second or of the third division. In certain cases, the muscles of the side of the face are thrown into violent spasms during the fit of pain, so that the patient makes horrible grimaces and contortions. Flushing of the face and eyes with lacrymation, usually following decided pallor, is very frequently present, and shows that vaso-motor and secretory fibres accompany the fifth nerve. The paroxysms may return every few minutes. Trousseau mentions one patient who had sometimes twenty in an hour; in the worse cases they do not intermit, even during the night. They are often brought on by movements of the jaws as in speaking, or in eating or drinking; and sometimes

* This remarkable "sympathy" between the viscera of the great cavities and the nerves (vaso-motor as well as sensory) of corresponding parts of the surface is probably referable to the fact that the visceral and parietal nerves were once united, before the splitting of the somatopleure and splanchnopleure, and are still connected, at their proximal point of union, with the centres of the several segments of the trunk.

pressure upon one of the teeth will instantly excite an attack. It is not worse at night, and is irregular in the time of its attacks. Sometimes remissions occur, the patient remaining free from the disease for several days together, or even for months. But sooner or later it returns, and is as severe as ever.

The "tender points" are well marked when tic douloureux has lasted for long; they are situated at some or all of the numerous spots at which branches of the fifth nerve emerge from bony channels, or perforate fasciæ. Pressure upon any of them is instantaneously followed by agonising pain, and a breath of cold air upon the face may have a similar effect.

As a result of the disease, when it is of long standing, the whiskers or beard disappear from the affected side of the face, the hair being worn off by friction. According to Trousseau, even the configuration of the bones may become altered, the malar eminence and the prominent part of the lower jaw being flattened down. Herpes zoster has been observed as the consequence of neuralgia, particularly when it affects the infra-orbital nerve. The hair of the scalp in supra-orbital neuralgia and of the beard in infra-orbital is sometimes turned white in the region and on the side of the affected nerve.

A patient who labours under tic douloureux acquires an expression of intense distress and suffering; his countenance is worn and wrinkled, and looks like that of a much older person. But, as Fothergill found, the disease scarcely ever occurs under forty, and often begins at a much later age. Anstie says that the worst case he ever saw was in a woman who was eighty years old when she was first attacked. According to this writer, a special feature in the ætiology of this form of neuralgia is that the sufferers from it almost invariably come of a stock which is tainted with insanity; indeed, they are themselves often the subjects of a suicidal melancholia, and their mental condition is almost always one of moody depression. This is no doubt partly caused by the severe pain which they have to endure, and partly by their inability to eat solid food; moreover, they are very apt to seek a temporary relief in drink, which brings its penalty in increased despondency afterwards.

Sir Thomas Watson gives an account of an autopsy which was made in the case of Dr Pemberton, a London physician of great repute in his day, whose career was ruined by this terrible disease. The os frontis was unusually thick, and a bony mass lay within the falx cerebri. It was supposed that these changes had set up an irritation which caused the neuralgia, but there is no evidence that any branch of the fifth nerve was involved in them, and outgrowths of bone of a precisely similar kind are often found in those who have had no pain in the head or face, nor any cerebral symptoms. Even in a celebrated case related by Romberg, in which the carotid artery was dilated to twice its usual size where it passed through the cavernous sinus, it seems very doubtful whether the so-called aneurysmal condition of the vessel had anything to do with the tic douloureux from which the patient had suffered on the corresponding side of the face; for that disease had existed for eighteen years at the time of his death, and one cannot suppose that the artery had been dilated during the whole of that time. The author has repeatedly seen a similar affection of the carotid at the same spot in the bodies of those who had made no complaint of neuralgic pain during life. Again, it is difficult to believe that the fifth nerve could have been pressed upon, without the sixth and the third

nerves having been first paralysed. Moreover, Romberg found other changes in the affected parts, the nature of which one cannot determine from his account of them, namely, a hard yellow nodule in the crus cerebri, and a softened state, with reddening, of the roots of the fifth nerve.

It does not appear that this form of neuralgia is ever of "reflex" origin or caused by peripheral irritation of any branch of the fifth or of other nerves. There is, indeed, Mr Jeffries' oft-quoted case, in which a triangular piece of china from a broken cup remained lodged in the cheek of a girl and gave rise for fourteen years to violent pains, which ceased a few weeks after its removal by excision; but that patient was too young to have suffered from true tic douloureux. Mr Tomes says that this complaint is never, so far as he knows, caused by irritation from diseased teeth; he quotes, as a warning, a case of Trousseau's, in which the pain came on when the patient touched with the tip of his finger his few remaining teeth, but in which they were extracted without the slightest benefit resulting. Patients, who have in vain had a large number of teeth removed, come to us with their disease unmitigated.

There is a less intense but much more common form of trifacial neuralgia, to which even young persons are liable, and which is often dependent on morbid conditions of the teeth. The pain shoots and darts along the branches of the fifth nerve, but it is generally accompanied with a dull aching or gnawing sensation. It is often distinctly paroxysmal, and (like all other forms of neuralgia) it is especially apt to come on when the patient is over-fatigued or exhausted from want of food. It is seldom severe enough to prevent him from attending to the duties or pleasures of life, although it may cause him great discomfort. In many cases it subsides at once if a glass of wine be taken, or a dose of quinine. According to Mr Tomes, the most frequent cause of this form of neuralgia is chronic inflammation of a tooth-pulp. But other conditions, which are mentioned by this writer and by Mr Salter as being capable of giving rise to it, are the difficult eruption of a wisdom-tooth (or its impaction in its socket), the presence of secondary dentine in a pulp cavity, exostosis, hypertrophy of the crusta petrosa, alveolar periostitis, decomposition of a dead pulp in a confined space, and even the exposure of sensitive dentine, or the crowding of the teeth together from insufficiency of room. In many instances the affected tooth is tender, so that sudden pressure on it, or the contact of substances much hotter or colder than itself, greatly augments the pain. Mr Salter has related one instance in which, although the offending teeth were painful when touched, doubt for a time prevailed as to the real cause of the neuralgia from which the patient suffered, because this returned again and again, there being only a temporary interval of ease after each tooth in succession had been extracted, until the last two were removed at one operation; when the pain entirely ceased. All the teeth had nodules of exostosis on their fangs. It might be expected that any affection of a tooth in the lower jaw would give rise to neuralgia in the course of the branches of the third division of the fifth nerve, and that a diseased tooth in the upper jaw would set up pain in the distribution of the second division. But this is not always the case. Indeed, Mr Salter mentions, as very frequent seats of "dental neuralgia," the supra-orbital nerve, the globe of the eye, the temple, and particularly a spot a little to one side of the vertex. The practical rule would seem to be that the only way of avoiding the risk of overlooking affections of the teeth as causes of

the milder forms of trifacial neuralgia, is to have the jaws thoroughly examined by the dentist in every case. Mr Salter has recorded some curious instances in which the nutrition of parts affected with reflex neuralgia from caries of the teeth, underwent perversion; in one, the iris of the affected eye, from being of a deep hazel colour, became of a dull grey.

The late Dr Anstie believed ('Lancet,' 1866) that in certain cases he traced neuralgia of the fifth nerve to the "reflex" disturbance caused by injuries to distant nerves; once to a knife-wound, by which the occipital nerve was divided, and twice to sickle-wounds of the wrist involving the ulnar nerve. In each instance the pain first came on when the injury was already repaired, and when the cicatrix had become firm.

It must also be borne in mind that pains undistinguishable from those of neuralgia often constitute the earliest symptom of various diseases and tumours of the maxillary and other facial bones, and that such cases may come under the observation of the physician at a time when there is no obvious deformity or swelling to lead the patient to seek surgical advice. There is, in the museum of Guy's Hospital, a specimen of Bright's, in which a morbid growth made its way from the sphenoidal sinuses into the middle fossa of the base of the skull, and doubtless pressed upon the fifth nerve; the patient, a woman of forty, whose countenance showed strong indications of suffering, had complained of an extremely severe pain on the corresponding side of the face, which became more violent in paroxysms.

Neuralgia affecting the neck, arm, and trunk.—Cervico-occipital neuralgia needs but brief consideration. Among the ascending branches of the cervical plexus, the great occipital, or posterior division of the second spinal nerve, is the one to which neuralgic pain is most often referred. Such cases are often due to the direct action of cold. Anstie speaks of a patient who was several times attacked after sitting in a draught which blew strongly on the back of the neck. One must be careful not to overlook disease of the cervical vertebræ.

Cervico-brachial neuralgia generally affects several of the branches of the brachial plexus. Numerous "tender points" are developed, most of them at spots where the nerves pierce the fascia, but some (as, for instance, the inferior angle of the scapula) where their occurrence is not so readily explicable. Glossy skin and causalgia are sometimes present, particularly in cases of traumatic origin. One special feature of this affection is its liability to be aggravated by muscular movements of the arm. Anstie alludes to a case in which, at a time when convalescence seemed to be almost established, the act of playing on the piano for half an hour at once brought the pains back. Salter ('Guy's Hosp. Rep.,' 1867) has shown that neuralgia affecting the nerves of the upper limb is sometimes due to diseases of the teeth. He says that pains in the shoulder and acromion, over the insertion of the deltoid, or at the bend of the elbow, not infrequently depend on this cause; and he cites the case of a lady who, whenever any of the teeth in the left side of the lower jaw became tender from caries, was immediately attacked with severe neuralgia at a circumscribed spot in the forearm.

Among the rarer causes of pains in the arms that might be mistaken for neuralgia may be mentioned disease of the articular processes of the cervical vertebræ. In 1876 a woman was admitted into Guy's Hospital under Dr Wilks, who had for four months been suffering from pains in the right shoulder, round the side, and down the arm to the tips of the fingers. No

cause for her complaints could be discovered, but after a few weeks she was attacked with pneumonia, and died of that disease. Dr Goodhart made a *post-mortem* examination, and found that there was a quantity of inflammatory exudation outside the notch between the seventh cervical and the first dorsal vertebræ. This had its origin in disease of the joint between these two vertebræ; the lowest root of the brachial plexus was involved.

Eruptions of zona and other cutaneous lesions are sometimes observed, but, as with supra-orbital neuralgia, it is often difficult to determine which is the cause and which the effect.

The neuralgiæ of the *dorsal* and *lumbar nerves* need not be described in detail. They are less frequent than those of the face or limbs, and agree closely with cervical neuralgia. The affection described by Sir Astley Cooper as irritable testis is probably neuralgia of the spermatic plexus.

Sciatica.—One of the most important of all the varieties of neuralgia is that which attacks the great sciatic branch of the sacral plexus, and has long been known by the special name of Sciatica (*passio ischiadica*). The part of the nerve which most commonly suffers is that which lies behind the upper half of the femur, but sometimes it includes the whole length of the main branches, and occasionally may be limited to the back of the knee, or to the terminal twigs in the calf or the foot. Its characters differ widely from those of tic douloureux. Instead of paroxysms of acute pain, darting like lightning through the nerve, and separated by intervals of comparative freedom, there is a constant, heavy, gnawing sensation; its intensity is not uniform, but the variations are comparatively slight and uncertain; and if it runs along the affected nerve at all, it travels slowly. It often appears to be worse at night, but this is ascribed by the late Dr Henry Lawson (who was himself a sufferer from sciatica) to the fact that the patient then has less to distract his thoughts than in the daytime. Few diseases induce more peevishness, restlessness, and misery, spoiling the appetite, perverting sleep, and interfering with every kind of work, as well as with most amusements and all athletic sports.

In advanced and severe cases the pain sometimes radiates into the sciatic nerve of the opposite side, or to branches of the lumbar plexus, or to nerves which are still more remote.

The pain is often greatly increased by pressure on the sacral plexus within the pelvis, or at its exit from the sciatic notch, and thus defæcation may be attended with much suffering. The patient is afraid to sneeze or cough, and is very susceptible to a draught of cold air.

When sciatica has lasted for any length of time several "tender points" are generally to be found; one is said to be situated where the nerve emerges from the pelvis, others where its branches pierce the fascia lata, and others over the sacrum, the crista ilii, &c. The whole length of the nerve also becomes unduly sensitive to pressure, and especially that part of the external popliteal nerve which lies under cover of the biceps tendon.

Wasting of the muscles is another symptom of all cases of sciatica which have lasted long. The buttock becomes flattened and flabby, the muscles of the thigh and calf are reduced in size, and feel soft and flaccid. In one case Anstie found the faradic excitability of the gastrocnemius much diminished; so that perhaps the loss of power does not merely depend upon disuse of the muscles. Even within the first few days the patient often

begins to limp in walking, so that he puts only his toes to the ground, and is glad to make use of a stick. Afterwards he lies on the sofa all day long, with every joint of the limb bent, dreading to make the slightest movement. At last the hip- and knee-joints may become rigidly flexed, so that any attempt to straighten them causes severe pain. Painful cramps in the flexor muscles of the toes may come on, especially when the patient is just falling off to sleep. Sometimes fibrillary tremors are observed, or even shaking movements of the limb.

A certain degree of anæsthesia or impairment of tactile sensibility appears to be of constant occurrence in the more severe cases of sciatica, and subjective sensations of numbness and tingling are often present. The affected limb is sometimes paler, and commonly colder than the opposite one; the patient occasionally complains of its feeling chilly, and it is said that the difference of temperature may be detected by surface thermometers.

It will be seen from this account that sciatica differs in the character of the pain and in other clinical features from those given on p. 384, which particularly apply to tic douloureux. Hence there is some doubt as to its being a true neuralgia, and some writers have regarded it not as a single malady, but as a group of affections due in different cases to widely different causes. Thus Sir Thomas Watson spoke of it as being sometimes inflammatory, sometimes "rheumatic," sometimes the result of irritation within the pelvis, or connected with a disordered state of the kidney, and, lastly, as sometimes purely neuralgic.

To some extent this is a question of terms: if we mean by Sciatica to denote any painful affection in the course of the sciatic nerve, we must admit that its pathology is very varied. But if we confine the term to idiopathic neuralgia, it is most important that we should not confound the true disease with the effects of pressure upon the sacral plexus and sciatic nerve from organic disease of whatever kind. In the 'Guy's Hospital Reports' (vol. x, 1864) a case was published that was regarded as an example of sciatica until the patient unexpectedly died; when a large aneurysm was found upon an abnormal artery, which passed down through the sciatic notch and along the back of the thigh. That case is, probably, unique, such a course of the vessel (which is normal in birds) being one of the rarest of abnormalities in the human subject; but a precisely similar pain might be caused by an aneurysm upon one of the regular arteries, or by a bony or sarcomatous outgrowth from the sacrum or os innominatum, pressing upon the nerve. After all, such affections are extremely rare.

More common are supposed to be cases of sciatica due to pressure of impacted scybala upon the nerve of the left side; and the result of emptying the sigmoid flexure is said to prove the fact. But in such cases, as in those which occasionally are met with from pressure during labour or from retroverted uterus or ovarian tumours which have fallen into the pelvis, the pain is accompanied with more decided numbness and paralytic symptoms than attend ordinary sciatica.

As Anstie maintained, the relation of sciatica to rheumatism is altogether imaginary, if any definite meaning be attached to the word "rheumatism;" but there is a kind of sciatica which accompanies lumbago, and probably owns the same cause. Moreover, apart from myalgia or "muscular rheumatism," there is no doubt that occasionally sciatica is due

to gout—probably to local gouty neuritis. We had a marked case of this kind in Philip Ward in February, 1887; a man of thirty-seven, who had suffered repeatedly from hereditary gouty arthritis in the foot, was the subject of severe sciatica of the left side. He had been treated in various other ways without any benefit, and was so quickly and effectually relieved after taking colchicum that one could scarcely doubt the justice of the diagnosis.

Again, some cases of sciatica occurring in syphilitic patients and becoming worse at night, are speedily relieved by iodide of potassium. Here, as in cases due to gout, the pain probably depends upon neuritis of the sciatic nerve; and if so, these and similar cases should be referred to Peripheral Neuritis rather than to idiopathic functional neuralgia.

Nevertheless, Dr Fagge believed that the majority of cases of sciatica are truly neuralgia, and that the points which distinguish it from other forms of neuralgia are not really essential, but in great part attributable to the large size and the superficial course of the affected nerve, and to the way in which it is exposed to irritation and to pressure. The first case of sciatica that he saw in private practice occurred in a gentleman who afterwards came to him for neuralgia of the brachial plexus; and Anstie records three or four cases of a tendency to neurotic diseases in relatives, or of other forms of neuralgia in the patients themselves.

Other writers, however, deny that inheritance or predisposition plays any important part in the ætiology of sciatica, and there is no doubt that it is far more commonly traceable than are other neuralgic affections to accidental circumstances, especially to the action of cold. Lawson states that nine tenths of all cases of sciatica are referable to this cause, and Erb affirms almost as much; exposure to a draught of air when the body is heated, wetting of the clothes, sleeping on damp ground or against a damp wall, have so frequently been followed by sciatica that no doubt can be entertained of their power to excite the disease.

Another frequent antecedent appears to be over-exertion of the lower limbs. In Lawson's own case the complaint came on after a long walk; and sciatica is said to be common in those who work at a sewing-machine.

The fact that certain classes of persons are exposed more than others to the exciting causes of sciatica probably explains the fact that it is more common in *men* than in women (according to Erb, in the proportion of four to one), and that adults from twenty to forty years old are most subject to it. Lawson saw one well-marked case in a boy aged fourteen, who was addicted to masturbation.

The *diagnosis* of sciatica is seldom difficult to the practitioner who first searches for the numerous other diseases that may cause pain in the thigh and leg. Where the calf is the part mainly affected one must think of thrombosis of the femoral vein, but this can be excluded by digital examination of the vessel at the groin, even if the complaint should be too recent to have caused œdema of the ankle. Disease of the hip-joint can be put out of consideration by the fact that neither pressure on the trochanter, nor forcing the head of the femur against the acetabulum, gives rise to pain; and disease of the sacro-iliac synchondrosis by the fact that no tenderness is elicited by tapping the articulation, or by pressure of the two ilia together. Advanced cases, in which the patient limps in walking and has wasting of the muscles, are very likely to be mistaken for spinal disease; this occurred some years ago in the case of a medical man whose fellow-

practitioners in his own neighbourhood all felt sure that there was something the matter with his vertebral column. Such disease is, indeed, very unlikely to have its effects confined for a length of time to one limb; but, on the other hand, when sciatica is at its height, the pain often radiates to the sciatic nerve of the opposite side. Even if one is satisfied as to the seat of the pain, one must still search carefully for local causes of irritation. In one instructive case a surgeon, feeling carefully along the course of the nerve, was fortunate enough to detect the presence of a bit of broken needle, the removal of which led at once to the cure of the patient.

Prognosis and treatment.—The duration of neuralgia in general and its amenability to treatment vary greatly in different forms of the disease, as well as in different cases of the same kind. The most protracted and obstinate form of all is Tic douloureux; most writers speak of it as incurable. Even this, however, is a very long time in wearing out the patient's life. Trousseau mentions a case in which it went on for thirty years. In the other varieties of neuralgia it is altogether exceptional for the disease to last more than a few months; but Anstie alludes to some instances of sciatica as defying all medical skill. In forming a prognosis one is to a great extent guided by the age of the sufferer; the older he is the more likely is the course of the disease to be prolonged. Recovery is generally gradual, and it is apt to be interrupted by relapses. Even after the pain is gone the affected part sometimes feels stiff for a long time, and its muscles are quickly fatigued by exertion.

The *treatment* of neuralgia is a matter which often taxes to the utmost one's patience and skill. Hygienic conditions must be carefully attended to; fresh air, regular bodily exercise, freedom from excitement, plenty of sleep, an abundant supply of wholesome nutriment, are each essential. Anstie laid special stress on the importance of a liberal diet, and particularly on the value of fat as an article of food. Whenever he could he made his patients take cod-liver oil; if the stomach revolted at that, he insisted on their eating butter, Devonshire cream, or sometimes olive oil, or even cocoa-nut oil. He strongly objected to allowing sufferers from neuralgia to have wine or brandy, excepting in very moderate quantities and with the meals, and all experienced physicians concur in his repeated protests against prescribing stimulants for the mere relief of pain.

Quinine in large doses is often of striking benefit, especially in trifacial neuralgia, and in cases which show periodic exacerbation.

When neuralgia particularly affects the lower jaw, and whenever it is connected with dental iritation, *butyl-chloraldehyde hydrate* ("croton-chloral") has sometimes striking curative effect, and is always worthy of trial. It often unmistakably succeeds, and often as unmistakably fails.

Fothergill gave *conium* in large doses, and not without success.

Ammonium chloride in full doses is believed, on excellent authority, to be an efficient remedy in certain cases.

Of all drugs, however, *arsenic* appears to hold the first place. Dr Fagge preferred the liquor sodæ arseniatis, because he had several times found that patients could take it without inconvenience who were attacked with diarrhœa and vomiting if they were placed on Fowler's solution. The liquor arsenici hydrochloricus may be used in combination with the tinctura ferri perchloridi, itself a remedy to which Anstie attached a special value.

Dr Ashburton Thompson has advocated the treatment of neuralgia by *phosphorus*. The formula to which he gives the preference is a solution of the drug in cod-liver oil; of this a fluid drachm, containing one twelfth of a grain, is administered every four hours. Phosphorus may be made into pills, and the Pharmacopœia contains directions for their manufacture; but it has been shown by Dr Rees that phosphorus pills are apt to pass through the intestinal canal without being dissolved. Dr Thompson gives a table of fifty cases of neuralgia treated by phosphorus, in several of which the effects of the remedy seem to have been remarkable; but he says that if decided results are not attained within three days it is useless to persevere.

Of late years *Antipyrin* has been much used for neuralgia, particularly facial neuralgia, and in many cases its effect is decided and complete. It seems to be of little use in sciatica, as perhaps might have been anticipated.

Apart from the pressure of gummata or other tumours on the nerve in the pelvis, it is probable that syphilitic neuritis may cause sciatica, and that full doses of potassic iodide and local mercurial inunction will effect a cure in such cases: similar treatment of gouty neuritis by colchicum has been also already referred to.

Galvanism is often very valuable in the treatment of neuralgia. One of the poles of a constant-current battery may be placed close to the spine of the affected side, or near the roots of the painful nerves, the other being applied upon the various "tender points" in succession, or being perhaps immersed in a vessel of water into which the hand is also dropped. A "descending" current is usually employed, the positive pole being put nearer to the centres than the negative one, but reversal of the poles seems to make no difference in the result. A good example of the effect of this form of electricity is afforded by a case related by Anstie in the fourth volume of the Clinical Society's 'Transactions.' A woman had for nearly five months suffered from cervico-brachial neuralgia, which lasted for several hours each day, and had defied all kinds of medicinal treatment. The very first application of a battery of from ten to fifteen cells arrested the pain; the attack which followed was much less severe than usual; and after thirteen days the complaint ceased to recur. The treatment was continued altogether for six weeks. Another case in which great relief was afforded was read to the Clinical Society at the same time by Dr Buzzard.

Faradisation of the nerve is rather injurious than beneficial, but the application of a powerful current to the skin by means of the electric brush is said to be sometimes useful as a mode of severe *counter-irritation*. Its action is similar to that of *blisters*, which are undoubtedly of great value in many cases. Anstie recommended that they should be placed not upon the painful part itself, but close to the spine at the level of the affected nerves. Even in the desperate tic douloureux of old age he sometimes found that a blister applied to the nape of the neck removed the pain entirely for a long time. But in sciatica he obtained better results by placing flying blisters over the nerve-trunk in the gluteal region. Mustard plasters, chloroform liniment and other rubefacient applications are only serviceable in mild cases.

Acupuncture over the sciatic nerve, and *deep injections* of morphia in that region, are powerful and frequently successful measures. Formerly it was the practice to divide the infra-orbital nerve in severe cases of neuralgia, and when the relief was found to be only temporary, to excise a piece: the writer saw this done by Nélaton in 1863. But even then the proximal part of nerve

grows out and reunites, so that the operation has fallen into disuse.* A more recently introduced practice is *stretching* the affected nerve, either by cutting down upon it and forcibly pulling the trnnk out of its bed, or by forcible flexion of the thigh upon the pelvis with the knee extended: both methods are reported to have been successful in a few cases, but neither is without risk of injury.

When all attempts at treatment by local measures or by drugs specially indicated have failed, we must fall back on the remedy which would naturally first suggest itself for so painful a disease as neuralgia—*opium*. One should be very cautious in administering it when there is any hope of the ultimate subsidence of the complaint; but in cases of incurable tic douloureux Trousseau employed opium with no little success, increasing the dose boldly until sometimes as much as a quarter of an ounce or even half an ounce of laudanum was taken daily. In a few instances the result was more than a palliation of the patient's misery. The frequency and severity of the attacks of pain were lessened, so that after a time the drug could be discontinued; and although a relapse occurred a few weeks later, the interval of ease was a great gain; sleep had been obtained and food had been taken, which had before been almost impossible, so great was the suffering caused by mastication.

In many cases the administration of opium by the mouth disorders the digestive organs. Hence the subcutaneous injection of morphia is preferred, for it is attended with no such ill effects; and of late years this method of treating neuralgia has been extensively employed. The quantity should be very carefully regulated; not more than a fifth of a grain (two minims of the Pharmacopœial solution) should be used on the first occasion; in mild cases it is even well to begin with one tenth of a grain. If relief is not afforded by such doses, larger ones may be tried; a quarter of a grain, half a grain, even a grain, is commonly injected under these circumstances; and sometimes much greater quantities still. One seldom has to use the syringe oftener than twice in the twenty-four hours; but Anstie laid stress on the importance of performing the operation as early as possible after the commencement of an exacerbation of the pain. It seems to be almost a matter of indifference whether the morphia is injected at the seat of pain or at any other part of the body, except so far as concerns the effect on the patient's imagination.

The effect of subcutaneous injection of morphia is often marvellous. Suffering which had been almost intolerable is removed, as by magic, within a few minutes. And this is not infrequently the first step towards the complete cure of the disease. It was so, for instance, in the severe sciatica of which Dr Lawson was himself the victim, and in which other plans of treatment had been tried without the least good result.

This method is not, however, free from evils of its own. Dr Oliver has related in the 'Practitioner' a case in which immediately upon the injection the patient cried out in great alarm, the pulse became very small, the face red, and the eyeballs prominent. Brandy was given, and in about half an hour these symptoms passed off. It is thought that the alkaloid may have been thrown directly into a small vein. But such an accident is excessively rare.

A much more serious risk is that of setting up a "morphia habit,"

* See Mr Bowlby's Astley Cooper prize essay on "Diseases and Injuries of Nerves, and their Surgical Treatment" (1886).

a peculiar state of depression and irritability, which can be relieved by nothing but a fresh dose. There is also reason to fear that the remedy itself sometimes renders the pain more intense than before, or at least diminishes the intervals between the exacerbations. Even when the original disease is cured patients sometimes experience great difficulty in discontinuing injections of morphia to which they have been accustomed; a vigorous effort may be needed, and the endurance of much discomfort and even misery for several days. For this reason, as well as for others, the syringe should not be entrusted to the hands of the sufferer himself, nor even to those of a relative or servant, unless perhaps in incurable cases. Anstie, however, laid stress on the fact that the evils connected with the establishment of a morphia habit never arise when the dose is kept below a certain point. In a valuable paper in the 'Practitioner' he insisted stongly on the importance of "economy" in the use of the alkaloid; and he related the case of a lady, who for three years had enjoyed a complete immunity from severe sciatica under the daily use of a dose of morphia, which was at first one twelfth of a grain, and was never raised above one fourth.

PERIPHERAL PARALYSIS.—Many points in regard to the causes and symptoms of paralysis from lesions of the nerve-trunks are the same, whatever parts of the body are their seat. It is convenient to take the nerves of the arm by way of illustration, because the movements performed by its several muscles are more distinct and definite than those of the individual muscles of the leg, and their functional integrity is accordingly more easy of determination.

Injuries of the *brachial plexus* may be followed by loss of power and of sensation in the whole upper limb. The surgeon sees cases in which this occurs as the result of severe accidents in which the shoulder-joint is dislocated, or the humerus or scapula fractured. But similar symptoms not infrequently follow pressure upon the nerves, or an injury so slight that its occurrence, especially in a child, may be altogether overlooked. Thus Paget ('Med. Times and Gaz.,' 1864) mentions the case of a boy whose left arm, while he was still an infant, was violently pulled by a little brother; the limb appeared altogether powerless for long time afterwards, and it remained permanently weak and much smaller than the other one. The employment of force to reduce a dislocated shoulder has sometimes been attended with like consequences; so also the pressure of a crutch, tying the arms of a prisoner with a cord, and carrying certain large vessels with handles through which the arm is passed, as among the water-carriers of Rennes. "Crutch paralysis" is not uncommonly seen in hospital practice in London; and the patient is often unaware of its cause. A similar effect may also be produced by lying on one side, with the weight of the body resting upon the arm; or, more often, by sleeping with the arm across the back of a chair. But in most instances of this kind the sensibility of the patient has been blunted by drink, and hence they used to be known in the hospital as "Sunday morning" paralysis. When the cause is doubtful, the axilla and neck must be carefully examined, lest an aneurysm, exostosis, or other tumour should be overlooked. Some writers believe that the brachial plexus is sometimes affected by external cold, as we shall presently see to be the case with the facial nerve; but this is doubtful. Mr Salter has related ('Guy's Hosp. Rep.,' 1868) two cases, in each of which caries of a wisdom-tooth, beside causing severe pain in the arm, led also to partial "reflex" paralysis of the

muscles, so that the patients could not grasp objects nor raise the hand to the head, nor hold a fork at dinner. In both cases all the symptoms vanished as soon as the tooth had been extracted or a few hours later.

It is exceptional for the whole of the brachial plexus to be paralysed at once by any of the above causes; and the loss of power may often be limited to the muscles supplied by a single nerve. In such cases the interpretation of the symptoms may be so obvious that no anatomist could possibly misunderstand them. But sometimes they require careful study.

Special motor paralyses.—Particular interest attaches to paralysis of the serratus magnus muscle. The *posterior or long thoracic nerve* which supplies it leaves the plexus at so high a point that it is never involved when the cause is the pressure of a crutch, or sleeping upon one arm. But it may be affected by a blow upon the shoulder; by carrying a heavy weight upon the shoulder; or by over-exertion of the shoulder-muscles, as in the case of mowers, puddlers, cobblers, or ropemakers. Sleeping on damp ground and exposure to draughts are also said to have given rise to it.

The principal symptom is an alteration in the position of the scapula. Even when the shoulder is at rest, with the arm hanging down, the scapula, as a whole, is drawn slightly upwards and inwards, and has its inferior angle tilted, so as to be nearer to the vertebral column than the rest of its posterior border. But what is more characteristic is that this posterior border stands off a little from the part of the back on which it should rest, forming a "wing-like" (pterygoid) projection. These peculiarities are greatly exaggerated when the patient is told to perform certain movements. Thus if he is directed to lift his arm straight over his head, he finds himself unable to raise it above the level of the shoulder, because the necessary rotation of the scapula by the serratus cannot be effected; but when once another person has moved the bone to the proper position for him and fixed it there, he acquires the power of lifting the limb to whatever height may be desired. During the attempt to raise the arm, the scapula, if left to itself, moves further than ever towards the spine; and where both serrati happen to be paralysed together the two bones may actually come into contact at their inferior angles. If the raised arm is brought forwards the "wing-like projection" becomes greatly augmented, the posterior border of the scapula standing off so as to leave a deep hollow. Another symptom, according to Dr Poore ('Clin. Soc. Trans.,' viii), is an alteration in the shape of the chest on the affected side, as shown by the cyrtometer. He finds that in a healthy person, when the arms are thrown horizontally forwards, the chest becomes flattened in front, and its transverse diameter is increased. Where there is paralysis of one serratus no widening occurs on that side.

An affection of more frequent occurrence is paralysis of the muscles supplied by the *circumflex* nerve, among which the *deltoid* is the chief. This, too, may result from direct injury to the shoulder. According to Erb it is sometimes caused by "rheumatic" affections of the articulation. One must, however, bear in mind that osteo-arthritis (to which disease Erb probably refers) is commonly attended with destruction of the long tendon of the biceps, and that unless this structure is perfect the arm cannot be raised; while inability to lift the arm from the side is also the principal symptom of paralysis of the deltoid. It may be thought that a sufficient proof of paralysis would be found in atrophy of the muscle, which, indeed, is often so marked that a deep groove is formed between the acromion and the head

of the humerus. But a considerable degree of wasting may be the result of mere disuse when the joint is diseased.

Paralysis of the *musculo-cutaneous* nerve is indicated by inability to flex the elbow-joint, the biceps and brachialis anticus muscles being paralysed. It must be borne in mind that the supinator longus (which receives its supply from another source) is also a flexor, and the patient must therefore be told to keep his hand supinated while the power of the other muscles is being tested.

Among the nerves which send filaments to the hand and fingers the *musculo-spiral* is much more apt than the other two to be separately affected. In a considerable proportion of the cases of paralysis due to the pressure of a crutch, or to lying on one side during the deep sleep of intoxication, the musculo-spinal nerve suffers alone; and as in the latter case the triceps often escapes, it is probable that the nerve undergoes compression where it is winding round the humerus, and after it has given off the branches to that muscle. The extensors of the wrist, of the two joints of the thumb, and of the metacarpo-phalangeal joints of the fingers are the chief other muscles supplied by this nerve; and accordingly one symptom of paralysis of it is a flexed condition of these joints, with inability to straighten them; the two remaining joints of the fingers being nevertheless capable of extension by the interossei and lumbricales, provided that the metacarpo-phalangeal joints are held up by another person. Another consequence of the loss of power in the extensor muscles of the wrist is impairment in the extent to which the fingers can be *flexed* upon the palm. The paralysis of no other nerve interferes so much with the motions of the hand as that of the musculo-spiral.

Isolated paralysis of the *median* (or of the ulnar) nerve is more commonly due to a wound or injury at the elbow, in the forearm, or at the wrist, than to any affection of the brachial plexus above. The symptoms, in the case of the *median* nerve, are inability to use the flexors of the wrist and fingers, the metacarpo-phalangeal joints being alone capable of flexion by means of the interossei. Indeed, the excessive action of these muscles is apt to cause an over-extension of the two phalangeal joints of the fingers, and especially of the index finger.

On the other hand, when the *ulnar* nerve is paralysed the muscles of the little finger and the interossei are more markedly affected than any others. The fingers cannot be abducted nor adducted, and the patient is unable to flex the metacarpo-phalangeal, or to extend any of the phalangeal joints of the fingers. There being nothing to antagonise the traction excited by the common extensor and flexor muscles, the joints in question become distorted, so that the last two phalanges are over-flexed, while the first phalanges, on the other hand, are over-extended. Accordingly, the prominences of the knuckles are replaced by hollows on the back of the hand, while they project towards the palm, and are the more noticeable there because the interossei and two of the lumbricales muscles are atrophied. The fingers of a hand so affected are often compared to the claws of a griffin—*le main en griffe, Klauenhand.*

The production of deformities, in affections of the nerve-trunks, by contraction of the muscles which are physiologically the antagonists of those that are paralysed, has not hitherto been mentioned; but it may occur in every form of these affections, and often complicates the symptoms considerably. On the other hand, at advanced periods of cases, the paralysed

muscles may waste and shorten, so as to produce distortions of precisely opposite characters.

The anatomical distribution of the nerves is not strictly adhered to in all cases of peripheral paralysis. Erb speaks of having seen several instances in which the deltoid, biceps, brachialis anticus, and supinator longus were chiefly, if not exclusively, attacked; and he throws out the suggestion that this depends upon an affection of the sixth cervical nerve where it is emerging from between the scaleni. In such patients the arm hangs by the side, and the elbow cannot be flexed. It is remarkable that a precisely similar affection was described by Duchenne in newly born children, as the result of traction or pressure upon the shoulder during birth. Erb has himself seen two cases of this kind.

Lastly, one must bear in mind that in exceptional cases a morbid process beginning in a single nerve at a particular spot is very apt to pass upwards along that nerve, and may afterwards extend to other nerves with which it is connected. Thus Dr Weir Mitchell relates the case of a man who received an injury to the nerve which supplies the pectoralis major; on the third day he was attacked with pain in the course of the median and musculo-cutaneous nerves, and the muscles to which they are distributed subsequently became paralysed and wasted. Another patient bruised his ulnar nerve at the bend of the elbow, and about five months afterwards the flexor muscles of the wrist became affected with paralysis, those supplied by the median nerve, as well as those supplied by the ulnar. These, however, were probably examples of peripheral neuritis.

The reaction of degeneration.—Cases of local paralysis due to lesions of the nerve-trunks are all examples of "peripheral paralysis," and the electrical reactions of the affected nerves and muscles present characteristic modifications. These were first minutely studied by Erb and Ziemssen.

Let us suppose that a nerve-trunk is cut across. The result is that throughout the whole length of the nerve and its branches below the affected spot (so far as one can test them without at the same time stimulating the muscles) its excitability undergoes a progressive diminution, both to the make and break of galvanic currents, and to the rapidly alternating faradic currents from an induction coil. In the course of the second week it is entirely extinguished, so that not the slightest contraction can be produced by the application of either form of electricity to the nerves. But the reactions of the paralysed muscles are very different. They, too, fail to respond to a faradic current; but when a galvanic current is employed they contract much more readily than under normal circumstances to the stimulus of opening or closing. The method of determining this fact is first to ascertain what number of cells of the battery are required to excite contractions in the corresponding muscles of the healthy limb; we then perhaps find that only half that number is needed to excite contractions in the muscles supplied by the injured nerve. Indeed, they can often be thrown into vigorous action by a current which is too feeble to be felt.

Moreover, there is what is termed a "qualitative" change, *i. e.* the order in which the two poles excite contractions is altered. According to Pflüger's law, stimulation depends upon rise of catelectrotonus or fall of anelectrotonus (*i. e.* in either case the passage of a segment of nerve from a lower to a higher degree of excitability); the descending current has

greater exciting power than the ascending, and closing or making than opening or breaking the galvanic current. Accordingly, the order in which currents of successively increased strength produce contraction in the muscle supplied by the motor nerve stimulated is as follows:*

	CC	AO	AC	CO
Weak current causes . .	c			
Strong current causes . .	C	c	c	
Very strong current causes	C′ or T	C′	C	c

The rule may be shortly remembered thus:—With increasing strength of galvanic current the order in which contractions appear is C.C.C., A.O.C., A.C.C., C.O.C.†

When, however, a muscle is paralysed by severance of its motor nerve in any part of its course, or by destruction of its anterior root, or of its corresponding large ganglion-cells in the anterior cornu, and when this has lasted for a week, not only is the muscle less excitable when the "motor point" of its nerve (*i. e.* the place where it becomes superficial and is most favourably stimulated) is excited by the induced (faradic) current, not only is it *more* excitable when the same point of its nerve is stimulated by the make or break of the direct (galvanic) battery current, but the above physiological order is altered.

So far from contraction at the moment of closure with the cathode (C.C.C.) being now the most marked effect, there is often as much or even greater contraction when the circuit is completed with the anode; while instead of cathodal opening contractions (C.O.C.) being the last of all to appear, they may precede the anodal opening contractions (A.O.C.). Accordingly the normal order for contractions with currents of increasing strength given in the above table is altered, and instead of C.C.C., A.O.C., A.C.C., C.O.C., we have A.C.C. before C.C.C., and C.O.C. before A.O.C.; *i. e.* the anodal closing contraction is for equal currents stronger than the cathodal, and the cathodal opening contraction is for equal currents stronger than the anodal.

This is called the Reaction of Degeneration.

* Here C.C.c. means that a small Cathodal Closing contraction is produced by a weak current. The current is descending, and the stimulus is the rise (in this case from zero) of catelectrotonus on closing (making) the galvanic circuit. A.O.C. means that an ascending galvanic current produces an Anodal Opening (breaking) contraction, the stimulus being the fall of anelectrotonus (to zero). A.C.c. or A.C.C. means that making contact again with the ascending current will, if it is as strong or stronger, produce a slight or a marked Anodal Closing contraction. Lastly, C.O.c. means that breaking contact with the descending current will, if it is strong enough, act as a slight stimulus and produce a Cathodal Opening small contraction.

† The German symbols used by Erb are Ka. for Cathodal, An. for Anodal, S. (Schleissung) for closure, O. (Oeffnung) for opening, and Z. (Zuckung) for contraction. These are wholly or partly adopted by some English writers. If it were important to avoid using the same letter for different words, it might be done by spelling Cathode with a K, and using Break and Make instead of close and open. The normal sequence would then stand: K.M.C., A.B.C., A.M.C., K.B.C. But in such matters whatever is most generally used and best understood is best.

It may be added that c. means a weak contraction, C′. a strong one, and T. a tetanic contraction, that > and < mean respectively *greater than* and *less than*, and that R.D. means the Reaction of Degeneration.

Lastly, the contractions themselves are slow and protracted, and resemble tonic spasms rather than the short clonic movements which occur in healthy muscles when stimulated by galvanic currents.

Little progress has hitherto been made towards the explanation of these remarkable facts. The seeming paradox that muscles insensible to powerful faradic currents should nevertheless respond to very weak galvanic currents has, indeed, been accounted for by Neumann, who has shown that it depends upon their requiring a current to pass for some length of time before they can react to it. Momentary galvanic currents, even when of considerable strength, are as inoperative as faradic currents themselves. But why the muscles should be incapable of responding to currents of brief duration, and why they should be sensitive to weaker galvanic currents than under normal conditions, we cannot at present say.

This, however, is established; that the peculiar reaction in question, as well as the loss of electrical irritability in the trunk of the nerve below the seat of injury, is coincident with and indicative of a remarkable series of degenerative changes which affect the structure of the paralysed nerve and muscle. Erb accordingly proposed the term "reaction of degeneration" to denote the presence of increased galvanic excitability in a muscle, when associated with diminished faradic excitability. Within a few days after the occurrence of any severe lesion of a nerve-trunk, the whole length of the nerve below is found to be greatly altered: the medullary sheaths of the fibres break up into fatty granules, and it is doubtful whether the axis-cylinders remains undestroyed. At the same time the neurilemma everywhere undergoes a greatly increased development; first it becomes crowded with corpuscles, and these presently develop into spindle-cells and fibres, so that the nerve-trunk itself becomes much denser and harder than before. In the muscles, too, similar changes take place. The fibres diminish in size, and their striation becomes indistinct; but their nuclei multiply, and the perimysium is crowded with cells, which after a time undergo development into fresh fibrous tissue. If the injury to the nerve should remain unrepaired the muscular fibres waste still further, and at length the whole substance of the muscles is converted into a whitish yellow, flattened mass of dense fibrous tissue, in which no striated fibres can be discovered. It often includes in its interstices a large number of adipose cells.

The cause of these curious changes is at present unknown. The easiest way of explaining them would be to attribute them to an extension of an inflammatory process along the nerve from the seat of injury. But, as Erb remarks, they ought not then to be confined to that part of the trunk which is to the peripheral side of the lesion, nor should they be traceable through a plexus or be accurately limited to certain fibres; nor should they occur simultaneously in distant muscles, or in those which are placed in the upper part of a limb. In some way they depend upon the separation of the affected structures from the nervous centres, in accordance with the law named after its discoverer, Dr Waller.*

* It is important to remember that this remarkable reaction of degeneration is not confined to the paralysis from injury to peripheral nerves now under consideration. We shall hereafter see that it accompanies paralysis dependent on lesions of the motor ganglion-cells of the anterior cornua of the cord and of their homologues in the bulb and mesencephalon. If, therefore, we speak of the R.D. as characteristic of "peripheral paralysis," we must extend the meaning of the latter phrase. The R.D. and the histological changes in the nerves which follow it are indicative of *severance of the nerve-fibres affected from their trophic centres, either by destruction of these ganglia, or by severance of the nerve in its course.*

It must not be supposed that the commencement of degenerative changes in the peripheral nerves and muscles is a proof that the injury of the trunk above is permanent and irremediable. On the contrary, nerves possess extraordinary powers of regeneration. Even after the muscles have for several weeks exhibited the reaction of degeneration it often happens that the connection between their nerves and the spinal cord is at length gradually restored. Erb has noticed in such cases very curious anomalies in the reactions of the nerves and muscles respectively to electrical and other stimuli. Nerves that have completely recovered the power of transmitting volitional impulses may still remain devoid of all excitability by faradic or galvanic currents; or if their regeneration should take place early, they may regain their sensitiveness to both kinds of current before the muscles have ceased to present the reaction of degeneration. That reaction, indeed, does not persist for any great length of time after the original injury. If the nerve fails to undergo repair, and the muscle-fibres begin to disappear, their excitability to galvanic currents quickly begins to diminish, and at length it becomes extinguished; and even when regeneration of the nerve does occur, the muscles commonly display a lessened rather than an increased galvanic irritability while it is going on, and for some time after it had been apparently completed.

On the other hand, it is only when the original lesion of the nerve-trunk is of a certain degree of severity that the reaction of degeneration presents itself. In the slighter forms of paralysis, such as that due to the pressure of a crutch, or to compression of the brachial plexus or musculo-spiral nerve during sleep, both the muscles and nerves often retain their normal irritability both to faradic and to galvanic currents.

Time of the reaction.—When a motor nerve is severed (to take the simplest form of peripheral paralysis), voluntary motion is, of course, at once and completely lost. For about the first week both muscle and nerve are less and less susceptible to all stimuli; but after a few days—while the galvanic and faradic excitability of the muscle, and the faradic excitability of the nerve continue to diminish, until after two or three weeks they are entirely lost—the galvanic excitability of the motor nerve rapidly increases with the other phenomena which accompany degeneration of the nerve-fibres and atrophy with degeneration of the muscle. In cases which recover, the faradic excitability of muscle and nerve returns as voluntary motive power is re-established, and the abnormal galvanic reactions of the nerve more gradually disappear. In cases which do not recover, the reaction of degeneration continues, though in slowly diminishing degree, for many weeks or months, until at last it also is lost, and the atrophied muscle responds to no kind of stimuli.

Anæsthesia.—Nerve-trunks possess sensory beside motor functions, and these too may be impaired by the same lesions which cause paralysis. Thus, if we still take the brachial plexus by way of illustration, we find that affections of its nerves may completely extinguish the transmission of tactile impressions from all parts of the limb to which those nerves are distributed, and may likewise render the patient unable to feel a prick or a cut, heat or cold, or the passage of a galvanic current. The resulting loss of common sensation is termed Anæsthesia, while the incapacity to feel pain is distinguished as Analgesia. They do not necessarily accompany one another. Mitchell relates two cases in which, without the slightest impairment of

the sense of touch, there was great diminution in the susceptibility to pain, one patient experiencing only a slight pricking sensation when a large needle was run into the palm, and the other being scarcely sensitive to the electric wire-brush. In ordinary cases, however, this instrument is a most delicate test for the presence of minute degrees of susceptibility to sensory impressions. The part to which it is applied must be perfectly dry, so as to oppose the current passing through to the underlying muscles and confine its effects to the skin. The numerous minute points of the dry brush help to the same result. Mitchell says that if no sensation can be produced by it one may generally conclude that the loss is absolute; but that on several occasions the use of it has saved him from despairing of cases which had seemed altogether hopeless.

On the other hand, there are all possible degrees of anæsthesia—to a point at which very careful investigation is necessary to show that the transmission of impression is not perfect. The best ways of detecting very slight anæsthesia are to bring the tip of a feather gently upon the surface of the skin, or to touch a single hair with a needle or the edge of a knife. If the patient can feel this on one side but not on the other, sensation is to some degree impaired on the latter.

A pair of compasses may be used after E. H. Weber's method, the object being to determine at what distance from one another the points are felt to be distinct. This "limit of separate perception" varies greatly in different parts of the skin under normal conditions, from 2·25 mm. on the extremities of the fingers to 77 mm. on the arm. But for each region there is an average, and any very considerable departure from it may be taken as proof that some perversion of sensibility exists. Or a direct comparison may be made between the opposite sides of the body in the individual whose case is being investigated. An improved form of the instrument is sometimes employed, in which the two limbs of the compasses slide upon a graduated bar; this is called the "æsthesiometer." Dr Weir Mitchell teaches that, to ensure the most correct results that are attainable, one must cover the patient's eyes, keep the part which is to be tested perfectly at rest, and apply the compass-points (which should be rounded) lightly upon the surface of the skin, but with equal force, at exactly the same time, and in a line bearing a definite relation to the axis of the limb. Experience has shown that it is useless to base precise numerical statements upon Weber's method. It often happens that widely different results are obtained from the same spot at an interval of a few minutes. All we can obtain are relative results, and these are after all what we most want.

It would be a great mistake to suppose that in affections of a mixed nerve-trunk the loss of cutaneous sensation is necessarily equal to the muscular paralysis. Very often, particularly when the disease or injury is of but slight severity, there is little or no anæsthesia, although the loss of power is complete; or sensation may be quickly regained while the motor power is being slowly recovered. Probably the sensory centres are readily affected by few nerve-fibres and by very feeble impulses, whereas a muscle is unable to respond unless the stimulus is conveyed to it by many channels at once. As Mitchell remarks, this accords with the well-known fact that by irritation of the ulnar nerve at the elbow sensation is far more readily excited than motion.

Lesion of a mixed nerve-trunk never produces anæsthesia, unaccompanied by motor paralysis.

Beside degrees of anæsthesia there are certain perverted sensations, conveniently grouped together as *paræsthesiæ*—numbness, tingling, burning, &c. These, however, except numbness or "pins and needles," which is the most constant effect of moderate compression of a nerve, are less frequent and less characteristic in cases of injury or disease of nerve-trunks than in spinal and cerebral diseases, and will therefore best be treated hereafter.

Pain.—A third symptom of paralysis of the nerve-trunks is pain. It is by no means constantly present, and, indeed, is wanting in most cases of ordinary peripheral paralysis. It commonly exhibits in a more or less marked degree the features of neuralgia, and is referred mainly to those parts of the skin to which the diseased or injured nerve is distributed. It is sometimes due to peripheral neuritis; and the trunk of the nerve may then, in thin persons, be felt to be swollen and exceedingly tender. In some cases it is periodic, returning every day at about the same hour, generally towards the latter part of the day. There is a special variety of it, to which Dr Weir Mitchell has given the name of Causalgia (καῦσις, cautery), on account of its burning character. This may vary in severity, up to the most unendurable agony, which the patient compares to a "red-hot file rasping the skin." In cases of peripheral paralysis due to wounds, this kind of pain seldom, if ever, comes on until the process of cicatrisation is beginning. Its most frequent seat is—in the upper limb, the palm of the hand; in the lower limb, the instep. Heat aggravates it, and allowing the limb to hang down has the same effect. It is relieved by moistening the skin, and two of Mitchell's patients always carried a bottle of water and a sponge, and would not allow the affected part to become dry, even for an instant. Others found some ease in walking, by pouring water into their boots. In course of time the severe suffering caused by this condition often leads to a general irritability of the nervous system, allied to hysteria. The patient may then complain of hyperæsthesia of the whole body, particularly when the surface is dry.

This peculiar kind of pain seems never to occur in parts which have been rendered altogether anæsthetic by the complete division of their nerves; nor does it accompany the subjective sensations that are so often referred to the distal ends of limbs which have been amputated. From these facts Mitchell argues that its immediate cause is some change in the peripheral extremities of the nerves, rather than in the trunks above.

Trophic changes.—That causalgia is due to a peripheral lesion is the more likely because the integument of the parts in which the pain seems to have its seat is liable to a special change, to which Sir James Paget first gave a name—that of *Glossy Skin* ('Med. Times and Gaz.,' 1864). The surface shines, as though it had been varnished. In Paget's words, "the fingers"—for they are most often affected—"become tapering, smooth, hairless, almost void of wrinkles, glossy, pink, or ruddy, or blotched as if with permanent chilblains." An eruption of small, pointed vesicles (somewhat like those of eczema) is also of common occurrence; or bullæ may form, which afterwards ulcerate; or painless whitlows, leading to shedding of the nails. Or, again, the nails themselves may become arched and incurved, and the skin may shrink away from their roots, leaving the sensitive matrix partly exposed.

Parts which are the seat of causalgia are slightly *warmer* than those on

the opposite side of the body; whereas the general effect of injuries of the nerve-trunks is to lower the temperature; sometimes, as in some cases of Hutchinson's, by as many as eight or ten degrees Fahrenheit.

Zona, or shingles, is the name given to a remarkable affection of the skin analogous to these results of lesions of trophic nerves. It will be considered in the last section of this work, among Diseases of the Skin.

Another occasional result of morbid conditions of the nerves of a limb is a painful *swelling of its articulations* which may go on to anchylosis, and greatly increase the deformity and the disablement experienced by the patient. The arthritic effects of injuries to nerves have been well illustrated by Mitchell; they are analogous to the affections of joints which occur in certain diseases of the spinal cord, and will be afterwards described as the arthropathies of Charcot. They are in the highest degree rebellious to treatment.

Erb also mentions, as sometimes following paralysis of the musculo-spiral nerve, a thickening of the *tendons* at the back of the wrist—a circumscribed painless swelling, as large as a hazel-nut, evidently due to their having been exposed to mechanical strain by the flexed state of the joint.

These various conditions appear to be sure indications of the existence of some disease affecting the nerves of the parts concerned. But they do not necessarily depend upon the same lesion. Paget mentions two cases in which glossiness of the fingers developed itself as the result of neuralgia after shingles; Mitchell relates one in which it was due to a chronic affection of the spinal cord; and some years ago the late Dr Moxon had in Guy's Hospital a remarkable case in which a precisely similar state of the fingers was caused by the pressure of a mediastinal tumour.

Diagnosis.—The recognition of paralysis from lesions of the nerve-trunks is seldom difficult for a good anatomist. Sometimes, indeed, progressive muscular atrophy in an early stage might be mistaken for peripheral paralysis, particularly if the deltoid muscle should be alone affected, or the interossei and the muscles of the little finger. The application of galvanic and faradic currents to the paralysed muscles would clear up the doubt in some cases, but not in all.

Having ascertained that the paralysis is of peripheral origin, one must search for all possible causes of compression of the nerves. Some years ago a woman came to Guy's Hospital complaining of numbness and loss of power in the right arm, which symptoms (she said) had already lasted six months; she was found to have a cancerous growth in the breast, which had extended to the axilla, and involved the nerves of the brachial plexus. One must also bear in mind the possibility that the paralysis may be "reflex," due perhaps to irritation from diseased teeth.

In the lower limbs the mistake most likely to be made is that of attributing to an affection of the spinal cord a paralysis really due to a pelvic tumour compressing the sacral plexus, or to a mass involving branches of the lumbar nerves. In 1876 a woman died in Guy's Hospital who for ten years had suffered from paralysis of the sphincters of the bladder and rectum, and for seven years from loss of power and sensation in the legs below the knee-joints, so that she was unable to walk, and could only crawl about on her hands and knees. The cause was found to be a large abscess in the pelvis, through which the cords of the sacral plexus ran; it was dependent on disease of the bone.

The *prognosis* of the different paralytic affections that may result from lesions of the nerve-trunks of a limb depends upon their nature and severity. Most of the cases that come under the care of the physician get well sooner or later, and one can do much by treatment to hasten their recovery. Thus crutch-paralysis commonly passes off in a week or a fortnight; that which follows compression of the nerves during sleep more often lasts from four to six weeks, and it may be prolonged for several months; that which is caused by severe stretching or bruising of the nerves, generally lasts more than a year, and may never be entirely removed. Even after very long periods improvement sometimes takes place. Mitchell says that only the careful notes previously made could have convinced him of the extent to which function had been restored, in some of the worst cases, at the end of three or four years.

As may be inferred from what has been stated above, most valuable aid in the prognosis of this form of paralysis is afforded by an examination of the electrical reactions of the affected parts. In cases which are to terminate rapidly in recovery, the muscles and nerves retain their normal irritability; in those which are to run a protracted course, the muscles present the reaction of degeneration; while in cases which are incurable, susceptibility to every form of galvanic stimulus is extinguished.

Treatment.—Electricity is useful, not only as a guide to prognosis, but also in treatment. There is, indeed, still much uncertainty as to how it acts, but all writers are agreed that it is beneficial. Sometimes its effects are extraordinary. After a single application of faradic or galvanic currents the patient may suddenly find that he can perform movements which he had for weeks or months been unable to accomplish. But in such cases it may perhaps be assumed that the regeneration of the affected parts was already far advanced—that they were, in fact, on the brink of recovery—independently of any treatment. At earlier periods the action of electricity is generally less striking, and an apparent success is still less capable of bearing a critical scrutiny. Mitchell, indeed, speaks strongly of the importance of the use of faradic or galvanic currents at the earliest possible moment. As regards the choice of one form of electricity rather than the other, he adheres to a well-known rule which has been laid down by several other writers, namely, that whichever current is found to produce muscular contractions most readily should be used. It has been thought that by stimulating the muscles to contract one helps in keeping them well nourished, besides possibly doing something towards opening up the path for volitional impulses. But Erb maintains that if the reaction of degeneration is present one cannot by galvanisation hasten the recovery of motor power, although he admits that when recovery has once begun its progress may be accelerated.

In employing galvanisation in the treatment of paralysis from an affection of a nerve-trunk one should use the "labile" method. The anode is placed over the plexus above, or on some convenient "indifferent" spot, and the cathode is then slowly moved over the skin, covering each of the affected muscles and nerves in turn. The strength of the current should be such as to excite distinct muscular contractions, but not to cause more than a slight sensation of heat, and a little redness of the skin. Its application should be continued for about ten minutes every or every other day.

If the faradic apparatus be chosen, its two electrodes should be

held in one hand between different pairs of fingers, and they should be placed in succession over the various muscles at a little distance from one another.

Another method of treatment on which Mitchell lays great stress is that of shampooing the affected parts, or (as it is termed) "massage." Persons are trained to perform this manipulation, which requires strength and endurance as well as gentleness, for it ought to be continued for an hour at a time. The limb should first be immersed in a hot bath. Afterwards every part of the skin, so far as the paralysis extends, should be lightly pinched and tapped, and moved to and fro on the tissues beneath. Then the joints are to be moved in turn, and, lastly, the muscles must be gently kneaded and rolled, the power employed being gradually increased. Mitchell says that at the close of the sitting the temperature of the limb is raised by 1° or 2° Fahr., that the muscles are less flabby, and respond more readily to faradisation, and that the skin is sometimes intensely reddened. The patient feels refreshed and sleeps better. It should be repeated every day. A further proof of the efficacy of massage is that when it is carried on for too long a time it sometimes causes lumbar pain, headache, nausea, and exhaustion.

For persistent anæsthesia Mitchell recommends the application of a rather strong faradic current by means of the electric brush. After two or three sittings he has commonly found sensation beginning to return. He has also employed as counter-irritants rags dipped in hot spirits of turpentine and covered with oiled silk. This affects patients very differently, causing unbearable pain in some, while others scarcely feel it.

He quotes the case of a man who had lost sensation in the whole arm as the result of a fall on the shoulder, and who completely recovered after his arm and back had been severely blistered by the sun.

Causalgia may to some extent be relieved by dressings with cold water constantly renewed. The injection of morphia into the affected part is also useful, but Mitchell seems to rely mainly upon the repeated application of blisters. Sooner or later this form of pain almost always subsides.

CRANIAL NERVES.—The account above given of paralysis of spinal nerves is applicable, *mutatis mutandis*, to the cranial nerves likewise. But the latter possess functions which, when disordered, give rise to special symptoms. We will therefore discuss first the disorder of the *motor* cranial nerves (the facial, the nerves of the ocular muscles, and the hypoglossal), then those of the *sensory* nerve of the face (the fifth), and lastly, those of the two *nerves of special sense* (the olfactory and gustatory), which are still left within the province of the physician.

FACIAL PARALYSIS.—The motor division of the seventh nerve—the *portio dura*, or facial nerve—is very liable to affections which destroy its conducting power, and paralyse the muscle which it supplies. This is known as facial paralysis, or *Bell's palsy*, from Sir Charles Bell having discovered its pathology in 1838.

Ætiology.—Of the causes that may give rise to this disease, the most frequent is the direct action of cold upon the side of the face. It may result from sitting at the open window, whether of a room or of a railway carriage, sleeping near a damp, cold wall, or with the face exposed

to a draught of air,—even when the face is only chilled along with other parts of the body, as in a patient of Sir Thomas Watson's who walked about the streets for some days without shoes or stockings during a thaw. Such cases are spoken of by some German writers as "rheumatic," according to that wide use of the term which really deprives it of meaning. It is supposed that the trunk of the nerve becomes affected with a slight inflammatory swelling, which (perhaps by extending into the stylo-mastoid foramen) leads to compression of its fibres. The comparative rarity of similar paralyses of the spinal nerves from cold may depend on the larger size of the intervertebral foramina, and on their greater distance from the exposed portions of the nerves.

Inflammation or tumours of the parotid gland, swelling of the cervical glands, wounds of the cheek, and pressure of the forceps in instrumental delivery, are the chief other causes that may affect the nerve beyond the aqueductus Fallopii.

In its course within that canal the portio dura is liable to be involved in the severe forms of disease of the petrous bone, usually caries and necrosis. In fracture of the base of the skull, the nerve may be torn across. Dr Moxon has recorded ('Path. Trans.,' vol. xx) a case in which a clot of blood was found compressing and destroying this part of the nerve. It seems probable that a similar condition, resulting from the rupture of some small blood-vessel, was present in an oft-quoted case of Sir Charles Bell's, in which facial paralysis followed a box on the ear; and also in one, related by Sir Thomas Watson, of a man who developed facial palsy within three hours of having a fall in which he struck his hip and his elbow, but not his head.

On the cerebral side of the internal auditory meatus, the facial nerve may be implicated in tumours and other diseases of the base of the skull; or its origin in the pons may be affected. As a rule, such cases are distinguished by other nerves being involved, or by further symptoms of cerebral disturbance. But the author met with an instance in which simple paralysis of the right facial nerve, in a man who died of granular disease of the kidneys, was found to be caused by a minute spot of softening which existed in the pons, rather to the left of its centre, with a cyst of the size of a pea; and Dr Gowers has "seen two cases of seizure, evidently apoplectic, in which the only paralysis that followed the seizure was seated in the muscles supplied by the portio dura."

Partial implication of the facial muscles is common in ordinary hemiplegia; and it is a constant feature of bulbar paralysis. In one remarkable case facial paralysis was the earliest symptom of tubercular meningitis.*

The symptoms of Bell's paralysis are very striking, and present many points of interest. When the affection is developed to the fullest possible extent, the patient is altogether unable to move the muscles supplied by one facial nerve. Whether he frowns, or smiles, or laughs, one side remains

* I have myself had a patient who was attacked with Bell's paralysis twenty-four hours after the development of an eruption of herpes zoster on the same side of the face. He came to me about seven weeks afterwards; the loss of power to move the facial muscles was then almost complete, and the "reaction of degeneration" was found to be present when the electrical test was applied. The purple scars caused by the eruption were still visible. No indication of any disease affecting the seventh nerve could be discovered; and I was inclined to regard the paralysis as reflex. He slowly recovered, after the lapse of several months.—C. H. F.

expressionless; the forehead is unwrinkled, no creases appear round the eye, the cheek and chin are marked by no dimples, the angle of the mouth remains in the same position as before. When he tries to close the eyelids forcibly, those on the paralysed side are motionless, a condition pedantically called lagophthalmos (hare's-eye); but since there is a physiological association between forced closure of the eye and elevation of the globe, the latter action, which is effected through the third nerve, still goes on; and the eyeball is involuntarily turned upwards, or upwards and inwards, until the pupil is hidden beneath the upper lid. The actions of spitting, whistling, and blowing are impossible; the sides of the mouth cannot be pursed up; the cheek is not held close to the teeth by the buccinator muscle, and bulges out with expiration of the breath. For the same reason, when the patient masticates, the food cannot be retained within the space between the jaws, and collects outside the teeth on the affected side, so that he has to dislodge it with his finger, unless he keeps his hand pressed against the cheek while he is eating. In speaking, he pronounces the labial consonants indistinctly—*p*, *b*, *f*, *v*, and *m*.

The degree to which the face is distorted when in repose varies considerably in different cases, even among those in which the loss of voluntary power is complete. In some patients the mouth is drawn far over to the opposite side, the eye stares fixedly, and the countenance has a constant and most comical aspect. This seems to be due to a contraction of the antagonist muscles analogous to that which causes distortion of the fingers when the interossei are paralysed. But perhaps one element in the result is an absolute loss of tone in the affected muscles, which occurs only when the nerve is completely destroyed, as in cases of necrosis of the temporal bone; for in most of the so-called "rheumatic" cases the patient's features are but little disturbed while they are at rest, so that at a cursory glance one may scarcely notice that anything is amiss with him. Even when he is altogether unable to close the eye, the palpebral aperture may appear but little wider than on the unaffected side, and the *tensor tarsi* muscle may still be able to keep the lachrymal puncta in contact with the globe, and to prevent the tears from running down over the cheek, as they do in the most extreme forms of the disease. Perhaps during sleep, when the levator palpebræ is relaxed, the eyelids may approach one another more nearly than would be expected from the patient's inability to close them when he is awake, with the levator acting in opposition to his efforts. In this way one may perhaps find an explanation of the not infrequent absence of inflammation, which one would expect to be a necessary result of particles of dust settling upon the exposed eyeball. As is well known, the conjunctiva is very apt to become inflamed under such circumstances, and the cornea may lose its transparency or ulcerate; but Valleix had a case of facial paralysis of twenty years' duration in which no such results took place. In the daytime, as Trousseau remarks, the patient is able to compensate for the failure of movement in the eyelids by bringing the globe into various positions so as to wipe it upon different parts of their inner surfaces; or he may push down the upper lid with his finger.

Among the indirect effects of facial paralysis, impairment of the sense of *smell* is sometimes mentioned; and it is attributed to a dry condition of the corresponding nostril, which results from the escape of the tears over the cheek.

A much more frequent symptom is a perversion of the sense of *taste*. Trousseau, for example, speaks of a man who said that his food tasted like "salt plaster;" and other patients have experienced a subjective metallic or sour taste in the fore part of the tongue on the affected side; or a loss of power to detect acid, sweet, or saline flavours. This appears to be directly due to interruption of the conducting power of a branch of the seventh nerve, the chorda tympani. It is also said that the secretion of saliva may be diminished from the same cause.

The sense of *hearing* is often abnormally acute; there is an augmented sensibility for all musical notes and sounds, the patient hearing a watch at a greater distance from his ear on the affected than on the healthy side; there is also an increased capacity for the detection of sounds of very low pitch; and there may be a subjective sensation of hearing an acute sound. This state of the auditory sense has been (barbarously) named "oxyakoia." Brown-Séquard propounded the theory that it is due to hyperæmia of the auditory nerve consequent on paralysis of its vaso-motor nerves. But Lucæ has since shown that it is the result of paralysis of the stapedius muscle, which receives a branch of the facial nerve; its antagonist, the tensor tympani, being no longer opposed, keeps the membrane too much on the stretch ('Berl. klin. Woch.,' 1874, Nos. 14, 16). Auditory hyperæsthesia, therefore, should never occur in facial palsy of peripheral origin.

Another branch of the facial nerve, which is often affected in Bell's paralysis, is that which goes to the soft palate. The late Professor Sanders showed ('Edin. Med. Jour.,' 1865) that there is then "a vertical relaxation or lowering of the corresponding half of the velum, with diminished height and curvature of the posterior palatine arch," a condition which he believes to be due to loss of power in the levator palati muscle. In certain cases the uvula is turned to the paralysed side; but this seems not to be constant, and it possesses little significance, inasmuch as lateral deviation of the uvula sometimes occurs in healthy persons. If the levator palati is paralysed, it would follow that the lesion of the portio dura must be on the cerebral side of the geniculate swelling of the nerve in the petrosal bone. But the symptom is more often looked for than seen.

Diplegia facialis.—The right facial nerve is as liable to paralysis as the left. It sometimes happens that *both nerves* are paralysed in the same patient; the symptoms are then to a certain extent peculiar, so that such cases have been dignified with the special name of "diplegia facialis." The chief point is that there is no distortion of the features under emotion of any kind. The face remains without expression, and, as Romberg put it, the patient laughs or cries behind a mask. Double facial paralysis may be caused by a new growth or gumma, involving both nerves at the base of the skull; but more frequently it is due to disease affecting the two petrous bones separately, or to the action of cold upon both sides of the face in succession.

Diagnosis.—This is seldom difficult when facial palsy is fully developed. One must not omit to notice whether any other nerves beside the seventh are paralysed, for if such is the case there is reason to suspect the presence of some new growth or syphilitic disease at the base of the skull. Even when the loss of power is very slight and incomplete, one can for the most part decide its peripheral origin by a little failure of expression limited to one side of the face, and by a narrow chink remaining between

the closed eyelids, for these symptoms do not accompany a partial paralysis of the facial nerve from disease of the brain.

Course.—The onset of Bell's paralysis is sometimes gradual, sometimes sudden. Among hospital patients it often happens that what first attracts the patient's notice is his finding himself unable to spit or to whistle; or he may find his face "all on one side" when he gets up in the morning.

The subsequent course of facial palsy varies greatly. When due to destruction of a considerable portion of the nerve, as in cases of necrosis of the petrous bone, it is of course permanent and incurable. Although recovery is possible after an injury, such as the division of the nerve in a surgical operation, yet this seldom occurs before the lapse of several months, and it is apt to remain incomplete. Even where the affection is the direct result of exposure to cold, it may last from four to six months; but sometimes it gets well much more rapidly, the patient beginning to regain power over the muscles at the end of ten or twelve days, and recovering completely within a few weeks.

Prognosis.—It might be expected that one of the best indications of the probable duration of the affection would be the presence or the absence of paralysis of the proximal branches of the facial nerve, the chorda tympani, and those to the stapedius and palate. Dr Sanders, however, maintained that the prognosis is not more unfavourable in cases in which the last-named branch is implicated. The question is not of much practical importance, because we seem to have in the application of electricity a method of determining the gravity of an attack of facial paralysis from the first.

This point has been carefully worked out in Germany by Brenner and by Erb. From their observations it appears that in the *mild* form of the affection, which recovers in two or three weeks, the muscles and nerves of the paralysed side of the face react both to galvanic and to faradic currents exactly in the same way as on the healthy side. In only a single instance of rapid recovery could Brenner detect a slight and transient diminution of excitability to both kinds of current. But in the *severe* form (which includes a large proportion of those cases which are due to the direct action of cold) those peculiar effects are observed which have been described above as the "reaction of degeneration." Indeed, it was in a case of facial paralysis that this reaction was first noticed by Baierlacher in 1859. The prognosis is then always so far unfavourable, that no improvement is to be looked for until two or three months have passed, that a complete recovery seldom takes place before the lapse of another period of equal duration, and that more or less weakness or stiffness of the affected muscles sometimes remains during the rest of the patient's life. Erb describes an intermediate form of facial palsy in which the excitability of the nerve is not completely lost, although the muscles present the characteristic sensitiveness to feeble galvanic currents with absence of faradic contractility; such cases, he says, recover in from four to six weeks.

In severe cases of Bell's paralysis *spasmodic affections* are observed at an advanced stage. When the patient is just beginning to recover voluntary power over some of the muscles, it often happens that the effort to induce contraction in one of them causes simultaneous movements in muscles of a different part of the face. He may wish to move his eyebrow or his eye, and he involuntarily draws his mouth to that side; he may intend to close his lips, and he finds that he also shuts his eye. In a patient who was under the author's care in 1877, the distortion of the mouth was so great when an attempt was made

to close the right eye, that the clinical clerk actually supposed the left side of the orbicularis oris to be paralysed. Hitzig, who has especially studied these curious phenomena ('Arch. f. Psych.,' 1872), notes that they sometimes occur while there is absolute paralysis (for all volitional efforts) of the muscles which are concerned in the associated movements. It seems difficult to avoid the inference that the motor impulse must find its way through the obstructed part of the nerve along the fibres which have regained their conducting power, and so reach the peripheral branches. But it would seem that the facial nucleus in the medulla oblongata is also in a condition of exalted irritability in many cases, for Hitzig has found that touching the affected side of the face sometimes gives rise to spasms which may even involve the muscles of the opposite side. In one case of facial paralysis, contractions actually extended to the muscles of mastication, and to those of the limbs. The occurrence of associated movements is commonly a precursor of the return of voluntary power over the paralysed muscles. They generally quickly disappear, but sometimes they seem to delay recovery, and (according to Erb) they have been known to last for as many as thirteen years.

On the other hand, when the paralysis is to be permanent, *tonic contractions* of some or all of the muscles often show themselves, which seem to have been described by Duchenne before they had attracted the notice of other observers. The result may be an elevation of the angle of the mouth, a deepening of the naso-labial groove, a narrowing of the palpebral aperture, or a general exaggeration of all the markings of the affected part of the face. The features are sometimes dragged over to such an extent that one might fall into the error of supposing that the healthy side was the one paralysed. Duchenne asserted that whenever in the course of facial paralysis some particular muscle recovers its tone earlier than one would have expected, one should be prepared for the supervention of tonic spasm in it. But this statement seems inconsistent with the fact that such spasms depend upon the occurrence of degenerative changes in the muscles themselves—a fact established by a case of Hitzig's, in which a patient who already had contraction of the orbicularis palpebrarum from a former seizure of Bell's paralysis was attacked a second time by the same disease, without the muscle in question undergoing relaxation.

Treatment.—This is still a doubtful matter. When the discovery was first made that the muscles in many cases exhibit an augmented susceptibility to galvanic currents, most observers assumed that the application of that form of electricity would cure the disease more quickly than the faradisation which had before been employed. But in 1869, Erb, after analysing the cases which had then been recorded, expressed the opinion ('Deutsches Archiv,' vi) that the "severe" form, in which alone the reaction of degeneration is present, cannot be materially shortened in its course by galvanism. According to him, the constant current is of little value until the conductivity of the nerves is re-established, although he believes that it then accelerates the recovery of voluntary power over the muscles. He recommends that the anode should be placed behind the ear, and the cathode moved over the paralysed half of the face; or else that the two poles should be placed one over each mastoid process, the affected side receiving the anode. In mild cases he says that each application of the current is usually followed by increase of voluntary power over the muscles. It must not be forgotten that Duchenne believed that he attained still better results from

faradisation; he speaks of cases which had lasted for years, and which were, without exception, cured or greatly relieved by this procedure, although all other treatment had failed. So impressed was he with its potency, that he regarded it as capable of inducing secondary spasms, and consequently advised that when the muscles began to regain their tone the intermissions of the battery should be reduced in frequency to not more than four in the second. He did not indeed recommend that faradisation should be laid aside altogether, even when contractions of the affected muscles had set in; but he suggested that they should also be stretched with the fingers at regular intervals, or that a wooden ball should be carried in the cheek.

On the whole, however, it would appear that as much (or as little) is to be anticipated from medicinal treatment as from electricity. All recent writers recommend leeches behind the ear, blisters, and mercury or iodide of potassium as the best remedies for facial paralysis during the first two weeks. Erb, in particular, says that the iodide in full doses has appeared to shorten the duration of the disease, even in some "severe" cases.

PARALYSIS OF THE MUSCLES OF THE EYEBALL.—Of the movements of the eyeballs some are direct results of the action of single muscles; while the remainder, for which several muscles are brought into play, are capable of a physiological analysis far more exact than that to which the movements of any other parts of the body can be submitted. Since three distinct cranial nerves are concerned, the study of ocular paralysis has very important bearings upon medical diagnosis. The affections of these nerves cannot be taken separately, because under normal conditions the two eyes are always moved simultaneously, and because the muscles of opposite sides which are associated together are often not the two of the same name, but one of a certain name and the antagonist of its fellow, receiving nerves from different sources. For example, the external rectus of one side, supplied by the sixth nerve, works, not with the *external* rectus, but with the *internal* rectus of the other side, a muscle which receives its branch from the third nerve. Now, one of the most important symptoms of loss of power in the external rectus is a deviation from the natural movements of the internal rectus of the opposite eye, and *vice versâ*, so that the affections of these muscles have to be discussed together.

At the same time it must be borne in mind that two of the ocular muscles receive each the entire distribution of a cranial nerve—the external rectus that of the sixth, the superior oblique that of the fourth. The consequence is that either of these muscles is more likely to be separately paralysed than any single one of the rest, which are all supplied by the third nerve, and that the affections of the former muscles are far the most important from a practical point of view. Moreover, these affections present differences in their symptoms which, *mutatis mutandis*, are typical of those which characterise the paralysis of all the other muscles; so that it is in every way convenient to describe them first.

Paralysis of the sixth nerve.—Each external rectus muscle is associated in its principal movement with the internal rectus of the opposite side. Their combined function is of the most simple kind; they merely carry the eyes to the right or to the left, without any change in the direction of the vertical axes of the globes. Thus, if an external rectus be paralysed, the corresponding eye cannot be moved outwards when the other eye is moved inwards. Let us, for example, suppose that the patient has his *left* sixth nerve

paralysed. The result is that he is unable to look with his left eye at any object to his left. If we hold a candle before him, and move it to his right side, his two eyes follow it until the right one has the edge of its cornea touching the external canthus, and the left has a small part of its cornea buried beneath the caruncle. But if we now carry the candle to his left side the right eye alone follows it; the left remains motionless, and looks straight forwards, or may perhaps perform a slight zigzagging movement to the left under the combined influence of the two obliqui. The further the object is carried to the left the greater is the difference in the directions of the two eyes; and this *primary deviation* or *squint* at once clears up the nature of the case when the paralysis is complete.

If the loss of power is partial the affected eye only lags behind its fellow, and no obvious squint may appear. In such a case one can generally make out the nature of the defect by covering the right or healthy eye; the patient then discovers that he has not got his left eye fixed upon the object, and he accordingly moves that eye further to the left. This correcting movement may be readily detected by a close observer; but what is far more striking is a movement which is simultaneously made by the right eye. For the left external rectus being partially paralysed, any voluntary movement of that muscle requires the exertion of far more effort than that which would have sufficed under normal conditions to produce the same degree of contraction. But whatever amount of nervous energy is put forth, it is at the same time thrown upon the associated muscle—the right internal rectus—which is in possession of its full vigour; and thus the right eye moves two or three times as far as the left one.

This movement of the healthy eye is called *secondary deviation;* and it is of the more importance because it affords a sure means of distinguishing a paralysis of the external rectus from a mere contraction or shortening of one or both of the internal recti, such as exists in cases of ordinary strabismus. When the loss of power is considerable this distinction is indeed made by the fact (above mentioned) that the primary deviation increases as the object looked at is carried further over towards the left side; for if the affection were a mere convergent strabismus the axes of the eyes would remain at exactly the same angle, the one lagging at a fixed distance behind the other, but nevertheless travelling with it, so that such an affection is sometimes called "concomitant." But in slight cases this criterion fails. Its place is then taken by the secondary deviation, the *augmented amplitude* of which is (as we have seen) dependent on the existence of paralysis. In cases of ordinary strabismus the secondary is exactly equal to the primary deviation, since the muscles on both sides possess equal power.

The other symptoms of paralysis of the sixth nerve are subjective sensations experienced by the patient, and of movements to which they lead.

When his eyes are turned in a certain direction he sees double images of the objects at which he looks; and this *diplopia* is not infrequently the first thing which draws his attention to the fact that something is amiss with him. We will assume as before that the *left* external rectus is the muscle paralysed, and we will call the image which is formed upon the retina of the affected (or left) eye "the *false* image;" the one which is formed on the unaffected (or right) eye "the *true* image." It is obvious that double vision occurs only when the eyes are directed to the left, for it is then that they fail to converge properly upon the object. And

a moment's consideration will show that since the left eye has its axis turned inwards (or to the right), the false image must be formed upon the inner side of its retina, and not upon its centre; and, consequently, that it appears to be *outside the true image* or *further to the left.* Both images are upon the same level, and both are upright. By placing a piece of coloured glass before one of the patient's eyes, we can enable him to distinguish very easily which image is formed by that eye, and which by the other; reddish violet is said to be the best colour for this purpose.

It must be borne in mind that diplopia is not necessarily a proof that paralysis of any of the ocular muscles is present. Double images are sometimes formed upon the retina of a single eye; and in other cases they are due to a mere "concomitant strabismus." Making the patient look at the object with each eye separately will exclude the former condition; but the latter can only be dismissed from further consideration if it is found that the two images get wider apart as the object is moved further to the left. One must not assume that diplopia which is of recent origin and which began suddenly cannot be due to concomitant strabismus; for it sometimes happens that a patient whose ocular muscles are imperfectly antagonised is able to keep up the balance between them until he overfatigues his eyes, or until he is weakened by some illness, when he may abruptly begin to squint.

On the other hand, it would be a mistake to suppose that whenever there is a loss of power in the external rectus muscle the patient must necessarily be conscious of diplopia. There is always the possibility that he may not previously have been accustomed to use the two eyes together, or that he may have been in the habit of concentrating his attention upon a single retina, in which case all that he is likely to notice is a blurring of the objects at which he looks, interfering with the distinctness of his vision. One can generally make such a person aware that he really sees double by directing him to fix his eyes upon some bright small object, especially if a coloured glass be held in front of one eye.

Another subjective symptom is that which is known as the "*erroneous projection*" of the visual field. We have seen that a patient who has paralysis of the left external rectus muscle refers the false image of any object to the left of him which he sees with both eyes open, to a position outside its true position, *i. e.* more to the left. The same thing occurs if he is told to look at an object with the right eye closed; except that his judgment is then based upon an appreciation of the degree of effort required to bring his left eye to bear upon it, which effort is augmented in proportion to the failure in the power of the muscle. One consequence is that such a patient feels giddy, and may even stagger, if he attempts to use his left eye alone—a point of some importance, because a careless observer might mistake it for a sign of serious cerebral mischief. Another result of "erroneous projection" may be employed as an aid in the diagnosis of paralysis of the ocular muscles. The patient is told to close the eye supposed to be unaffected, and to strike suddenly at an object placed towards the outer side of the other eye. If the external rectus muscle is paralysed he is sure to miss the object by going to the outer side of it.

Some patients free themselves from the uncomfortable sensations to which this affection gives rise, by keeping the head fixed over towards the paralysed side, so that the images of the objects at which they look may be referred to their right positions.

When paralysis of the external rectus muscle has lasted for a considerable length of time the position of the eyeball often undergoes a further change. It is now drawn inwards by the uncontrolled action of its internal rectus, so that a convergent squint is constantly present, whatever may be the direction in which the patient looks, unless perhaps it be very far to the right. Such a condition was designated by von Graefe the "secondary contraction of the antagonistic muscle;" and he pointed out the curious fact that it is sometimes altogether disproportionate in degree to the loss of power in the muscle originally affected.

Paralysis of the fourth nerve.—The action of the *obliquus superior* is far from being as simple as that of the rectus externus; and its paralysis is proportionately difficult of detection. Each of the two recti, when acting alone, rotates the cornea slightly inwards, beside raising or lowering it; but as one of the obliqui tends to rotate it in an opposite direction, the resultant of their combined action is a straight movement in a vertical plane. The left inferior rectus, by itself, would lower the cornea and carry it towards the right; the left superior obliquus, by itself, would lower it, and carry it round to the left. Acting together, they move it straight downwards. It is further evident that the rotatory action of the two recti upon the (left) cornea must be more apparent when the eyeball has been previously directed outwards to the left; that of the obliqui when it has been previously directed inwards, to the right.

Now let us suppose that the left superior oblique muscle is paralysed. The result must necessarily be that when the patient looks downwards the cornea will be carried to the right by the inferior rectus, which has now no antagonist capable of counteracting its tendency to rotate the globe in that direction. In other words, he will squint to the right and slightly upwards with the affected eye when it is directed upon an object towards his feet. The "secondary deviation" of the sound eye will be downwards and to the left. These symptoms, however, are comparatively little marked, so that the subjective phenomena of diplopia acquire a far greater relative importance in the diagnosis of the paralysis of the fourth than in that of the sixth nerve. The double images are seen chiefly when the patient looks downwards, and their position in regard to one another at once indicates what muscle has lost its power. Thus, firstly, the false image lies *below* the true one; secondly, it is placed to its *left;* and thirdly, it appears to be *tilted* in a particular manner. This last circumstance depends upon the way in which the affected eye is rotated, for since the upper end of an object necessarily has its image formed upon a part of the lower half of the retina outside the normal vertical meridian of the retina, the object itself seems to that eye to be placed obliquely *with its upper end tilted to the right.* Another point noticed by von Graefe is that the false image seems to be on a plane nearer to the eye than the true one; this, he says, depends upon their both being referred to positions upon a horizontal surface spread at the patient's feet, so that the true image, being the upper of the two, is naturally supposed to be the more distant. Moreover, the patient, in order to avoid a sensation of giddiness when he looks with both eyes at objects before him, keeps his head turned downwards and to the right—a position which is very characteristic.

Paralysis of the third nerve.—Since this nerve, unlike the sixth and fourth, is distributed to four of the ocular muscles, the symptoms produced by its

paralysis must of course be different, according as the affection is limited to particular branches or involves all of them alike.

In paralysis of the *internal rectus* the symptoms are the converse of those which belong to paralysis of the abducens. If we take, as before, the muscle of the left side, the "primary deviation" occurs when the eyes are directed on an object towards the right; the affected eye cannot then be moved inwards while the other one is being moved outwards, and a *divergent* squint is produced. The "secondary deviation" of the right eye is towards the right. Double images are perceived by the patient when he looks to the right, and the distance between them increases the further the object is moved in that direction. They are both upon the same level and both upright. They are said to be "crossed;" that is, the false image (which is seen by the left eye) lies to the *right* of the true image (which is seen by the right eye). The "false projection" of the image takes place toward the right; so that the patient, if he aims suddenly at an object with the right eye closed, misses it by going too far to the right. To avoid giddiness the patient keeps his head over to the right.

In paralysis of the left *inferior rectus* most of the symptoms are the converse of those which are produced by paralysis of the superior oblique muscle. As in that affection, the affected eye squints a little upwards when the patient looks downwards, but the rotation of the globe is now to the left. Diplopia occurs when the eyes are directed upon an object below them; the false image is below the true one, lies to its right, and has its upper end tilted to the left.

In paralysis of the left *inferior oblique* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the right. The false image lies above and to the left of the true one, and has its upper end tilted to the left.

In paralysis of the left *superior rectus* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the left. The false image lies above and to the right of the true one, and has its upper end tilted to the right.

Lastly, if the *whole of the third nerve* on the left side is paralysed, the symptoms are necessarily in great measure identical with those which indicate affections of the muscles severally supplied by its branches. There is complete loss of power to move the eye inwards or upwards. It cannot be moved straight downwards, but it can be lowered slightly if at the same time it is carried a little outwards. In the direction horizontally outwards its play is perfectly free. These last two movements are effected respectively by the muscles supplied by the fourth and sixth nerves, and in the former of these the globe is of course made to revolve on its antero-posterior axis by the uncompensated action of the superior oblique muscle. Indeed, the rotation is so marked under such circumstances—especially when the eye is allowed to move as little outwards as possible—that it affords a striking confirmation of the modern views with regard to the action of the ocular muscles.

The visual range of the affected eye is reduced, according to von Graefe, to about one twelfth of its normal extent; it is limited by a straight horizontal line above, but below by a curved line which sweeps downwards and then outwards.

The direction of the "primary deviation" of the left eye, and that of

the "secondary deviation" of the right eye, necessarily vary according to the position of the object towards which the patient directs his sight; the relations of the false to the true image of course undergo corresponding variations, and so also does the "erroneous projection" of the objects that meet his eye on different sides. The consequence of this is that giddiness is a far more marked symptom when several of the ocular muscles are paralysed than when one alone is affected; and it cannot be obviated by adopting any particular posture for the head, nor, indeed, by any method except that of keeping the affected eye closed. It is only when the patient looks at an object in the extreme left of the visual field that he ceases to squint and sees a single image with the two eyes.

In paralysis of the entire third nerve, beside the affection of the four ocular muscles, there is loss of power in certain other muscles which are also supplied by that nerve. One of these is the levator palpebræ, and the result is that the upper eyelid is dropped, a condition which is termed *ptosis*. The patient is altogether unable to open the eye in the ordinary way; at most he can only slightly separate the lids by wrinkling the forehead—by means of the occipito-frontalis. Ptosis may exist without paralysis of any other muscle; indeed, it is much more common than an isolated affection of any other branch of the third nerve.

Again, the sphincter muscle of the pupil receives filaments from this nerve, and paralysis of them leads to dilatation of the pupil, or (as it is termed) *mydriasis*. Ophthalmic surgeons state that the aperture of the iris never becomes so wide from paralysis as it does under the influence of atropine; but it is often large enough to interfere very much with the accuracy of vision, owing to the formation of "circles of diffusion" upon the retina.

Lastly, the ciliary muscle is also supplied by the third nerve, and hence *paralysis of accommodation* may be one of the symptoms of disease of that nerve. Except in very short-sighted persons, this greatly interferes with distinctness of vision for small print and other near objects. To detect it, one must test the range of accommodation in the usual manner with a convex lens. The head is usually turned obliquely towards the sound side.

Ophthalmoplegia externa.—When all the muscles of the eyeball are paralysed together, there is immobility of the globe with ptosis, but with no squint, and with no double vision for distant objects—unless one eye only is paralysed. This term was used by von Graefe. The paralysis is usually double. More than half the cases are syphilitic and curable. The term *Ophthalmoplegia intima vel interna* has been applied by Hutchinson to paralysis of the iris and ciliary muscle only ('Med.-Chir. Trans.,' 1878, p. 215, and 1879, p. 307).

Ætiology of ocular paralysis.—With regard to the causes of paralysis of the third, fourth, and sixth nerves, our knowledge is at present imperfect. One point of great practical importance is that almost all those cases in which the affection remains limited to a single nerve or to a single branch of the third nerve, terminate sooner or later in recovery. Their anatomy is consequently unknown, but oculists say that they are either syphilitic or "rheumatic" (*i. e.* non-syphilitic).

In 1876 a man died in Guy's Hospital of aortic aneurysm who had about nine months before been in another ward suffering from ptosis of the left eye and pain in the left side of the head. An old meningeal apoplexy was found; the termination of the left internal carotid artery

was dilated, and its coats much thickened; moreover the third nerve on that side was adherent to the side of the artery and stained of a deep brown colour. Sir William Gull used to say of the third nerve that it ran a "dangerous course," on account of its liability to compression in passing between the posterior cerebral and the superior cerebellar arteries, if these vessels should happen to become diseased; and this view certainly accords well with the fact that ptosis is very apt to occur in old people whose arteries are dilated and tortuous.

Of the fourth and sixth nerves it must be remembered that their long and exposed course along the base of the brain renders them very liable to be affected by tubercular or other effusions in that region.

When two or more nerves are simultaneously affected, one suspects the presence of some malignant growth or aneurysm or gumma at the base of the skull. An intra-cranial carotid aneurysm is very likely to compress the sixth nerve for some time before it reaches the rest.

A syphilitic affection of isolated nerves is not necessarily a gumma, for in one case there was ptosis of each eye, while the ocular muscles entirely escaped, and it is difficult to suppose that the corresponding parts of the third nerves were both occupied by a gumma. Probably the lesion is often syphilitic neuritis. Von Graefe is said to have traced to syphilis about one third of all cases of ocular paralysis.

We shall hereafter find that these paralyses may accompany various diseases of the base of the brain, and that they are sometimes early symptoms in locomotor ataxy; but when an organic lesion of the brain or spinal cord is present, one can almost always discover other symptoms, which exclude the supposition of peripheral paralysis.

Prognosis.—Most cases of ptosis or of paralysis of the various ocular muscles recover, except when they are due to aneurysm or malignant growth compressing the affected nerve. But it must be added that recovery is sometimes slow, taking many months; and that it may be imperfect, so that one or more of the affected muscles remain permanently weak.

Treatment.—When the palsy can be traced to syphilis, treatment by mercury is necessary and is usually successful. Iodide of potassium is also useful, and is often given when there is no evidence of syphilis. The application of blisters behind the ear is also believed to be serviceable. Benedikt and Erb have found galvanism useful, a current from six, ten, or fourteen cells being applied for two or three minutes at a time, with the anode on the temple or the back of the neck, and the cathode on the closed eyelids. The improvement is said to be often instantaneously manifested, and Benedikt thinks that if no good is effected within the first fortnight there is no object in continuing this treatment; but according to Erb many cases require to be galvanised for several months before any result is discoverable. Faradic currents also are sometimes useful.

Paralysis of the ninth or hypoglossal nerve is a common symptom of cerebral, pontine, and bulbar paralysis, but as a lesion of the nerve-trunk it is a comparatively rare affection. It is attended with one very remarkable effect, —an extreme degree of wasting on the corresponding side of the tongue. The author has seen two cases of this kind: one was in an old woman, in whom a cancerous nodule (secondary to a cancer of the breast) involved the nerve where it passes through the base of the skull; the other in a boy, who had extensive caries and necrosis of the atlas and neighbouring bones.

In both instances the paralysed half of the tongue was remarkably flaccid and wrinkled; and its tip was curved round towards the affected side. Similar cases have been recorded by Dupuytren, Sir James Paget, and the late Mr Fairlie Clark. In Paget's case ('Clin. Trans.,' vol. iii) the affection was caused by necrosis of a part of the occipital bone, from an injury. Several pieces of dead bone were removed by operation; a few days afterwards the wasted part of the tongue began to grow larger, and within a month it had nearly regained its former size and muscular power. Cases of double hypoglossal paralysis are on record apart from those due to cerebral hæmorrhage and to narcotic poisoning, as in the administration of chloroform. The tongue lies motionless and low between the rami of the mandible, and articulation, deglutition, and even respiration are impeded.

Paralysis of the fifth nerve.—The principal symptom of this affection is anæsthesia of the face. The loss of sensation or the corresponding subjective sensations of numbness and painful tingling may in some cases be traced with great accuracy to the median line of the forehead, nose, and mouth. A circumstance which commonly first attracts the patient's notice is that when he puts a cup to his lips he feels only half of it; it seems to him exactly as though it were broken. The eyelashes and conjunctivæ are perfectly insensible; so also is the nostril, and liquor ammoniæ may be applied to it without causing sneezing; but there is no impairment of the sense of smell, except from diminished secretion of tears and consequent dry state of the mucous membrane. On the other hand, the sense of taste has in several cases been found absent in the anterior portion of the tongue. Three instances of this are given by Romberg in which quinine was used to test the gustatory powers of the patient. It is the same part of the tongue which is deprived of the sense of taste in cases of facial paralysis; and the only possible explanation seems to be that the chorda tympani is a true gustatory nerve, and that in different parts of its course it runs both with the fifth and the seventh nerves, the great petrosal nerve being probably the connecting branch.

Another effect of lesion of the fifth nerve is paralysis of the masticating muscles on the affected side. This only deprives the patient of one movement completely, that in which the lower jaw is carried forwards and towards the healthy side by the external pterygoid muscle. His power of chewing food is much less impaired than might have been expected, but he really masticates only with the muscles of the unaffected side. If he is thin, one can see that the temporal and masseter fail to swell out when the mouth is forcibly closed; even if he is well nourished one can easily feel that they do not harden.

In certain cases of facial anæsthesia the circulation in the affected parts is disordered, or their nutrition is to some extent impaired. Romberg mentions one instance in which the cheek became livid when exposed to cold, in marked contrast with the healthy hue of the opposite side of the face. Sponginess of the gums, ulceration of the mucous membrane of the nose and mouth, and hæmorrhage from their surfaces have been observed. But such changes are rare and comparatively unimportant, whereas there is another affection which is of frequent occurrence, namely, inflammation of the eye, leading to sloughing of the cornea. Physiologists have made this the subject of elaborate investigations on account of its bearing upon the question of trophic nerves. It is now agreed that the inner portion of the trunk

of the fifth nerve contains all those fibres which are specially concerned in the nutrition of the eyeball; but there is still a difference of opinion as to whether the result is due to a mere interruption of their conducting power, or (as Charcot thinks) to some directly irritant influence. Snellen's view that the anæsthesia is its immediate cause, the eye being exposed to mechanical injuries from the loss of the protection afforded by the sensitive nerves, is now generally abandoned; indeed, Charcot cites several instances in which the cornea sloughed as the result of affections of the fifth nerve, unattended with loss of sensation.

We have seen that in affections of the great nerve-trunks of the limbs, when the continuity of their fibres is not completely destroyed, anæsthesia, if present at all, soon passes off. We should therefore expect that a nerve running so protected a course as the fifth would be little liable to have its conducting power for sensory impressions interfered with except by lesions of a serious kind. And this appears to be the case. Romberg, indeed, mentions that a watchman, who had to spend his nights in a hall where the left side of his face was constantly exposed to a draught, experienced violent pains in that part, and subsequently anæsthesia of the second division of the trigeminal nerve; within three weeks, under treatment with iodide of potassium, he recovered. But such instances are extremely rare. The same writer relates two or three other instances in which loss of sensation in the face was due to morbid changes in the Gasserian ganglion, that structure having been found after death to be swollen, indurated, or discoloured. The nature of the change in question seems, however, to be doubtful, and we are not told why a fatal termination occurred.

In the great majority of cases paralysis of the fifth nerve is due to destruction of the trunk or of its ganglion, by caries and necrosis of the bones, syphilitic disease of the pons or meninges, cancerous or sarcomatous growths, or, lastly, aneurysm of the internal carotid artery.

Paralysis of the olfactory nerve.—We will next consider loss of smell, or, as it is termed, *anosmia*. The corresponding affections of the auditory and the optic nerves are too important to be treated adequately in a work on general medicine.

A man may be deprived of the sense of smell in one side of the nose, or in both. When the affection is unilateral he is very likely to remain ignorant of his loss, unless one is particular to test the power of the olfactory nerves separately, carefully closing each nostril in turn. We shall hereafter see that this form of anosmia frequently accompanies hemiplegia. When both nerves are affected the patient is sure to notice the defect His inability to perceive odours is then complete; he is altogether insensible to the most fragrant perfumes and to the strongest stinks. But he retains his susceptibility to pungent vapours, such as those of ammonia and of acetic acid, and snuff is still capable of making him sneeze. The impressions from all these substances are conveyed to the brain by the branches of the fifth nerve which are distributed to the nasal mucous membrane; and it is when that nerve is paralysed that they fail to excite sensations.

But the symptom which chiefly forces itself upon the notice of a patient affected with complete anosmia is not the loss of the sense of smell (as he understands it), but that of a large part of what he supposes to be the sense of taste. He can, indeed, still recognise bitter and sweet, sour and salt; and he can distinguish the rough or smooth character of the food

that he takes into his mouth. But in all other respects his sense of taste appears to be extinguished. He cannot tell one kind of meat from another; apples, onions, and turnips appear the same to him; all kinds of wine seem to have lost their flavour, tasting merely like sour or sweetish water, except that they are more or less rough to the palate. If it were not from habit and prejudice he would probably be altogether indifferent as to the nature of his food. Evidently, therefore, physiologists are right in maintaining that the true gustatory sense is limited to four tastes—sweet and bitter, acid and saline; and that the other perceptions known as tastes belong to the sense of smell.

The pathological significance of anosmia was first fully worked out by Dr Wm. Ogle ('Med.-Chir. Trans.,' 1870). He refers, in the first place, to five cases in which this condition resulted from blows upon the head, the part struck being generally, if not always, the occiput. He believes that in such cases the olfactory nerves are torn across as they pass through the holes in the ethmoid bone. It seems difficult to suppose that all the filaments of both nerves should be simultaneously ruptured; and one would be rather disposed to think that the olfactory bulbs themselves had shared in the bruising of the anterior lobes of the brain, which is so common a result of injuries to the back of the head.

The remaining causes of loss of smell are independent of any affection of the olfactory nerves, but it may be well to enumerate them here. One is, perhaps, an absence of the pigment in the mucous membrane of the upper part of the nose; at least, a case has been recorded in which a negro boy lost the power of smell more or less completely at the same time that his skin became white. Dr Ogle supposes that the nasal pigment also underwent absorption, and he cites some other evidence to show that there is a relation between the presence of this pigment and the olfactory sense. Another cause of anosmia is closure of the passage into the posterior nares by adhesion of the palate to the pharynx; this prevents the patient from drawing air through the nose, and so deprives him of susceptibility to odours, and also to flavours. But there are other conditions in which the one kind of impressions is lost without the other, and which therefore would correspond exactly with the popular conception of an affection of smell, independently of taste. Such cases always depend upon an obstruction to the passage of air upwards to the olfactory region through the anterior nares, while the passage through the posterior nares is open. They generally result from thickening of the Schneiderian membrane by chronic catarrh, so that one part of the lateral wall of the nose is brought into contact with the septum. Dr Ogle mentions the case of a woman who for several years had entirely lost smell from this cause. Some years ago a lady came to the writer who said that for ten months she had lost both smell and taste as the result of a severe cold. Probably what she was really devoid of was the power of appreciating flavours. Some liquor arsenici hydrochloricus was prescribed, and in less than a fortnight she perfectly recovered.

With respect to the loss of the sense of taste, the cases on record, which are certainly of nervous origin, are almost entirely referable to paralysis of the chorda tympani (cf. p. 404). Taste is sometimes affected on one side in hemiplegia. It is unaffected in glosso-pharyngeal paralysis.

PERIPHERAL NEURITIS.—It has long been known that nerve-trunks are liable to inflammation; but we have only lately learnt that many cases of

neuralgia, of trophic changes, and of motor and sensory paralysis are due to this lesion. Leyden explains reflex paralysis generally by ascending peripheral neuritis, and his views have been confirmed by histological evidence. The Wallerian degeneration of a nerve severed from its trophic ganglia is due to parenchymatous neuritis.

In describing neuralgia and peripheral paralysis, we have taken the most uncomplicated examples of both as types: neuralgia of a primary or idiopathic character, not due to pressure or inflammation affecting the nerve; and paralysis such as would be produced by direct injury severing the nerve from its centre. But we have already stated that the less typical kinds of neuralgia, particularly sciatica, are probably pathologically interstitial neuritis; while the trophic disturbance of the skin called shingles and the neuralgia which follows it are due to neuritis of the posterior roots and their ganglia.

Again, the remarkable form of paralysis which sometimes follows Diphtheria is probably due to neuritis; and we shall see that the same pathology most likely belongs to the affections known as Saturnine palsy and Alcoholic paraplegia. Diphtheritic paralysis was treated of in the chapter on the disease of which it is a sequel; lead-palsy will find its place with other atrophic forms of paralysis, and alcoholic paraplegia in the following chapter.

Moreover, progressive neuritis of extensive tracts of peripheral nerves has been clearly ascertained to occur not only as a concomitant or secondary condition, but also as an idiopathic affection involving many nerves, usually in a symmetrical manner, and having its definite course, associated symptoms, and results; in fact, the characters which constitute a "disease" as distinct from a pathological process.

History.—Chomel appears to have first described the clinical features of this affection, which occurred epidemically in Paris in the spring of 1828, and his account was corroborated by Graves, who saw it there in the summer following. The great Irish physician describes it as beginning with pricking and severe pain in the hands and feet, followed by excessive tenderness to the touch. This was succeeded by anæsthesia, and finally by loss of motor power. It thence spread up the arms and legs, but after the patients had lain helpless for weeks they in most cases gradually recovered.

Duménil described a case of the disease in 1864. The clinical symptoms leave little doubt of its nature, and the microscopical examination of the nerves (but not of the cord) by Pouchet confirms that opinion.*

Dr Buzzard described in the 'Clinical Transactions' for 1874 a case of bilateral facial and general paralysis, which recovered under anti-syphilitic treatment, and which he has since regarded as due to multiple peripheral neuritis; but there was no increased excitability of the paralysed muscles to interrupted galvanism, and of course no anatomical proof of the nature of the lesion. There is no mention of the disease as now recognised in Erb's article in 'Ziemssen's Handbuch' (Bd. xii, 2te Auflage, 1876). But in 'Virchow's Archiv' for 1877 (vol. lxix, p. 265), Eichhorst described, under the title "Neuritis acuta progressiva," a remarkable case occurring in a woman of sixty-six, with successive invasion of the nerves of the legs and arms, accompanied by pyrexia. She was suffering from chronic Bright's

* 'Gazette Hebdomadaire,' 1864, p. 203. The same writer contributed a monograph on the subject, with several other cases, to the same Gazette in 1866 (pp. 51—84).

disease. The nerves were examined after death, and were found in a state of neuritis (pl. viii).

Professor Grainger Stewart published three cases of the same affection in the 'Edinburgh Medical Journal' for April, 1881, under the heading "Paralysis of the Hands and Feet from Disease of the Nerves." There is an excellent account of the disease under the title "Progressive Multiple Neuritis" in Dr Ross's treatise (vol. i, p. 354), and Dr Buzzard has published an interesting monograph in his lectures on "Paralysis from Peripheral Neuritis" (1886). He shows the probability that many cases of alcoholic paralysis which recover belong to this pathological group, especially those described by Dr Wilks in his well-known 'Lectures on Diseases of the Nervous System' (p. 272).*

Course and symptoms.—Multiple symmetrical neuritis (called peripheral to exclude affections of the nervous fibres which run in the brain and cord, and of the roots of the vertebral nerves) usually begins somewhat abruptly, and runs a rapidly ingravescent course, though it is less acute than infantile paralysis. Pyrexia is sometimes present. Pain in the course of the nerves affected is quickly followed by more or less complete loss of power in the muscles they supply, and this paresis is accompanied by tingling, numbness, and loss of tactile sensibility, and is frequently followed by trophic disorders of the skin, particularly vesicles and bullæ.

The pain is that of neuralgia, often the peculiar burning pain described as *causalgia* (p. 404). The *anæsthesia* extends over broad patches of skin, sometimes independently of a particular nerve-trunk, and the affected regions are frequently bordered by a zone of hyperæsthesia. The *loss of power* is variable in degree, from the slightest paresis to complete akinesia.

The distribution is symmetrical, and affects the extremities or the face rather than the trunk; and the hands or feet, or both, rather than the proximal parts. The legs are most often affected of all parts of the body, and the extensors of the foot suffer most, so that the patient has a characteristic gait, in which the toe drops and catches the ground.

Faradic contractility is lost, as was noted by Duménil in his first case, and most often, as we should expect, the galvanic contractility is increased, with the other characters of R.D. (p. 400). But Dr Buzzard has found this not to be a constant feature, and it was wanting in a case of peripheral neuritis which recovered under the writer's care.

The *prognosis* is generally good; the most typical cases appear to become rapidly worse, then to remain at a standstill, and then slowly to recover. But in practice our prognosis depends greatly on the clinical features of the case, and especially on its origin.

Diagnosis.—The distinction from neuralgia is not a real one, *i.e.* the pains *are* neuralgic; but anæsthesia (often present to a slight degree in neuralgia) is here nearly absolute, and there is motor paralysis as well. The course and clinical features are different from those of mere neuralgia, and probably the histological condition of the affected nerves is different also. Its symmetry distinguishes it from ordinary hemiplegia and from crossed hemiplegia; while lesions of the pons which affect both sides of the trunk and both sides of the face, without producing rapid death, have rather a theoretical than a practical existence. More often there is difficulty in

* Other cases of paralysis due to alcohol, *e.g.* those recorded by Dr Broadbent ('Med.-Chir. Trans.,' vol. lxvii, 1884), are clearly different in clinical, and probably also in pathological character.

distinguishing multiple neuritis from symmetrical affections of the cord and its membranes, particularly spinal meningitis. Some cases of cervical pachymeningitis, in particular, closely resemble peripheral neuritis. So also do certain central lesions which run an acute course, like Landry's ascending paralysis, and some forms of essential spinal paralysis in adults.

The absence of implication of the bladder and rectum, and of bedsores, distinguish it from destructive transverse lesions of the cord. Hysterical paraplegia does not affect the hands, and is not limited to the feet; while hysterical anæsthesia is unilateral and without akinesia.

The severe pains, along with disturbance of sensation and voluntary movement, are the symptoms which most resemble spinal meningitis. Loss of power in the extensors of the hands ("dropped wrist"), with deep-seated tenderness, resemble the ordinary symptoms of lead palsy, but it is probable that this is itself due to neuritis.

Apart from the characteristic distribution, and the relation to definite nervous tracts, the most distinctive symptom is deep-seated tenderness on pressure, and particularly tenderness of the affected nerve-trunks. The usually favourable course, and the absence of atrophy or rigidity in the affected muscles, are additional helps to diagnosis.

In some cases there are striking exceptions to the usual symptoms. Thus the R.D. may be absent or imperfect; there may be some loss of control over the bladder, as in a case of the writer's above noticed; and there may be, as Leyden has recorded, paraplegic bedsores. Atrophy, commonly slight, may be well marked, or rigidity of the affected muscles may supervene. Probably in these cases the lesion is not confined to the peripheral nerves. Loss of knee-jerk is very common, particularly in alcoholic cases, and with the pains and unsteady gait has led to the diagnosis of tabes. The gait, however, differs from that of locomotor ataxia, as well as from that of cerebellar disease, and is very characteristic of one of the forms of alcoholic paraplegia. If we call the tabid gait "unsteady" or "staggering," and the cerebellar "reeling," we might apply the term "halting" to the dragging of the feet described above.

Histology.—The inflammation affecting the nerve-fibres leads to "breaking up of the axis-cylinder" (Stewart), and "sclerotic atrophy" of the nerves with loss of myelin and thickened perineurium (Leyden).* To the naked eye the affected nerves appear normal, and in the typical cases no secondary changes are found in the nerve-roots or in the cord. By the administration of lead to guinea-pigs, Gombault (quoted by Ross) produced similar changes, not continuous but in segments of the nerves.

Ætiology.—While some of the best-marked cases of peripheral neuritis are apparently idiopathic, others are certainly due to *alcohol*, others are decidedly *gouty*, others are caused by *lead*, and others again are *sequelæ* of enteric fever and other acute diseases, particularly *diphtheria*. The cases which recover cannot, of course, be tested by anatomical investigation; but, judging by clinical features, most cases of lead palsy, certain cases of alcoholic, and certain cases even of *syphilitic* paralysis may be grouped with those described by Chomel and Graves, Eichhorst, Leyden, Stewart, and Buzzard, while diphtheritic paralysis seems always to be of this nature. Some pathologists believe that the enigma offered by the so-called "ascending paralysis of Landry" will be solved by the discovery of its seat in the peripheral nerves.

* See plates 3, 4 and 5 in Mr Bowlby's 'Injuries and Diseases of Nerves.'

At present, however, it is well to leave the best marked of these in their ætiological relations, and still to speak of alcoholic, syphilitic, plumbic, and diphtheritic paralysis.

Of the cases clinically conforming to the character above stated which have fallen under the writer's observation, one was apparently idiopathic, and occurred in an elderly man with great acuteness and severity. He gradually and slowly recovered. Another, of decided alcoholic origin, in a powerful man about thirty, proved fatal after a short course, and the brain and cord were found free from disease. A third, more certainly genuine, occurred soon after an attack of gout, and subsided without local treatment. It was confined to the hands and feet, and showed all the features above described. A fourth was also in a gouty patient.

Treatment.—In gouty, plumbic, and syphilitic cases the appropriate treatment is that which is directed to their respective causes. For patients of intemperate habits, entire abstinence from liquor and assiduous feeding will often succeed against expectation. In "idiopathic" cases no therapeutical measures are at present generally accepted, and it is most rational to treat them, not "on general principles," but according to the "indications of the disease" as successive symptoms demand relief. One plan, however, seems to be almost always beneficial, namely, the application of slowly interrupted galvanic currents.

Neuritis in Beri-beri.—It is worthy of mention, before leaving this subject, that some of the symptoms of a disease endemic in Japan, and known as *Kakké* (*i. e.* weak legs), are remarkably like those of symmetrical peripheral neuritis. It is sometimes epidemic, like the cases described by Chomel, and it is said to be contagious. It is in many cases associated with dropsy, affects young adults especially, and is very apt to recur. In India, including Ceylon, and in Malacca the same malady has long been known under the name of *Beri-beri*. It is also endemic in Brazil. See Mr Wm. Anderson's "Lectures on Japanese Beri-beri," 1879; Morehead ('Br. and For. Med.-Chir. Rev.,' 1855); Fayrer ('Med. Times and Gaz.,' 1880), and Vilette ('Brit. Med. Journ.,' April 23rd, 1887), and a paper in the 'London Medical Record' for January, 1889, p. 13. In Dr Buzzard's second lecture (p. 64), Harada ("Die Japanische Kakké," 1882), and Palm ('Edin. Clin. and Path. Journ.,' September, 1884) are also quoted.

AFFECTIONS OF THE SPINAL CORD

PARAPLEGIA

Παραπληγίη δὲ πάρεσις ἀφῆς καὶ κινήσιος, ἀλλὰ μίρεος, ἢ χειρὸς ἢ σκέλεος.—ARETÆUS.

Arrangement of diseases of the cord proposed.
Symptoms of paraplegia—Localisation—Sensory symptoms—Physiological interpretation of symptoms—Incomplete paraplegia—Reflex movements—Knee-jerk and clonus—Muscular atrophy and rigidity—The bladder and urine in paraplegia—The rectum and genitalia—Bedsores.
INTRINSIC PARAPLEGIA—*Myelitis—Pathology and histology of acute myelitis—Of chronic myelitis or sclerosis—Ætiology—Course and symptoms of acute myelitis—Prognosis—Diagnosis—Treatment.*
Paraplegia due to Hæmorrhage and Tumours within the cord—Anæmia and Congestion of the cord, Divers' palsy—Concussion of the cord.
Acute Ascending paralysis of Landry—Alcoholic and Syphilitic paraplegia.
Reflex and Hysterical paraplegia—Neurasthenia spinalis—Rachialgia.
HEMIPARAPLEGIA—*Its pathology and symptoms, origin and course.*
EXTRINSIC PARAPLEGIA—*Compression of the cord from vertebral caries or cancer—from aneurysm, hydatid cyst, or meningeal tumour—Symptoms, Diagnosis, Prognosis, and Treatment of paraplegia from compression.*
SPASTIC PARAPLEGIA—*in adults—in children—Primary and secondary—Anatomy—Mixed forms—Symptoms—Prognosis and Treatment—Cases.*
MENINGEAL AFFECTIONS—*Hæmorrhage—Acute and chronic spinal meningitis.*

OUR knowledge of affections of the spinal cord has of late made great progress; positive diagnosis can now be made of lesions which were unknown to the pathologists of the last generation. Unfortunately, however, the advance has been mainly with respect to diseases comparatively rare. The diagnosis of the more common forms of paraplegia still remains a matter of presumption rather than of certainty, for it is based mainly upon our knowledge that under given circumstances one kind of disease is more frequently met with than another. No doubt the same interpretation of symptoms by probability often guides our diagnosis of diseases of the brain or the abdomen; but compared with the spinal cord there is no other region in which the results of an autopsy are so often unexpected.

Classification.—The arrangement of diseases of the cord is not easy. Very few can be grouped on an ætiological system, since their origin is often most obscure. The pathological processes are comparatively few, and vary

greatly in the symptoms they produce according to their exact seat. Anatomical lesions are peculiarly difficult to determine, from the frequent slowness of their course, from the difficulties of histological examination, and from the rarity of some of the most obscure. Moreover, certain affections of the cord have, so far as the most skilled observers working with modern methods can determine, no anatomy at all. A clinical classification, is therefore, in the present state of our knowledge, most practicable; but the distinction between functional and organic affections, the local distribution of chronic myelitis, and such ætiological facts as bear upon treatment or prognosis must also be regarded.

The plan here adopted will be to describe, first, the most frequent and obvious symptom produced by lesions of the cord, namely, Paraplegia. We will then discuss the peculiarities of paraplegia produced by Myelitis, or by other causes operating *within the cord.*

Next will follow an account of paraplegia when it is the result of direct compression of the cord *from outside.*

We shall next consider a form of paraplegia in which the paralysed muscles become affected with *spasm;* it is separated by other clinical features and by the anatomical lesion which usually accompanies them.

An account of the symptoms produced by affections of the spinal *meninges* will complete the present chapter.

The *atrophic* forms of spinal paralysis—some paraplegic, but others not—are conveniently grouped together, and will form the subject of the next chapter.

Most of these various spinal paralyses are defined by the anatomical lesions to which they may be traced; but there remain two remarkable nervous disorders, which also (while associated with definite changes in the cord) have far more constant and characteristic clinical features than the preceding affections, so that each may be regarded as a natural and connected series of events. These two "diseases"—Tabes and Insular Sclerosis—complete the list of spinal maladies, and will occupy the third and fourth chapters of the present section. The latter, being cerebral as well as spinal in its anatomy, forms a transition to the next section on Diseases of the Brain.

PARAPLEGIA.—This is a form of paralysis possessing two essential features: first, that it affects both sides of the body, generally to an equal extent and in the same degree; and secondly, that it affects all parts, up to a certain level, according to the functions of the several pairs of spinal nerves, ascending from the sacral to the cervical.* It is easy to see why affections of the cord should produce this combination of symptoms. First, the motor tracts belonging to the two halves of the body are so close to one another in the cord that they are generally involved in the same lesion; secondly, since each segment† of the cord contains fibres belonging to all the nerves below, they are all likely to suffer together.

* This is the modern meaning of the word, dating from the last century. Thus, Cullen defines "Paraplegia" as *paralysis dimidii corporis transversim sumpti.* But Hippocrates and Galen applied the word παραπληγία or παραπληξία (for both forms are used) to paralysis of a single limb, and distinguished it from apoplexy as a "side-stroke," *i. e.* a partial palsy only. See the quotation from Aretæus on chronic diseases printed at the top of this chapter (Lib. i, cap. vii). In 1703 paraplegia is defined as *paralysis quæ omnes corporis partes capiti subjectas occupat* (Blancardi, 'Lexicon Medicum').

† A "segment" of the cord is included between any two adjacent horizontal sections.

Although Sir William Gull and other writers have spoken of a "cervical paraplegia," when the arms are paralysed without the legs, the phrase is not strictly accurate. In such cases the affection, if it lies within the cord at all, must be definitely limited to a part only of the segmental area; whereas the morbid changes which cause true paraplegia acknowledge no such limitations. They affect one or more segments in their entire width, sometimes by gradual extension, more often at once, and always at last.

Locality.—As regards their distribution in the length of the cord, the lesions in question vary widely. They may be strictly confined to a single spot, or may extend from the cauda equina to the cervical region, or may transcend the limits of the cord and invade the bulb.

The *upper limit* of a spinal affection is, as a rule, roughly indicated by the extent upwards of the motor paralysis to which it gives rise. Disease of the lumbar enlargement causes paralysis of the lower limbs; disease in the dorsal region causes in addition paralysis of the abdomen and chest, corresponding with the level to which it reaches; disease of the cervical enlargement adds paralysis of the upper limbs; and disease still higher up produces paralysis of the diaphragm and of the cervical muscles in addition to that of all the parts below, and so soon as developed is of necessity fatal.

The *lower limit* of a spinal affection is more difficult to fix, since a complete transverse lesion of the cord will, however short, paralyse all the fibres below. Inasmuch, however, as the cord is a collection of centres as well as of conducting tracts, we can to some extent define the lower limits by a study of its reflex functions (*infra*, p. 433).

In some cases the feet and legs become powerless before the thighs and the hips. This is generally supposed to mean that the lesion is at first confined to the extreme lower end of the lumbar enlargement; but, since Woroschiloff found that in the lateral columns of the lumbar cord of the rabbit the motor paths for the distal parts of the lower limbs lie outside those for the proximal parts, it has been thought that the symptom in question may sometimes be due to gradual extension of disease from the surface inwards.

When paralysis attacks the upper extremities in detail, as the result of disease spreading upwards through the cord, the muscles of the hands are as a rule affected before those of the elbow and the shoulder. The fact

It takes the *whole thickness* of the cord, and a single pair of nerves. We thus recognise eight cervical, twelve dorsal, and a number of lumbo-sacral segments, although the last are too closely packed together for us to be able always to distinguish their several lesions.

It must be carefully borne in mind that while each segment generally resembles the rest, there are important differences between them: first, in the total size and shape; secondly, in the proportion of grey to white matter, and the size of the anterior cornua; thirdly, in the development of the several "tracts" of the cord.

Thus, as we pass downwards, the crossed pyramidal tract becomes smaller and more superficial; the direct cerebellar and direct pyramidal tracts gradually disappear; the postero-median column of Goll becomes smaller and deeper. The anterior cornua are very large in the cervical and lumbar enlargements, very small in the dorsal region; while the important group of cells known as Clarke's vesicular column is well represented in the dorsal (especially the lower dorsal) region, and is almost absent in the cervical and lumbar.

It must be remembered that the lumbar or lumbo-sacral enlargement is so named from its giving origin to the nerves of the lumbar and sacral plexuses, but that it does not, like the cervical and dorsal regions, correspond to the vertebræ of the same name. The cord ends, in the adult, at the first lumbar vertebra, and from the mid-cervical region the nerves pass downwards to their foramina of exit from the vertebral canal, with increasing obliquity, until they are collected together to form the cauda equina.

that the nerves to the ulnar side of the hand come chiefly from the lowest part of the brachial plexus would lead one to expect that the inner fingers would suffer earlier than the outer fingers or the thumb.*

Sensory symptoms.—When any segment of the cord is completely destroyed, there is of course an absolute loss of *sensation*, as well as of motion, in all parts of the body below the lesion. And when a total disorganisation spreads upwards through its substance, the gradual progress of the disease from day to day can be determined from the extension of the anæsthesia with far greater accuracy than is possible from that of the motor paralysis alone. But in the great majority of cases, parts affected with paraplegia either retain perfect sensation, or have that function but little impaired.

This preponderance of loss of motion over loss of sensation is not only met with in all diffused spinal affections, but belongs to them in common with diseases of the peripheral nerves and of the brain itself. And the true explanation is, no doubt, that which was given when the diseases of the nerves were under consideration; namely, that sensory impulses are transmitted far more easily than motor ones, so that there is no anæsthesia unless the conducting fibres are completely disorganised.

Beside the abolition of the sense of touch in completely paralysed limbs, there is loss of the sense of temperature and of the muscular sense and absence of pain (*analgesia*). Many paraplegic patients who retain the sense of touch complain of "pins and needles," "pricking" or "tingling" in the toes or in paralysed parts, "creeping" or "crawling" (*formicatio*), or subjective sense of heat or of cold—perverted sensations which are conveniently called *paræsthesiæ*.

Physiological explanation of symptoms.—There are certain affections which are strictly limited to particular tracts of the grey or of the white matter; but we shall find that none of these are characterised by purely motor or sensory paralysis (*akinesia* or *anæsthesia* respectively) of the parts below. The lesions which are diffused over the whole segmental area of the cord commonly cause more loss of motion than of sensation.

All physiologists are agreed that volitional motor impulses pass mainly along the crossed pyramidal tracts of the antero-lateral columns. With regard to the paths for sensory impulses, there still is doubt. One opinion, maintained by Brown-Séquard, was that they are all situated in the grey matter. Another, which was first taught by Schiff, is that tactile sensations are transmitted through the posterior columns, but sensations of pain through the grey matter. A third and better supported hypothesis places the chief sensory tracts in the deeper part of the lateral columns of the opposite side.

We shall find that a central tubercular nodule is occasionally the cause of paraplegia. But anæsthesia does not precede motor paralysis in cases of this kind, as on Brown-Séquard's theory it should. Again, if Schiff's doctrine were correct, such an affection should cause analgesia, tactile sensation being retained; but as yet nothing of the kind seems to have been noticed. It may be replied that a central tubercle of the cord does not completely disorganise the grey matter before it presses

* According to Erb ('Ziemssen's Handbuch,' xi, 2, p. 62) paralysis of all four limbs, and of the trunk muscles generally, sometimes occurs without any interference with the respiratory movements, as the result of an exceptional limitation of disease to a part of the segmental area of the cord in the cervical region, leaving the lateral columns intact. Strictly speaking, such an affection is not paraplegic in the sense defined above.

on the white columns; but this is the very difficulty: the diseases which cause paraplegia are not so localised as to admit of the application of rules deduced from experiment. One physiological result, however, is of great clinical importance, namely, that the sensory fibres do not cross at the decussation of the pyramids in the bulb, but pass over to the opposite side, at once or gradually, at no great distance from the posterior roots.

Partial paraplegia.—Hitherto we have referred to complete motor paraplegia, a condition in which the affected muscles are altogether incapable of being influenced by the will; but in a large number of cases the paralysis is incomplete. Every degree of loss of power may be noticed. One patient can just move his toes, another can flex the knees so long as they lie horizontally on the bed; a third can draw the thighs up to the abdomen; a fourth can stand for an instant and then falls down; a fifth can walk a few paces, and so on, up to a condition in which all that can be detected on the most careful examination is a slight uncertainty of step, with a tendency to catch one of the feet against any slight obstacle. When "paresis" (as partial mator paralysis is called) is more marked the gait is peculiar, and it is well worthy of study because it contrasts with that which is observed in some other spinal affections that will be discussed further on. Erb describes it in the following terms:—"The foot hangs down in walking, the toes are dragged, the sole is brought to the ground clumsily, and for the most part upon its outer edge; the knee is too much raised or carried forwards without being bent; there is generally a kind of stiffness about the legs. The patient uses one or even two sticks, or he is held up by crutches or by the arms of other persons; he totters but slightly, and stands quietly and steadily. When left unsupported he sinks down upon the ground. The gait of course varies somewhat according to the number of muscles which are paralysed. When only those below the knees are affected it is waddling and very peculiar."

In those cases of partial paraplegia in which sensation is also impaired the patient feels when he stands upon his feet as though they were wrapped in wadding or covered with thick woollen stockings; or a hard floor may seem to him like a thick felt carpet. In such cases he requires the guidance of vision to enable him to stand firmly or to walk evenly. When a patient who has his feet close together totters or falls as soon as he is made to close his eyes, he is often assumed to be suffering from a special disease of the cord—sclerosis of the posterior columns (tabes or locomotor ataxy). But although the symptom in question is present in that disease, it is yet by no means peculiar to it, being met with whenever there is an imperfect transmission of those sensory impulses from the feet by which the equilibrium of the body in the erect posture is normally maintained.

Reflex symptoms.—The *lower limit* of a lesion of the cord cannot be determined with any great accuracy. We have seen that disease in the cervical region renders the legs as well as the arms paraplegic, and this equally whether the affection is confined to a small part of the length of the cord or involves the whole of it down to the cauda equina. An easy experiment, however, enables us in many cases to say that at least some of the lower segments retain their functions, although their connection with the brain is cut off. We have but to see whether *reflex movements* can be excited in the legs by impressions upon the cutaneous nerves. If this experiment succeeds, it proves that the lumbar enlargement is still undestroyed. On the other

hand, its failure is not to be taken as conclusive evidence that the disease extends throughout the whole of the lower part of the cord. On the contrary, it often happens in cases of very limited lesions that reflex movements can only now and then be elicited. For example, surgeons find, when there is fracture with displacement of the upper dorsal or cervical vertebræ, that for a few days after the accident there is an entire absence of reflex excitability in the cord; it seems to have been in some way deprived of its functions by the shock.* So, also, we shall hereafter learn that the reflex movements are generally less readily obtained when disease has begun in the interior of the cord than when it is merely compressed from without; the reason being that in the former case an "inhibitory" influence is transmitted downwards upon the lower centres. Even when these retain or have regained their susceptibility, it may sometimes be easily exhausted for a while by repeated stimulation. In a well-known paper, read before the Royal Medical and Chirurgical Society in 1839, Dr William Budd mentions the case of a young lady in whom the slightest disturbance of the bedclothes caused very energetic contractions of the right leg, which was paralysed; but there was a successive diminution in the vigour of the spasms on each renewal of the stimulus. The impressions that produce reflex movements are sometimes felt, and in the case of this young lady the movements themselves were attended with pain like that of cramp. On the other hand, Dr Budd relates an instance in which all the limbs were paralysed by an injury to the cord in the cervical region, but in which convulsions in the arms were excited by pulling the hair of the head or by tickling the chin; the efferent impulses must then have traversed the seat of lesion, through which no volitional efforts could be made to pass. As a rule, however, cases in which all communication with the brain is absolutely cut off are those in which the reflex activity of the cord is most marked; the patient then has no consciousness either of the application of the stimulus or of its effect, unless he sees the jerking of his legs. It seems probable that the susceptibility of the lower spinal centres is often directly augmented by an irritant influence transmitted downwards from the diseased part; for the administration of strychnia excites spasms in paralysed limbs much earlier than in those which remain obedient to the will. Sensitive impressions upon the skin of the soles of the feet seem to set up reflex movements in the legs much more easily than simular stimuli applied to any other part of the cutaneous surface; lightly touching the hollow of the foot with a feather often throws the whole limb into spasm. Again, a hot metal plate produces much more marked effects than one at an ordinary temperature, even when the transmission of sensory impressions is so imperfect that the patient cannot tell the difference. But the most violent convulsions of all are those which are sometimes excited by micturition and by defæcation. One of Dr Budd's patients was obliged, whenever he was placed upon the night-stool, to have his feet inserted into two large loops of saddlers' webbing which were nailed to the floor, to have two larger loops adjusted over his knees, and to hold down his thighs; otherwise he was liable to be thrown forwards upon the ground.

Reflex movements are more or less co-ordinated. There seems no reason to suppose that muscles are individually represented in the spinal centres. And whatever may be the machinery in the brain by which the

* See the instructive remarks of Prof. Goltz on this subject in his 'Verrichtungen des Grosshirns,' S. 78—81.

several groups of muscles are harmonised and co-ordinated in their actions so as to produce definite movements, it is certain that cross-connections exist within the cord by means of which a similar result can be brought about. We do not see such marked purposive reflex movements in a paraplegic patient as are observed in decapitated frogs; but a near approach to them was seen in a case of Dr McDonnell's, recorded in the 'Dublin Quart. Journ.' for 1871. The patient was paralysed in all four limbs; when a catheter was passed into his bladder his left arm would "wriggle up and down, and the hand come fluttering over the genital organs, although he was absolutely unconscious of the operation, and even when his eyes were shut." Another curious case is related by Virchow. It is that of a man who was paraplegic, and who generally lay in bed with his lower limbs rigidly flexed. When he wanted to stand upright he would pinch the right thigh sharply, or give it a smart blow on its outer surface; this threw both the legs into a position of extension; still the gastrocnemii remained contracted, and before he could put his foot to the ground he had to bring down the heels with both hands ('Gesammelte Abhandlungen,' p. 684).

A curious illustration of the way in which one set of reflex actions may be made to conteract another set is afforded by a case of Dr Budd's. The patient was recovering from paraplegia and could walk a few steps, until the contact of the soles of his feet with the ground would make his knees bend beneath him; this he was able to overcome by rubbing the surface of his belly, when the legs were extended with a jerk.

As a rule, when reflex movements are excited by impressions upon the cutaneous surface, they remain confined to the limb on the same side. Leyden says that they do not spread to the opposite limb except when the lesion is in the bulb, but many instances to the contrary may be found. The movements generally consist in a rapid series of intermittent (*clonic*) spasms; but sometimes the contraction is continuous (or *tonic*) in character.

Beside the well-known reflex movements called *plantar* and *cremasteric*, there are several others of diagnostic value as localising symptoms in cases of paraplegia.

(1) Some are obtained by touching, pinching, or stroking the skin so as to produce contraction of the underlying muscles. The following are the most important of these superficial reflexes:—The *abdominal*, when the external oblique muscle contracts; the *epigastric*, when tickling the skin over the fourth to the sixth ribs produces contraction of the upper segment of the rectus; the *gluteal*, when irritation of the skin of the buttock leads to dimpling over the insertion of the glutæus maximus into the fascia lata; and the *scapular*, a slight movement of the teres major in the fold of the axilla when the skin between the shoulders is pinched. All these are most easily produced in children; and others may be discovered at an early age, as the *palmar* reflex or closure of the fingers produced by gently touching the palm of a sleeping infant. In elderly people several are absent, and all are less readily elicited.

As criteria of the seat of a spinal lesion:—the *plantar* reflex corresponds to the sciatic nerve and lower part of the lumbar enlargement of the cord, situated in the adult opposite the last dorsal spinous process; the *gluteal* reflex corresponds to the fourth and fifth posterior lumbar branches, the lumbar enlargement, and inferior gluteal nerve; the *cremasteric* to the first and second anterior lumbar branches; the *abdominal* to the lower three or four intercostal nerves and dorsal cord; the *epigastric* to the middle intercostals

(fifth to seventh), and the upper dorsal cord; and the *scapular* to the upper bundle of the brachial plexus, and the cervical enlargement of the cord.*

(2) There are what may be termed Visceral Reflexes:—*vesical*, when the bladder is caused to contract by irritation of the urethral orifice, of which the incontinence of urine in children, by reason of a long prepuce, is an example; *penile*, when erection is produced by the passage of a catheter or by the slightest contact, a condition sometimes seen in cases of partial paraplegia, short of that which produces priapism; *rectal*, as when contraction of the bowel follows the introduction of a suppository; lastly, the familiar reflex actions of *vomiting* from tickling the fauces, *sneezing* from a draught of cold air or a brilliant light, *blinking* from touching the cornea, and contraction of the *pupil* to light and during accommodation for a near object.

The movement of the ribs in *respiration* enables us to judge of the integrity of the connection of the intercostal nerves with the brain, or of the point where it is severed by transverse lesion of the cord. The act of *swallowing* depends on the centre situated in the bulb, and that of *contraction of the iris* on the centrum ciliospinale in the same region.

(3) A closely connected set of phenomena have been called *deep reflexes*, but it is still doubtful whether they are due to reflex or direct stimulation of the muscles involved.† Dr Gowers suggests "*myotatic* movements" as a safe synonym.

The most important was first described in 1875, by Westphal and Erb independently; it was called by the latter the "patellar tendon-reflex," by the former the "knee-phenomenon." The best English name is *knee-jerk*, and it has long been known to schoolboys. A smart tap is given with the side of the hand upon the tendon of the great quadriceps extensor muscle of the leg, above or below the patella, while the knee is crossed over the opposite thigh; and the foot is jerked upwards. A similar *elbow-jerk* may be obtained in most healthy persons by striking the stretched tendon of insertion of the triceps extensor brachii.

The absence of the knee-jerk is usually, perhaps always, a morbid sign; but different persons vary much in the readiness with which it is procured. It is exaggerated in paraplegia, but, contrary to what we should expect, is as a rule diminished in the paralysed leg of hemiplegia. In morbidly excitable conditions the jerk may sometimes be obtained by stretching the ligamentum patellæ and striking the subcutaneous surface of the tibia; or, by stretching the tendo Achillis and tapping the tibialis anticus and extensors of the toes, an extension of the foot may be obtained (Gowers' front-tap contraction).

The so-called deep reflexes do not always increase or diminish in correspondence with the superficial reflexes.

Ankle-clonus is the name given to the clonic contractions produced in some patients by forcibly bending up the foot so as to make the tendo Achillis tense. It was first described by Charcot, and is included by Erb and Westphal among tendon-reflexes. It usually goes with exaggerated knee-jerk, and is probably always a morbid phenomenon. But a somewhat

* See Dr Gowers' useful diagram ('Diagnosis of Disease of the Spinal Cord,' p. 58).

† See papers by Tschirjew ('Arch. f. Psychiatrie,' 1877, and Du Bois's 'Arch.,' 1879), Gowers ('Lancet,' i, p. 156, 1876), Waller ('Brain,' July, 1880), and De Watteville ('Brit. Med. Journ.,' 1882, p. 736); also by Bowditch and Warren ('Journ. of Physiology,' vol. xi, 1890).

different kind of clonic spasm of the gastrocnemius can be obtained in most healthy persons by bending the knee and ankle with the ball of the foot resting on the ground, and moving the knee rapidly up and down.

The paralysed muscles.—The condition of the centres in the cord has a direct influence on the nutrition of the paralysed muscles.

We have seen that when disease destroys the entire substance of the cord up to a certain level no reflex movements of any kind can be obtained in the limbs below. Under these circumstances the muscles rapidly lose their electrical contractility, and they undergo marked *atrophy*. Before complete loss of susceptibility to galvanic currents supervenes, there is generally a period in which the reaction of degeneration is present (cf. p. 400). On the other hand, when paraplegia is due to a lesion limited to one of the upper segments of the cord, so that reflex movements persist in the paralysed limbs, the muscles, as a rule, retain their normal electrical reactions, and they remain tolerably well nourished for months and years, or show merely such slight flaccidity and wasting as may fairly be attributed to disuse. They are still in connection with their trophic centres in the anterior cornua.

Again, paraplegic muscles may become *rigid*, so that the legs are either forcibly extended, or drawn up in a state of flexion. Cases of this kind will be separately described hereafter, under the name of "spastic paraplegia." The knee-jerk is almost always exaggerated and clonus is present.

The bladder in paraplegia.—Unstriated as well as striated muscles can be excited to reflex contractions. Erb mentions a curious case of paraplegia, in which a fluid fæcal evacuation was passed whenever a large bedsore was dressed. In other instances micturition was directly produced by pressure over the bladder, or by the introduction of the finger into the rectum. It does not follow that impulses were reflected to the vesical muscular fibres after being conveyed to the cord by cutaneous nerves, for the slightest compression of any part of the distended bladder appears to be a sufficient direct stimulus to its muscular fibres. Indeed, in the apparently volitional act of micturition in health the influence of the will is really limited to relaxing the sphincter and throwing the abdominal muscles into contraction, so as to press upon the bladder and set in action its reflex machinery. The experiments of Goltz upon young dogs ('Pflüger's Archiv,' 1874) have shown that in these animals the bladder is capable of emptying itself at regular intervals, and in a perfectly normal manner, after the cord has been completely cut across by a sharp knife.

Thus physiology would lead us to expect that micturition would take place naturally in those cases of paraplegia in which the lesion is limited to the upper part of the cord. In practice, however, it is found that retention of urine almost invariably occurs, even when reflex movements can be easily excited in the lower limbs, proving that the lumbar centres are intact. The explanation doubtless is that an inhibitory influence is transmitted downwards upon those centres from the diseased part above. Goltz, in fact, states that in some of his experiments it was not until several days after the operation of section of the cord that the animal was able to micturate properly; and he suggests that in cases of injury to the spine in man the bladder would after a time be found to have regained its functions if catheterism were not continued as a matter of routine. However this may be, it is certain that the theoretical accuracy of Goltz's views with regard to micturition is established by some exceptional cases of paraplegia, in which the urine is passed quite naturally. It is remarkable how many of the patients

whose cases are related by Dr Budd in his well-known paper were able to micturate, though not always in a perfectly normal manner. In the case already cited as presenting such violent convulsive movements of the legs (p. 432) the urine at first began to flow in interrupted jets, but as the spasms grew more feeble its stream became continuous. Another patient, at a certain period in his illness, emptied his bladder involuntarily and with a sort of jerk about every two hours. In a third case, one of fracture of the spine, in which there was at first retention, the power of voiding urine naturally was regained on the fifth day, whereas no volitional movements in the legs occurred until the ninth day. So, again, one of the cases related by Ollivier, in his classical work,* is that of a man who had complete paralysis of his lower limbs as the result of caries of some of the higher dorsal vertebræ, but in whom the bladder performed its functions normally.

It is an interesting question whether the converse can occur—that is, whether the lumbar centre for micturition is ever the only part of the cord diseased, so that while the bladder is paralysed the lower limbs nevertheless retain the power of movement. It seems probable that such a condition exists, though it is rare.†

In some instances in which the power of micturition is partially impaired one can distinguish paralysis of the *sphincter* from that of the *detrusor urinæ*, by which expulsion of the urine is mainly effected. It might have been supposed that in the former case there would be complete incontinence, the fluid dribbling away incessantly. But physiologists have found that even after destruction of the lumbar cord the neck of the bladder can withstand a pressure of six inches of water—as compared with one of twenty inches when the cord is intact; and this result is entirely confirmed by some observations of Hutchinson ('Brit. Med. Journ.,' 1877) upon persons who have the remains of a shrunken spina bifida, involving the fourth pair of sacral nerves, so that the sphincters of the bladder and rectum alone are permanently paralysed. In such cases he finds that the bladder may be able to hold a large quantity of urine, but if once its reflex contractions are excited the will is, of course, powerless to restrain them, and therefore there is a constant risk of accidents. So in many cases of partial paraplegia the patient says that as soon as he feels the inclination to micturate he is obliged to indulge it, so that the urine is apt to escape before he can get to the chamber vessel. But when the detrusor is paralysed the complaint is that he has to strain for a minute or two before he can pass any water, that it comes away slowly in a feeble stream, and trickles for some little time after voluntary effort has ceased.

On the other hand, when the "micturition centre" in the lower part of the cord is wholly deprived of its functions by disease or injury, the sphincter and the detrusor of the bladder are of course paralysed together. The

* 'Traité de la Moelle Épinière et de ses Maladies,' par C. P. Ollivier: 1st ed., 1821; 3rd, 1837, with plates.

† I have notes of more than one case in which retention of urine has preceded all other indications of the onset of paraplegia by an interval of a few days, but I do not remember any instance in which it has continued to be the sole or even the main symptom of a spinal affection. Some years ago I saw a gentleman who was one of the sufferers in the Thorpe railway collision, and whose chief complaint was that he could pass his water only in a very feeble stream; he said that "it flowed from him like oil," and it quickly became ammoniacal and very fœtid. He had pain and tenderness in the back, but the only sign of any loss of power in the lower limbs was that in walking he sometimes seemed to totter, especially if his foot caught against any slight obstacle; he also had "twitchings" in the legs three or four times a day. He was not long in getting perfectly well —C. H. F.

result is that the organ becomes distended, and that the excess of urine beyond what it can hold dribbles away through the urethra. The Germans call this condition "ischuria paradoxa." It has often been mistaken for incontinence; an unskilled nurse is very apt to suppose that there can be no retention of urine in the case of a patient who is constantly wet, and her statements may mislead a careless practitioner; but one can always avoid such an error by passing one's hand over the lower part of the abdomen. In incontinence of urine from paralysis of the sphincter alone, the bladder keeps firmly contracted and almost perfectly empty, while its walls become greatly hypertrophied. It seems to be still doubtful whether this kind of incontinence always depends upon an exaltation of the reflex activity of an intact lumbar centre, or whether it may occur after that part of the cord has been destroyed, as the result of contractions of the detrusor, which may either be spontaneous or dependent upon minute intrinsic ganglia of the bladder.*

The urine in paraplegia.—We have still to consider certain changes in the urine, and in the organs that secrete and expel it, which are of the highest clinical importance. Sir Benjamin Brodie many years ago pointed out (in the 'Med.-Chir. Trans.' for 1836) that in cases of injury to the spine the urine is often voided alkaline, ammoniacal, and turbid, even as early as the second or third day; it is well known that in most cases of paraplegia due to disease of the cord the same thing occurs, but generally not until a longer time has elapsed. Its explanation is still a matter of doubt. Charcot believes that it is in some way due to a direct action of the nervous centres upon the urinary organs. Other writers regard it as a result of decomposition of the urine, after secretion, while it is stagnant in the cavity of the bladder. This does not mean that only those patients who have complete retention pass urine which is alkaline and fœtid, for there can be no doubt that a very slight impairment of the power of the bladder may prevent it from emptying itself completely. But the difficulty is to understand how the change in the urine is brought about, apart from the access of air and of the *micrococcus ureæ*. Traube, of Berlin, made the important suggestion that an ill-cleansed catheter might start decomposition; and one should always use antiseptic precautions before introducing an instrument into the bladder. But in more than one case where the catheter was never used the urine has become putrid.†

Moreover, although we may admit that decomposed urine is capable of exciting inflammation of the bladder and of the urinary passages, it does not appear that there is always such an interval of time as would be required for the production of this effect. Charcot cites cases in which the urine contained blood as early as the third and the fourth day respectively, and the presence of blood is generally followed in a very few days by that of pus, or of the gelatinous substance which is formed out of pus in a strongly alkaline liquid. It is therefore possible that cystitis is in some cases the primary, and ammoniacal urine the secondary event.

On *post-mortem* examination, in cases of this kind, one finds the most intense cystitis and suppurative nephritis; and, indeed, these affections have most commonly been the direct cause of death. The mucous membrane of

* See a paper by Dr Francis Darwin, 'Quart. Journ. of Micr. Sci.,' 1874, p. 109.

† Dr Goodhart, in the 'Guy's Hosp. Rep.' for 1874, suggests that an almost stagnant column of urine filling the urethra might suffice to enable the *micrococcus ureæ* or other putrefactive microbes to pass into the bladder.

the bladder is ecchymosed or swollen, infiltrated with inflammatory products, lined with a "diphtheritic" layer or with a granular deposit of phosphates, or actually sloughing. Its muscular wall is often free from hypertrophy, but sometimes it is greatly thickened; in one case at Guy's Hospital it measured at least a twelfth of an inch across, although the disease had lasted only four or five weeks. The kidneys are enlarged, intensely congested, and full of suppurating points and streaks; in other words, they present all the characters of an "ascending inflammation."

Rectum and genitalia.—Some other symptoms connected with the pelvic organs may be conveniently mentioned here. One of them belongs to the *rectum*. Although the peristaltic movements of the intestine are not directly dependent on the spinal cord, the sphincter ani is under the control of a lumbar centre. Consequently, as a rule, it becomes paralysed with the bladder; and incontinence of the fæces generally accompanies or follows retention of urine.

Another symptom concerns the male genitalia. *Priapism* is of frequent occurrence in paraplegia. It is one of the results of the reflex activity of the lower part of the cord. Sometimes it is constant; sometimes it only appears when the bladder is distended, or when a catheter is passed. Goltz, in his experiments on paraplegic dogs, traced out carefully the various ways in which this symptom could be induced by irritating the skin of the abdomen, or of the thighs, or the sheath of the penis, and he found that he could easily inhibit it by simultaneously irritating the sciatic nerve, or by pinching the hind foot. The last observation—showing that the centre for erection is very sensitive to disturbing influences—may serve to explain the fact that a temporary loss of sexual power is not infrequently observed in persons who have been in a railway accident, but in whom there are no other symptoms of serious spinal injury.

When the lumbar enlargement of the cord is destroyed, priapism is of course altogether absent.

As regards the female organs, it is worthy of mention that *parturition* may take place naturally in a woman suffering from paraplegia, provided that the lumbar centres remain intact.

Bedsores.—A circumstance which points towards the conclusion advocated by Charcot—that morbid changes in the bladder and urine are at least sometimes directly dependent upon an influence transmitted from the nervous centres—is that their development is commonly coincident with that of gangrene of the skin over the sacrum and the gluteal regions adjacent. An "acute bedsore," in fact, sometimes begins to form as early as the fourth day. Many instances of this kind are quoted by Charcot; but none is more striking than a case recorded by Sir William Gull, of a man who on November 22nd, 1856, was working in the docks, when he felt a sudden pain in the back after lifting some deals; he became paraplegic on the 24th, and on the 26th was admitted into the hospital with a small bedsore already present. It is clear that in such cases the affection cannot be regarded as a mere result of pressure upon the skin, nor of the irritant action of excreta passed without the patient's knowledge; but there seems to be no doubt that each of these conditions is concerned in the production of the more chronic bedsores which do not appear until after the lapse of weeks or months. An acute bedsore may sometimes be made to heal, if carefully attended to; this occurred in the case just quoted, but afterwards another one formed at the same spot, and it rapidly increased in size, so that, when the disease

ended fatally, six weeks from its commencement, the whole of the sacrum was exposed. Indeed, a bedsore may destroy life, either by exhaustion, or by pyæmia, or by extension of putrid inflammation into the spinal canal.

The four symptoms last described—retention of urine, incontinence of fæces, priapism, and acute bedsores—are apt to go together, and may be conveniently grouped together as the "pelvic symptoms" of paraplegia.

The morbid conditions that give rise to paraplegia fall naturally into two groups. The first includes all those affections which have their seat *within the cord itself*. The second is made up of those in which the primary lesion is *outside the cord*, and subjects it to *slow compression*. The division is not exhaustive; but it is one of practical importance, because in the second group there is a characteristic chain of symptoms dependent upon interference with the nerve-roots at the level of the lesion.

MYELITIS.—Among the affections beginning within the spinal cord itself and capable of causing paraplegia, the most important and by far the most frequent is one which is now believed by most pathologists to be a more or less diffused inflammation of its substance, and which they therefore term *myelitis*. This, again, presents many varieties in its symptoms and course, and in the morbid appearances to which it gives rise; but in the main they may be arranged into two classes, according as their progress is *acute* or *chronic*.

*Acute diffuse myelitis.**—It has long been known that in many cases of paraplegia the cord is found after death to be more or less extensively softened; and from the time of Ollivier the opinion has been maintained by most pathologists that the morbid process in question is of an inflammatory nature. There are, indeed, obvious objections to such a view. It involves the admission that a remarkable contrast exists between softening of the cord and a similar change in the brain. The latter (as we shall hereafter find) is most frequently the result of a deficient supply of arterial blood to the affected part of the cerebral substance; and when it is inflammatory it is almost always traceable to some definite local irritation. The former, on the other hand, can very seldom be shown to be secondary to interruption of the blood supply, and its chief causes are supposed to be functional exhaustion or exposure to cold. A more serious difficulty is that the histological appearances correspond only partially with those which are seen in inflamed tissues generally, and that even the naked-eye changes are not altogether such as are met with in the spinal cords of animals when inflammation has been artificially set up. Leyden, for example, induced myelitis in dogs by injecting solution of arsenic, and obtained greenish patches of purulent infiltration, surrounded by vascular zones; but, in human pathology, the presence of anything that can be recognised as pus in the spinal cord is quite exceptional. On these and other grounds Dr Bastian regards most cases called acute myelitis not as strictly inflammatory, but as a process of softening secondary to thrombosis or other vascular changes.

The late Dr Moxon, in his Croonian Lectures before the College of Physicians, drew attention to the scanty vascular supply of the lower part of the cord owing to the fewness, small calibre, and prolonged course of its

* *Synonyms.*—Acute softening of the cord. Red and yellow softening. Myelomalacia.

arteries, and to its not having with the brain an internal set of vessels in addition to those of its pia mater; and justly argued that this anatomical condition may explain some of the peculiarities of anæmia, thrombosis, and inflammation as they affect the spinal cord.

Anatomy.—The colour of the spinal cord when softened is very variable. Sometimes its tissue is pink or bright *red* from vascular injection; sometimes it is mottled with small ecchymoses; or it may be so saturated with extravasated blood (hæmato-myelitis) that the case seems to be one of spinal hæmorrhage, an affection, however, of which the occurrence is denied by many modern pathologists. Probably all these appearances are confined to an early stage of the inflammatory process; for afterwards the blood undergoes changes which give to the diseased parts a tawny *yellow* tint, due to the presence of hæmatoidin crystals and of fatty degeneration in the form of "compound granule-corpuscles." If no hæmorrhage has occurred the softened spot as a rule is *white*;* or in rare cases of a *greenish* tint from suppuration.

The fact that one part of the spinal cord is softer than the rest, or that it looks diffluent when cut across, is not of itself a proof that disease is present. The softening may result from bruising with the bone forceps during the autopsy though made by the hands of a practised assistant. The microscope here affords valuable assistance. In the great majority of cases one has only to spread out a little of the softened tissue between a slide and a cover-glass in order to find with a quarter objective numbers of opaque mulberry-like aggregations of fatty granules, the well-known "compound granule-masses" or "corpuscles of Gluge." The exact pathological nature of these bodies is still uncertain; but there is scarcely any doubt as to their practical significance, as indicating that the part of the cord in which they are detected is in a morbid condition. Leyden, indeed, quotes statements made by different observers that they are normally present in the fœtus, and that they may also be found in persons at an advanced age, or after death from tuberculosis, pneumonia, or anæmia, when no spinal symptoms had existed during life. But at any rate one need not hesitate to regard them as a proof of disease when they are thickly scattered through some parts of a cord and absent elsewhere. The difficult question of their origin may be left to be discussed hereafter, in the section on softening of the brain. The most remarkable points about them are that they are observed under such diverse pathological conditions, and are fully developed at so early a period. Thus there were large numbers of them in the spinal cord of a patient who was under the care of Dr Frederick Taylor in Guy's Hospital in 1873, and who died within eleven days from the commencement of his symptoms, and not more than fourteen days after the occurrence of a slight injury that perhaps set up the disease. In such cases they are not generally accompanied by an obvious infiltration of leucocytes, or by the presence of any diffused granular matter, but lie embedded among nervous elements which to a superficial observer seem to be normal.

Recent investigations, however, have shown that the histological changes are really far more considerable than would at first sight appear, and it is

* Whether white softening is ever observed when death has occurred at a very early stage I am not sure. I have not found any instance of it among those cases which have proved rapidly fatal at Guy's Hospital. Although Jaccoud speaks of a *myelite suppurée* ('Les Paraplégies,' p. 545), he does not affirm the actual presence of true pus corpuscles.—C. H. F.

to be noted that granule-masses are not infrequently discovered when there is no obvious softening, the texture being, of the two, rather firmer than is natural; and, again, when there is no marked change of colour, or, at most, only a pinkish-grey tint, with some blurring of the pattern on a cut surface. Nor is even the failure to detect granule-masses a proof that morbid changes may not be recognised where the cord has been specially prepared for the purpose. In many cases after it has been soaked in a diluted solution of chromic acid for two or three weeks one can see at a glance what parts of it are diseased, since they remain colourless instead of turning green. And a microscopical examination of thin sections shows that the nerve-fibres have their axis-cylinders swollen and varicose, that the ganglion-cells are enlarged and granular, or perhaps vacuolated; and (according to Erb) that the network of the neuroglia is thickened, while its cells are increased in size and augmented in numbers, and sometimes contain more than one nucleus. Erb also says that a colloid substance is sometimes found diffused along the course of the swollen connective-tissue bundles, and Leyden that a scanty infiltration of leucocytes is now and then seen in the interstices between the nerve-fibres. Such appearances are doubtless conclusive as to the inflammatory nature of the process, and so is the presence of recently effused lymph in the tissue of the pia mater of the cord or in the subarachnoid space. This is sometimes in sufficient quantity to be plainly visible to the naked eye.

Acute myelitis is never limited to the anterior or posterior cornua of the cord, nor to any one of its columns. It is a "diffuse" or "indiscriminate process," not what the German pathologists call a "system-lesion," or, as we may say, a "columnar" disease. It may begin from the surface, in sequence to meningitis, or it may occupy the grey matter around the neural canal ("central" or "peri-ependymal" myelitis), or it may affect the entire breadth of the cord, including both cornua and columns (transverse or segmental myelitis); it may in rare cases be "disseminated;" or lastly, it may spread over large portions at once as a progressive ascending myelitis.

*Chronic interstitial myelitis.**—The disease just described does not in all cases run the course of an *acute* inflammation. Sometimes, indeed, it sets in with fever and ends fatally in a few days, but in many instances it is unattended with febrile disturbances, and goes on for many months before it terminates, either in the death of the patient or in his more or less complete recovery. What justifies the name of acute softening or acute myelitis is the fact that its onset is more or less sudden and its development rapid, so that the paralysis is complete within a week or two.

In marked contrast with the most protracted cases of the disease just described is one which always begins insidiously and gradually, of which the duration is never less than two or three years, and which, instead of leading to softening of the cord, rather renders it firmer and harder than before. The naked-eye appearances in this form of myelitis are variable. Sometimes it is altogether impossible to distinguish those parts of the cord that are diseased from those that are healthy; in such instances no morbid change was recognised until the accurate microscopical investigations of the last few years revealed its presence. In other cases the substance of the cord is obviously tougher than natural. Ollivier long ago aptly compared it to boiled white of egg; the knife meets with resistance in cutting through it,

* *Synonyms.*—Sclerosis of the cord. Grey induration.

and the exposed surface is smooth and even. On close scrutiny it often looks gelatinous, and has a greyish or greyish-yellow tint, instead of the milk-white colour of a healthy cord. It is rather shrunken than increased in size. When held up to the light the diseased parts are translucent. They are untouched by osmic acid, and when stained with carmine they take the colour much better than the unaffected white columns.

With the aid of the microscope the neuroglia is found to be thickened and to have a fibrillated texture, or even, in extreme cases, to be converted into a dense mass of connective tissue with delicate parallel, wavy fibres. Its cells and nuclei are increased in numbers, and are far more conspicuous than in the healthy spinal cord. Cells possessing a large number of radiating processes (Deiters' cells) are often seen; it is said that similar cells may be discovered in the normal neuroglia, but they are at any rate much more obvious in cases of chronic myelitis. The nerve-fibres are generally atrophied; they have lost their medullary sheaths, and their axis-cylinders are described as having a hard, glistening look. The ganglion-cells are shrivelled and granular, or may be converted into homogeneous, bright-looking, angular bodies without processes, and having apparently no nuclei, so that they can hardly be identified. Corpora amylacea are generally abundant, and at the same time granule-masses are present, often in large numbers, but sometimes very few. The smaller blood-vessels are thickened.

This morbid process is now generally known as "sclerosis." It is not, however, peculiar to cases of diffused or segmental chronic myelitis attended with paraplegia, but lies at the foundation of several other affections of the spinal cord, which will be described separately, because their limitation to special parts of its substance gives them an independent place in nosology. In the cases now under consideration the lesion so far extends over the segmental area of the cord that it interferes with or altogether prevents the transmission of motor impulses from the brain to the parts below. Its longitudinal extent varies greatly. Sometimes it is confined to a single spot, and it is often spoken of as "chronic transverse or segmental myelitis;" sometimes it advances slowly along the entire length of the cord (generally from below upwards) until it reaches the bulb. These are the cases known as "creeping palsy."

Ætiology.—According to Erb, inflammation of the cord is more frequent between the ages of ten and thirty years than in older persons. But twenty-five consecutive cases of fatal primary diffused myelitis at Guy's Hospital were pretty uniformly distributed over the several decennial periods from ten to fifty, and three of them occurred in patients between fifty and sixty years old. It is certainly a rare disease in children and in old people—as would be supposed from the nature of its antecedents. Most cases occurred in men, the proportion being 19 to 6. This, probably, depends on the fact that men are much more exposed than women to its various exciting causes. For, although myelitis is certainly not always due to any one morbid influence, its exciting cause can often be traced.

Sometimes it is the result of *exposure to cold*, particularly to wet. Several instances of this have been published,* and our records at Guy's contain others scarcely less striking. Thus, in 1876, a Custom-house officer was admitted

* See, for example, the first three of Dr Bristowe's cases ('Brit. Med. Journ.,' 1888, ii, p. 1369).

into the hospital with nearly complete paraplegia, who had been well until eight days previously, when he was obliged to remain for twenty-four hours in a barge, where everything was soaking wet. On the very next day he was attacked with pains in the joints, which he believed to be rheumatic; his urine began to run away from him; and two days later his feet and hands and back already felt numb. Another patient, who was brought in on January 29th, 1875, had on the 18th of that month got wet and caught cold; three days afterwards he passed his water into his bed without knowing it, and his legs became numb and powerless. Cases of acute myelitis have followed sleeping on the damp ground or in snow and prolonged immersion in water, as when a person narrowly escapes drowning. Ditchers and other men who work up to their knees in water, and women who have tramped through rain till their skirts are soaked through, seem particularly liable to this disease.

Great *bodily fatigue* also seems often to play an important part in the ætiology of the disease. When it occurs in soldiers on active service, as is said to be frequently the case, it may either be due to exhaustion of the spinal cord from forced marches, or to exposure to cold and wet, or to both causes combined. A case in point has been recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856, Case 10). A young man, after walking twenty-eight miles to seek for work, passed the night of July 8th, 1855, in a brick-field. On the 15th he walked thirty-two miles; the day was wet, and he allowed his clothes to dry upon him. Next day he felt as well as usual, but in the afternoon, while he was sauntering in a garden, his legs suddenly gave way under him, and he fell down. He was admitted, six days later, completely paraplegic. He was healthy and florid-looking; but it should be stated that he had, about a fortnight previously, been unable to pass his water for a period of twelve hours. He died in less than a month.

A great muscular effort, such as often causes a pain in the back, is now and then followed, after an interval of a day or two, by paraplegia. An instance of this has already been cited (p. 438) in the case of a dock labourer. Another example of it occurred in Guy's Hospital in 1863: a railway porter, aged thirty-two, was pushing a carriage with his back, when he suddenly gave a jump to escape falling into a pit, and so ricked his spine; for a moment he felt powerless, but was able to resume his work, and worked as usual on the next day; on the day after, however, he was walking in the street, when he suddenly became paralysed and fell down. Both these cases ended fatally in about six weeks; at the autopsy no injury to the bones or the ligaments could be discovered; but in the dock labourer there was softening of the cord in the dorsal region, and the affected part had a greenish colour, from the presence of extravasated blood. The microscopical examination revealed no further proof of inflammation; but if modern methods of investigation could have been used, we might have been more successful.

Again, it occasionally happens that paraplegia develops itself as a sequel or complication of *a specific fever*. Virchow has recorded ('Ges. Abhandl.,' p. 683) an instance in which chronic myelo-meningitis came on a few months after recovery from enteric fever. Westphal examined the bodies of two patients, who became paraplegic during the eruptive stage of smallpox, and in both cases he found disseminated myelitis.

Either *pregnancy* or the puerperal state, too, may be attended with paralysis of the lower limbs. A woman is at the present time (Dec., 1889)

in Mary Ward who is slowly recovering from complete paraplegia, perhaps due to myelitis, which followed severe puerperal convulsions with albuminuria. In one remarkable case of acute myelitis, which ended very quickly in death, Baumgarten found the characteristic bacilli of anthrax in the blood as well as in the spinal cord ('Arch. f. Heilk.,' 1876).

Erb, with many others, believes *syphilis* to be an undoubted and frequent cause of myelitis, and states that the affection may run either a very rapid or a chronic course. That acute paraplegia often follows syphilis there is no doubt; the only question is whether it is due to myelitis (*v. infra*, p. 459).

Lastly, there is reason to believe that *sexual excesses* play a considerable part in the ætiology of diffused myelitis, although it is very difficult to say exactly how large. Men who have been indulging their passions too freely, especially if they are weakly or very young, often experience pains in the back and limbs, which seem to be due to exhaustion of the lower spinal centres, and it is possible that these symptoms may pass into those of actual myelitis.*

Clinical history.—The symptoms of *primary, acute, diffused* myelitis, are first those of paraplegia as above described, including retention of urine and other pelvic symptoms.

Beside, however, causing akinesia, impairment of sensation, dyssesthesiæ, disorder of the reflex functions of the cord, and the like, myelitis is often attended with another set of symptoms, namely, *painful sensations* referred to various parts of the body. In some instances the seat of pain is in the back; it there varies greatly in intensity; it may either be confined to one or two spinous processes, or diffused along the whole length of the cord; there may be extreme tenderness on pressure, or this may be altogether absent. Occasionally spinal pain may be the earliest symptom of the disease, but in the author's experience this is rare. In other instances the pain is referred to the front of the chest, or to the epigastrium, or to one or more of the limbs. One patient, a girl of nineteen, was attending as an out-patient for pain in the chest, when she one day fell down in the waiting-room, struck with paraplegia. Another, a man aged forty-nine, had complained of a fixed pain in the left hip for eight weeks before any definite sign of spinal mischief showed itself. A third, a man aged fifty-one, suffered at first from severe burning pains in the soles of the feet, which continued night and day for a considerable time. These cases all terminated fatally, and in each of them the bones and ligaments and meninges were found to be healthy, and the disease to be limited to the substance of the cord itself.

Sometimes, when pain is referred to the fore part of the body, it is accompanied by a sense of constriction, as if a cord were tightly bound round the waist, or as if the chest were fixed in a vice. We shall hereafter see that this symptom, to which German writers give the special name of *Gürtelgefühl* (girdle-feel), is particularly marked in that other form of paraplegia due to compression of the cord by disease of the surrounding structures. But undoubtedly it occurs now and then in cases of primary myelitis.

* Whether coitus in the erect posture is especially injurious I do not know, nor whether onanism is so, except in so far as it is apt to be repeated frequently, without instinctive desire, and at an early period of life. On these points there is no evidence, nor can one desire to collect it, since, whatever the physical injury of such practices, it is far outweighed by their moral evils.—C. H. F.

The majority of cases of myelitis are altogether unattended with either pain or girdle-feeling.

The *course* of myelitis differs widely, not only according to the nature and seat of the morbid change in the cord, but also in individual cases which appear to resemble one another closely. Erb speaks of the disease as sometimes setting in with malaise and fever, and even with rigors. A case in point occurred at Guy's Hospital in 1872. A man aged fifty-one, who was much exposed to weather, one day began to shiver, and was attacked with severe pains running down the back of the thighs and calves, and the insides of the arms. He remained in bed for four days, after which the pains left him, and he went to work again. Next day he lost all power in his limbs, and he died of dyspnœa about eight days afterwards. Paralysis often shows itself suddenly in that form of myelitis which leads to "softening" of the cord. Indeed, as Erb remarks, the rapid development of this symptom is almost the only reason for calling cases of this kind "acute," since they are often unattended with febrile symptoms, and their duration may be prolonged over a period of many months. In most cases of acute myelitis, however, death takes place in a few days or two or three weeks. It is comparatively rare for the disease to be prolonged in a chronic form, and then also it is almost always fatal.

Prognosis.—Until within the last few years there were no grounds upon which writers could speak with confidence of the possibility of complete recovery from myelitis. It was well known that many patients suffering from paraplegia got perfectly well, but whether inflammation of the cord had existed was uncertain.*

An observation made by Michaud, on a woman who died from caries of the hip and vertebræ, shows that, in spite of the occurrence of well-marked myelitis, recovery from paraplegia due to external compression may take place, though the affected part of the cord retains signs of a morbid change. This case may perhaps be taken as proving the possibility of complete subsidence, with return of normal function, of those local and slight forms of myelitis which we may suppose to be present in partial and transitory forms of spinal paralysis. This conclusion is further supported by the fact that in such cases recovery is often incomplete; since it is more reasonable to suppose that an organic affection of the cord may subside than that a merely functional disorder should be permanent.

The prognosis is generally unfavourable when there is complete paraplegia with pelvic symptoms; but even to this rule there are some remarkable exceptions. Two have been recorded by Wilks in his 'Lectures on Diseases of the Nervous System.' One is the case of a gouty man, aged fifty-two, who had been losing power over his lower limbs and his bladder for a fortnight, so that on admission into Guy's Hospital he could not move his legs, and had partial loss of feeling up to the umbilicus. Reflex movements persisted. The urine, which was ammoniacal, had to be drawn off twice daily. Afterwards he had a feeling of tightness round the lower part of the chest and the abdomen, and he experienced numbness

* Eichhorst and Naunyn found in experiments upon young dogs, that after they had cut through the spinal cord in the lower dorsal region the lesion was slowly and partially repaired; a few nerve-fibres with double contour were developed in the new material which filled up the gap, and motor power and sensation were to some extent regained. These results, however, have not been confirmed by the more recent investigations of Goltz and Freusberg, and it is certain that in man the cord is far from possessing such powers of restoration.

passing down the arms into the fingers. He gradually got wo
a fortnight after his admission he became feverish, with a qu
tongue, rigors, and hiccough ; a bedsore was forming, and the
from him ; moreover, his mind became clouded. Dr Wilks t
was suffering from suppurative nephritis, and had not many
For some days he remained in a precarious state, and then
abated. He began to regain some degree of power in his legs
time he made a rapid recovery ; he sat up in bed, he ceased
catheter, he got into a chair, he asked for crutches, he wa
ward, and finally he left the hospital: this was exactly tw
his admission, and six weeks from the time when his pa
complete. This patient showed himself several weeks later,

The other case is that of a woman of middle age, who ca
from almost complete paraplegia, which had begun a few d
It was accompanied by pain and swelling of the joints—a syn
disease which will presently be described. There was some
ance. She complained of great pain in the limbs, with tw
muscles, and of a sense of constriction round the waist.
ammoniacal and was passed involuntarily, and the sphincter ani
A bedsore formed which extended until there was a deep
back. All the other symptoms continued for three weeks, a
began to recover. She then took tonics and was galvanised,
of five months she was discharged perfectly well. Dr Wilks,
rejects hypotheses of reflex paraplegia and of anæmia or
the cord, confessed that he does not know what was the
disease in these two cases.

The following case of apparently acute anterior myelitis
recovery.

The writer saw in January, 1888, with Dr Cock, of Peckham
years old who a week before had been unable to pass his water
off, and there has been more or less incontinence since. Three or
his legs began to feel weak. When seen he was almost compl
below the waist, with sensation unimpaired, the knee-jerk ab
plantar and cremasteric reflex almost absent. He had scarcel
expel either urine or fæces. There were no convulsions, and
nor pain of any kind. Sensation was apparently normal, so
ficult to fix the upper limit of paralysis. The next day pri
and bullæ appeared on the paralysed legs. The tempera
normal or subnormal. There was no tenderness along the
pressure or to contact with a warm sponge. The paralysed
to faradism as well as to interrupted galvanism. In a week
of the attack there was some return of power in the leg
symptoms gradually disappeared, and he at last recovered
about two months' illness.

Diagnosis.—The decision that myelitis is the cause of part
paraplegia is generally effected by a process of exclusion.

For practical purposes we must first exclude mere shamm
paraplegia—hysterical, alcoholic—and paraplegia due to
peripheral neuritis. The presence of "pelvic symptoms"
generally suffice for this. Next we must exclude "extrinsic
compression of the cord, and chronic and spastic cases of slo

Lastly, we have to decide which intrinsic disease of th

likely to be the cause of the paralysis. Myelitis is at once the most frequent of these affections, and by far the most varied in its symptoms and course.

The occurrence of paraplegia in a person known to be suffering from phthisis or any other tuberculous disease would lead one to suspect *caries of the vertebræ* producing compression of the cord without deformity. It is impossible to distinguish the paralysis caused by a *tumour* within the cord from more common cases due to circumscribed transverse myelitis; but the latter is the more likely. Severe and long-continued pain would probably lead one to suppose that the lesion was in the *bones* or in the *membranes;* but, as we have seen, acute myelitis sometimes causes pain. In two of the cases of paraplegia on which this chapter is founded *hæmorrhage* was, in fact, correctly diagnosed; but, taking into consideration its extreme rarity, perhaps both of them were as likely to have proved myelitis, for suddenness of onset is not of itself a sufficient ground for attributing paraplegia to effusion of blood. As regards *syphilis*, its recognition as the cause of paraplegia must be based upon the clinical history and upon the presence of other syphilitic lesions rather than upon any peculiarities in the spinal symptoms themselves; and no doubt, as with other remote effects of the venereal poison, there are many instances of syphilitic paraplegia in which the most careful scrutiny would fail to elicit proof of their real nature.

There is one positive indication of myelitis that must always be carefully sought, namely, gradual creeping up of the paralysis and anæsthesia from the thighs to the groins, and from thence to the abdomen; it warrants a confident diagnosis of diffuse ascending myelitis. Its early stage is probably indistinguishable from Landry's paralysis (*infra*, p. 456); but after a time the integrity of the recto-vesical functions, and the other negative characters would differentiate the latter.

The diagnosis between myelitis and other varieties of paraplegia, particularly hysterical, alcoholic, and other *functional* cases, on the one hand, and those which depend on *external compression* on the other, will be further referred to under their several heads.

Treatment.—The question remains, whether we can by medical treatment influence the course of myelitis so as to increase the proportion of cases in which recovery occurs; and here we have little positive knowledge to guide us. Of late years it has not been the practice in England to adopt active measures for the acute form of myelitis. The modern German plan seems to be to use leeches or cupping-glasses freely, to apply bags of ice along the spine, to rub in blue ointment or to give calomel internally—in fact, to carry out the "antiphlogistic" treatment very much as English physicians used to employ it thirty years ago. In proof of the value of *ice-bags* a case recorded by Dr Tibbits, of Clifton ('Med. Times and Gaz.,' 1871), is quoted: that of a man suffering from myelitis, which was rapidly spreading upwards, which had already produced paralysis of the arms, and which was attended with a temperature of 103·6°, while the pulse was 132, and the breathing 40 in a minute. Soon after trial was first made of the cold applications he became easier; the temperature fell to 99·2°, the pulse to 100, and the respirations to 24. In the course of the next ten days he completely regained the use of his arms, but no recovery of power took place in the parts which were first paralysed, and he died six weeks later of exhaustion from bedsores. Possibly the issue of the case might have been more favourable if the ice-bags had been employed earlier; paralytic symptoms had been coming on for twelve days, and there had been

complete paraplegia for eight days. Erb recommends blisteri and even the actual cautery, in spite of the risk of setting which he fully recognises.

It is of great importance for the patient from the first to be a water-bed. He should not be allowed to lie constantly on h should be shifted from time to time. It is sometimes danger him to sit up. Some years ago a man, who had recently be into hospital with symptoms of acute myelitis limited to the l the cord, was taken out to have his bed made. When put bac much exhausted, and soon afterwards he died.

The utmost care must be taken to prevent the formatio The sacral region must be kept dry by being cleansed and wip towels. Some good may also be done by sponging the skin wit alternately with hot and with cold water, or by constantly a lotion, so as to harden the skin. Or if a spot is already redde piece of felt plaster may be applied, having a hole in the cen an ulcer has formed, Hammond, of New York, speaks in the h of Mr Golding Bird's plan of placing a thin plate of silve exactly the same size as the sore; a wire six or eight inches lo from this plate to another made of zinc, which is laid on s the skin above, but separated from it by a piece of wash-lea in vinegar ('Guy's Hosp. Rep.' for 1876, 3rd series, vol. xxi, p. vanic action is set up; and Hammond states that he has "fre bedsores three or four inches in diameter, and half an inch deep, in forty-eight hours." Sir Spencer Wells is said to have obta striking results.

Retention of urine must be relieved. A soft catheter sho as soon as interference is necessary, but not before, and care m to depress the free end of the instrument between the thighs, s off the whole of the urine; for if any should be left it will qu pose. The tendency to putrefaction may be checked by the tion of salicylates, or of some other antiseptic medicine, by This practice seems to have been first proposed by Fürbringe seen excellent results since Dr Pavy introduced it at Gu From half a drachm to a drachm must be taken in the c twenty-four hours; it often restores the urine from an offen condition to one of normal acidity and odour. Benzoate instead of soda may be used, in ten or fifteen grain doses three times a much pus is discharged the bladder should be regularly washe weak antiseptic solution. One of the best is Sir Henry Thom lotion.

In some of the less complete and acute forms of mye evidence of the value of *ergot* administered internally; Hamm case which he considered to be one of "congestion of the c which it seemed clearly to be of great service. The patient affected with partial paraplegia, and afterwards with paralysis of from exposure to cold and damp. He was unable to walk u crutches and had a man on each side holding his shoulder. H constant dull aching pain in the loins, and occasional starting while in bed. Under treatment by the extract of ergot (3 recovered in a month. He had a relapse a few weeks later, b in ten days under the same treatment.

The tincture of *belladonna* in fifteen minim doses is said to be useful in similar cases, especially when there is severe pain in the back.

When there is evidence of syphilis, there is little doubt that active mercurial treatment of myelitis by inunction or otherwise is of value; and in more chronic cases of paraplegia following syphilis the Liquor Hydrargyri Perchloridi is sometimes of service.

Many writers recommend full doses of iodide of potassium, not only when there is probability of syphilis, but in other forms of myelitis.

Galvanism should not be employed early in cases of myelitis, but it is probably useful and certainly harmless after the paraplegia has lasted for several weeks and acute symptoms have subsided. The electrodes should be large sponges placed at a considerable distance apart, one upon the neck, the other over the lumbar vertebræ; they may in turn be moved gently up and down, and the direction of the current may be from time to time reversed. When there is reason to believe that the disease is limited to a segment of the cord, one pole may be placed on the affected spot, the other over the front of the chest or of the abdomen. Faradisation of the paralysed muscles may be useful if they are flabby or wasted.

Strychnia is of doubtful value in the later stages of myelitis, and is certainly harmful before.

On the Continent certain spas have great reputations for the treatment of the more chronic forms of paraplegia, with what amount of justice it is hard to say. Erb says that hot baths are apt to be injurious, except at very advanced periods of such cases. Among the places most frequently visited by patients suffering from paralysis are Schlangenbad, Ragatz, Pfeffers, Gastein, Wiesbaden, Teplitz. It is said that in proportion as the elevation above the sea is greater, baths can be borne at higher temperatures without ill effects.

There are two anatomical conditions which, like myelitis, may produce paraplegia of intrinsic origin. Both are rare; and difficult or impossible of recognition during life. They naturally follow the above account of myelitis, which they resemble clinically.

Hæmorrhage into the cord. "*Hæmatomyelia.*"—As a primary cause of paraplegia this is so rare, that Hayem* endeavoured to show that in all the supposed cases which have been recorded since those of Ollivier and Cruveilhier, there was antecedent softening, the result of myelitis. This view would dissociate it altogether from the common cerebral hæmorrhage of old people; but there certainly seem to be some instances in which extravasations of blood into the spinal cord and those into the brain are strictly comparable. A case in point is one of Jaccoud's—that of a woman, aged sixty-two, who was brought into the hospital with complete paraplegia, which had suddenly appeared four days before. The diagnosis was given that there was hæmorrhage into the lumbar enlargement of the cord. Six days afterwards she was found dead in her bed. The autopsy showed that the grey matter of the cord, up to the highest roots of the lumbar plexus, was occupied by a clot which was beginning to undergo disintegration. The cause of death was an immense effusion of

* His monograph, 'Des hémorrhagies intrarachidiennes,' 1872, contains a collection of thirty-two cases with copious references. Goltdammer finds about thirty cases recorded of non-traumatic hæmatomyelia, most of them hæmorrhagic myelitis ('Virchow's Archiv,' lxvi).

blood into the left lateral ventricle, the crus cerebri, the bulb. Erb says that the part of the cord into which hæmo apt to occur is the central grey substance. Sometimes the b throughout its whole length; in other cases it destroys the of the cord at a certain level.

This affection is believed to be less rare in men than in wom the ages of fifteen and thirty-five than at a later period of li instance is recorded by Goltdammer ('Virchow's Archiv,' between fifteen and sixteen years of age. She was one d sitting quietly on a chair, when she suddenly experienced a pain in the back between the shoulder-blades, which compel out. The pain quickly passed into the right arm, and the arm and the lower part of the chest: at the pit of the stom round the body like a girdle. She fell from her seat, and that she could not move her right leg; half an hour later was paralysed; within two hours she was taken into the ho found to be perfectly paraplegic, with anæsthesia up to the n of power over the bladder. Reflex movements could be lower limbs. The case was diagnosed as one of hæmorrhag below the cervical enlargement. She lived almost exactly a autopsy there was found a firm cicatricial mass in the grey stance of the upper part of the dorsal region of the cord elements were completely destroyed; hæmatoidin crystals an ment were present in large quantity, giving a reddish-brown

A few years ago a healthy labourer was admitted into under the writer's care, who after a sudden and severe eff give way under him, and became completely paraplegic with two. There was no pain, no sign of local injury, and steadily recovered. The suddenness of the attack, the com and the complete recovery seemed to point to hæmorrhage in meninges.

Hæmorrhage into the cord, though often fatal, may repaired by absorption of the clot. A remarkable case in which, after severe symptoms terminated favourably, is rec (vol. ii, p. 154). It is doubtful whether any special treatme

Tumour within the cord.—For practical purposes one may this head syphilitic gumma and also tubercle of the cord, j class them with tumours of the brain. Still, the group is a

A *gumma* very rarely begins in the cord itself; it is far (or rather, less rare) in the meninges.

Tubercle in the cord forms a rounded or elongated mass, reaches any considerable size. Two cases were observed in in the year 1870; in one the tubercle was no larger than a p it was compared to a small cherry.* The main substance of a is firm and caseous; its centre may be softened into a crea periphery is sometimes grey, showing a well-marked lymphoi is surrounded by a soft, vascular, pinkish zone, so that it rea

* It is worthy of note that in one of them no sign of the lesion surface of the pia mater. I made a series of sections at the usual di another, and missed it altogether; it was afterwards found by Dr Regin cutting across the cord in fresh places.—C. H. F.

its bed when it has been cut across.* In two cases at Guy's Hospital there was extensive pulmonary phthisis; in a third the peritoneum was tuberculous, and the mesenteric glands were caseous.

If not tubercle, a growth in the cord is probably a *glioma;* for an intrachordal sarcoma is almost unknown; one that was found in the body of a woman, aged twenty-six, at Guy's Hospital in 1860, was an oval soft mass made up of spindle-cell tissue. Such tumours are somewhat apt to undergo mucous softening (myxoglioma). This may probably be the origin of some instances of the remarkable cysts which are occasionally discovered as an elongated cavity in the centre of the cord. It looks like a dilated central canal (*hydromyelus*), but is really not so, as is proved by the fact that in transverse sections the remains of the canal can still be seen in front of it. This condition is described under the name of *Syringomyelus.* Dr Frederick Taylor showed a beautiful specimen in which no clear indications of the presence of a new growth could be made out ('Path. Trans.,' 1878 and 1884). Probably when not a new growth, it is a congenital malformation.

The symptoms produced by tumours of the cord vary widely in different cases. As a rule, paraplegia is present and differs in no respect from that dependent on any other cause. But sometimes the paralysis is of a more limited kind. In a case under the writer's care in Stephen Ward (Nov. 1878) the tumour (a glioma) occupied the cervical region of the cord and caused acute hæmorrhagic myelitis around it. The symptoms were paraplegia and cystitis with paresis of the right arm, and the temperature rose to 108·5° F. shortly before death. Theodor Simon has recorded several remarkable cases in which there would appear to have been a complete absence of all symptoms ('Arch. f. Psych.,' 1874).

Tumours of the meninges producing paraplegia by compression of the cord are not so rare as growths in the cord itself (v. p. 485, *infra*).

There are certain groups of paraplegic cases which are at present without any known morbid anatomy; some are toxic, others functional, but since all of them are believed to have their cause and seat in the cord, their description naturally follows that of intrinsic paraplegia.

Anæmia of the spinal cord.—Unless the modern interpretation of a very old observation is incorrect, one of the possible causes of paraplegia is an arrest of the arterial blood supply to the lower part of the cord. Nicolas Steno in 1667, and Haller afterwards, stated, that after tying the abdominal aorta in animals, the lower limbs became powerless, and remained so until the ligature was removed. This experiment was long afterwards again repeated by Sir Astley Cooper and by others; but until 1869 the explanation universally accepted was that the muscles were paralysed because they and the peripheral nerves were deprived of blood. Schiffer, however, has recently shown that if this were the case an interval would elapse, instead of the effects being observed a few seconds after the operation. Whether a similar form of paralysis is ever seen in man as the result of disease appears to be somewhat doubtful. The most frequent cause of

* Leyden says that tubercles more often occur in the upper than in the lower part of the cord. But of three cases of which I have notes there are two in which the lumbar region was the seat of the disease, while in the third it was in the lower dorsal region. Two of the patients were women, aged twenty-eight and fifty-eight; one was a man, aged thirty-six. Dr Frederick Taylor has told me of a case that he saw in a child.—C. H. F.

obstruction of the abdominal aorta is embolism; but in t might be expected, the clot becomes impacted close to the b vessel—too low to interfere with the circulation in the lum of the cord.

In the 'Guy's Hospital Reports' for 1857, Sir William clinical history of a patient whose aorta was from some obstructed, and who was attacked with paraplegia, which peripheral, rather than spinal. The man was a shipwrigh beginning of March, 1855, while apparently in perfect suddenly seized, being in a stooping position, with pai This went off after he had rested for a few minutes; but his work it returned and extended down the legs, with a sen soon followed by entire paralysis, both of sensation and m loins downwards. After a few days sensation returned, an improved, but the legs remained unsteady. The paraplegic to his coming under Gull's care in June of the same year. that there was no pulsation in the abdominal aorta, nor in the lower limbs. The right superior epigastric artery was alr enlarged, and could be seen pulsating. In the course of the years he regained power to walk tolerably well, but at the en his gait was still languid, and from time to time he had sli weakness and numbness in the legs. His feet also were c Using the legs brought on increased weakness and numbness.

An intermittent form of paraplegia has been noticed in ho of obstruction of the aorta; a loss of power in one or both limbs coming on when they are driven, and subsiding w still. Charcot is said to have observed a similar condition in a right common iliac artery was obstructed. When this man he was obliged to stop every fifteen or twenty minutes, paralysis of the corresponding limb; after a few minutes' rest passed off and he was able to go on again.

Leyden found capillary embolisms in the cord in ulcerativ but no impairment of its functions had been traced to th quotes a single case from Weiss in which a lad suffering tive endocarditis was attackedby myelitis which was pro embolism.

There is some evidence that a defective supply of blood cord may lead to the occurrence of paraplegia in persons who severely from hæmorrhage, or who are highly anæmic. Am of this kind quoted by Leyden there is one of a woman, age who had lost blood profusely after a confinement; she was exce but she was resuming her domestic duties when, at the en month, she suddenly became unable to stand. Another case,

* Leyden has suggested in reference to this case that a sudden obstruct minal aorta could scarcely have occurred without more serious symptoms d selves, and that a collateral enlargement of the epigastric artery would h seen at so early a period afterwards. It must be admitted that the case r "coarctation of the aortic arch" in many respects; among others, in the p tolic murmur at the lower angle of the left scapula. On the other hand, t tomoses over the back and the abdomen underwent gradual enlargement whi observation. And even if the obstruction had existed long before the para appeared, it still may have caused them; for a supply of blood adequate requirements of the lower limbs (or, on the other view, of the spinal cord) insufficient for some specially prolonged effort, or when the man was exhaus

Dr Moutard Martin, occurred in a man as the result of severe hæmorrhage in the course of dysentery. Others were in persons who had suffered from menorrhagia, but it must have been difficult, if not impossible, to exclude hysteria, which, as we shall presently see, may itself cause paraplegia. The same source of fallacy exists in reference to those cases which are recorded as examples of paraplegia due to chlorosis in women. Paraplegia has not hitherto been observed in the intense and fatal form of idiopathic anæmia to which men are liable as well as women.

But, after all, the mere fact that persons who have lost blood are now and then attacked with this form of paralysis is far from showing that anæmia of the spinal cord is capable of directly producing the effect in question. For the proof to be complete there must be no interval of time during which impairment of the nutrition of the cord could have occurred as an intermediate link in the chain of events. In any future case it would be important to note whether the patient, after the hæmorrhage, took violent exercise or worked hard in any way, using his legs more than his arms, so as to cause the lower spinal centres to make special claims upon the nutrient powers of the blood. We might thus be able to see why the lower limbs should alone be paralysed, and not all parts of the body alike.

Some years ago Brown-Séquard professed to be able to distinguish paraplegia due to anæmia from that due to hyperæmia by the simple test that, whereas the former was benefited by keeping the patient recumbent on the back, the latter underwent aggravation when this posture was adopted, but was lessened if he lay on the abdomen, or stood upright, or walked. This method of diagnosis has been endorsed by Dr Hammond, of New York, who mentions a case which he considered to be one of congestion, and in which "all the symptoms were worse in the morning;" but the value of his opinion is diminished by the readiness with which he adopts this kind of hypothetical pathology, and assumes the existence of two distinct forms of spinal anæmia—the one limited to the antero-lateral, the other to the posterior columns. The last affection is one with which we are all well acquainted under the name of "spinal irritation" (or rachialgia); and we have a striking proof that such speculations are baseless in the fact that Stilling and Ollivier both referred this disease to the very opposite condition —congestion of the cord—for reasons which are neither better nor worse than those of Dr Hammond.

Divers' palsy—caisson palsy.—It has repeatedly been observed that men who work at considerable depths under water, as in searching wrecks and sinking the caissons for bridges, are liable to lose the power of their legs after coming to the surface. The paraplegia seems to be chiefly or entirely motor, and usually passes off without leaving traces behind. It has been plausibly attributed to the increased pressure on the surface gradually driving the blood from the surface into the great vertebral plexus of veins, and upon the more rapid return to the surface producing a sudden reflux towards the skin, and temporary anæmia of the cord.

Hyperæmia of the cord.—All pathologists are agreed that distension of the blood-vessels within the spinal canal, if found in the dead body, affords no proof that they were over-full during life; it may be due either to the mode of death, or to gravitation afterwards.

There are sometimes good reasons for believing in the occurrence of morbid processes, in spite of the fact that they cannot be recognised by the

anatomist. But there are no such reasons for recognising h
congestion of the cord as a permanent morbid condition. I
days of pathology, writers assigned all diseases their anatomi
an ascending order of severity. They found that almost eve
liable to certain chronic changes which could be regarded as i
and any cases which seemed to stand lower in the scale were
hyperæmia or to anæmia, according as the one condition
seemed the more easily to be brought into harmony with
symptoms.

Even now a similar method prevails. In 1866 Dr Radcliffe
System,' ii, p. 619) had under his care a woman who had
paralysed in all her limbs the morning after having had her m
checked by an alarm of fire. Among the other symptoms we
the fingers and toes, some degree of general hyperæsthesia
burning aching in the limbs and along the spine, without speci
on pressure over the vertebræ, but with increased sensitiven
sponge. The bladder and rectum performed their functio
There was no over-excitability of the reflex centres in the lowe
cord. Within two or three weeks she began to improve, and
five months she left the hospital convalescent. Considering
stances under which her attack commenced, we can see that t
of spinal congestion is not unreasonable, although other interp
possible.

It is easy enough to pick out cases of paraplegia present
symptoms as those of Dr Radcliffe's patient, and to label them
but no ground appears for giving to these cases the title of spin
rather than to others in which the paraplegia is incomplete a
in recovery. Dr Radcliffe laid stress upon the sudden comm
characteristic; while Erb affirms that a *slow* development of t
distinguishes hyperæmia of the spinal cord. He also mentions,
most important signs of this affection, its fluctuating and chang
but in some cases of myelitis inexplicable variations in the de
paralysis and anæsthesia occur from day to day.

Concussion of the cord.—In ordinary cases of fracture and
the spine, such as come under the care of the surgeon, the pa
is commonly present from the first, is attributed either to cr
cord by a displaced vertebra or to effusion of blood.

A case recorded by Gull shows how a comparatively slight ac
attended by fatal consequences in this way. A man was carrying
back down some cellar stairs when his foot slipped, so that he fell
of coals upon him. He died in thirty-four hours. Two of the cer
were found torn asunder, but this was not the cause of the pa
which he had suffered immediately after his fall, for there was no
of the bones nor any injury to the posterior ligament of the spin
There were, however, several spots of ecchymosis in the posteri
of the cord, as well as in the posterior white columns and the
of the left lateral column. It is perhaps worthy of notice th
lesions did not actually explain the paraplegia, for there is no
motor tracts on both sides were injured. This, however, illus
the view which should be taken of capillary ecchymoses of
centres, whether cerebral or spinal. It is not that they thems

paralytic or any other symptoms, but that their presence, showing that the violence was sufficient to tear asunder vessels, shows also that it must have been enough to lacerate the nervous elements themselves, which are more delicate.

These considerations tend to explain the fact that spinal injuries are sometimes followed by paraplegia, and may even terminate fatally without any morbid change whatever being discoverable even with the microscope. Leyden says that he met with an instance of this kind in which death occurred within five days, and in which he failed to detect any lesion, even after having hardened the tissues with chromic acid. Such cases are commonly described as cases of *spinal concussion*, and it is probable that they depend upon a molecular change in the nerve-fibres and cells of the cord due to the jarring force of the injury. The occurrence of this change would of course be prevented if the vibrations should in any way be hindered from reaching the nerve-centres. Mr Erichsen, for instance, says that after a railway accident those persons who are sitting with their faces to the engine are less likely to suffer from spinal injuries because they would be thrown forwards and could break their fall with their arms and hands, whereas those who sat the other way would be driven against the back of the carriage when its motion was suddenly arrested so that there would be scarcely anything to prevent the spinal cord from being violently shaken.

The cases of which one can most confidently speak as being examples of concussion of the cord are those in which a blow or a fall is instantly followed by paralysis, and in which recovery takes place so rapidly that the alternative diagnosis—hæmorrhage—can be satisfactorily rejected. Such an instance perhaps is one related by Erb. A man, aged fifty-five, fell twenty feet from a tree upon his heels and ischia. He was not unconscious, but was at once unable to walk, and had to be carried home. He experienced severe pains in the sacrum and in the lower limbs, but these passed off after a time. There was no loss of sensation, but the legs were said to be entirely motionless for a week. When he came under medical observation at the end of four weeks he could only make a few steps at a time, and slowly and with hesitation. Reflex movements could be produced as usual. The excitability of the nerves and muscles by electricity was much diminished. Galvanic treatment, applied both to the spine and to the legs, was attended with great success. He was soon able to walk well, and in three weeks he was discharged cured.

But in most cases in which railway accidents are followed by paralysis this does not manifest itself until some time has elapsed. The interval is often of several days' duration, and Mr Erichsen says that it may even last two or three months. During this time the patient is not, indeed, well; he is suffering from other effects of the injury, but he frequently has no idea that his spine has been hurt.

It is difficult to see how the diagnosis of "concussion" is to be established as against that of myelitis, when the symptoms are thus remote. However slight they may be, one may surely conceive them to be caused by an inflammation of the cord limited to a sufficiently small area of its substance. And the fact that they generally subside after a time affords no argument to the contrary, now that we know that myelitis is a disease from which recovery is possible.

SCENDING PARALYSIS.*—Diffuse myelitis sc
ds along the cord, and destroys life in a few
re not very infrequent, the name of acute asc
irly applied. Recent writers, however, reserve i
one in which the most minute scrutiny has hi
reveal any lesion of the cord. Landry published ten cases
regarded as this form of paraplegia in the 'Gazette Hebdoma
It is said to be definitely characterised by its symptoms; for in
ing acute myelitis there is marked anæsthesia, the bladder
totally paralysed, bedsores are formed at a very early period,
excitability of the muscles soon becomes extinguished; when
ascending paralysis of Landry none of these phenomena are

Onset, course, and symptoms.—Sometimes there are prodr
of slight febrile disturbance, malaise, dragging and shootin
back and limbs, sensations of numbness and formication i
in the tips of the fingers, and, above all, a feeling of
exhaustion and weakness. The patient may go on comp
way for a day or two, or for a week, or even (in one rec
longer. More often no such symptoms arise, the earliest
anything is the matter being a loss of strength in the lowe
rapidly passes into complete paraplegia. The feet first become n
the legs, and afterwards the thighs. Soon the trunk is invol
becomes impossible during defæcation, and in coughing or
intercostal muscles are paralysed in succession from below upw
same time, or even earlier, the hands are affected; the patie
write or to feed himself; his grasp rapidly becomes enfeeble
power extends to the arms and shoulders. Lastly, the musc
the upper cervical nerves, including the diaphragm, fail in t
act of swallowing becomes impossible, and death by suffocat
scene. Towards the last it is sometimes noticed that the sp
rassed, that liquids regurgitate through the nose from paralysi
and that the masticating muscles and those of the face are we
pupils are sometimes unequal, and transitory diplopia has t
served. There may also be very rapid action of the heart, w
has reached the upper part of the cervical cord.

In some cases the order in which different parts are paraly
be reversed. The fatal illness of the great naturalist Cuvie
kind, and terminated in less than seven days. His first sy
sensation of discomfort at the epigastrium. Next morning h
a difficulty in deglutition, and in the evening he could swa
and had marked loss of power in the upper limbs. The paraly
became absolute, and affected the lower limbs also. Such
events can only be explained on the supposition that the m
(of whatever nature) descends the cord, instead of ascending;
limited to grey matter, leaving the white columns unaffecte

* *Synonym.*—Landry's paralysis. Poliomyelitis anterior acutissima wa
on a wrong diagnosis of its nature, both clinical and pathological.

Kussmaul's two cases of "Fatal Paraplegia without assignable cause"
were published in the same year as Landry's paper. The account of Lan
in the text is founded on the statements by Erb, Eichhorst, Ross, and
Remak's article "Spinallähmung" (p. 651) in Eulenburg's 'Real-Encyclopä

Neither Dr Fagge nor the present writer ever saw a case, and one or tw
Dr Ross appear to be all that have occurred in England during thirty years.

course contain at each level fibres belonging to all parts of the body below. It is doubtful whether these cases can fairly be brought within the narrow definition of Landry's paralysis recognised by systematic authors; they only resemble the typical cases in there being no recognisable lesion, and in the sensibility of the paralysed parts remaining perfect.

The most remarkable and characteristic features of the disease are these two. The affected limbs lie flaccid and free from spasm. At first reflex movements can be excited; after a few days both superficial and "deep reflexes" are lost; but the electrical contractility of the muscles persists to the last, nor do they waste. The functions of the bladder and rectum are unimpaired; there may for a time be retention of urine, but this quickly passes off, so that a catheter has seldom to be used. The patient may complain of slight feelings of numbness, or of formication, but the paralysed parts are still sensitive to touch and to painful impressions. No pains are experienced in the affected limbs, nor is there any tenderness of the spinal column. The patient may be so completely free from discomfort, and from all the ordinary signs of serious illness, that it may be difficult for those about him to realise the gravity of his state. In one of Landry's original cases, M. Gubler thought for a day or two that the paralysis was feigned, nor was there any apprehension of danger when the patient was within eight hours of death.

Pathology.—Morbid anatomy has hitherto thrown no light whatever upon the nature of this disease. Westphal ('Arch. f. Psych.,' 1876) examined the spinal cord by the most approved methods in three typical cases, and with absolute negative results. The same result followed autopsies by Vulpian and Hayem.

Before, however, accepting any but an organic change as the origin of the symptoms, it would be necessary to examine the whole of the motor tract, including the anterior roots of the nerves.*

Westphal supposes that the affection of Landry may be due to the operation of some hitherto unrecognised poison; others suggest the presence of a microbe; and Erb thinks that its nature is the same as that of tetanus—an analogy which explains very little.

There is no febrile reaction, but Westphal observed the spleen to be enlarged, and this has been regarded as evidence of the malady belonging to the group of "blood diseases."†

Prognosis.—The mean duration of fatal acute ascending paralysis is said to be from eight to twelve days. It has been known to destroy life in two or three days, but sometimes it runs on for as many weeks, and may end in the patient's recovery. Landry spoke of eight out of his total of ten collected cases (only four of which he had seen himself) as having terminated favourably; but his paper contains no details to justify the diagnosis. Erb says that the disease may stop at any period of its course, even when respiration and deglutition are affected.

Ætiology.—With regard to its causes, nothing is known. It appears to occur mostly in young adults, and more frequently in men than in women,

* Since the above was written, Nauwerck and Barth have published, in the fifth vol. of Ziegler's 'Pathologische Beiträge,' a case of Landry's acute ascending paralysis, in which they found after death the cord normal, but neuritis of many of the nerve-roots and extensive destruction of the fibres in both sciatic nerves.

† A fatal case of paralysis at St Thomas's Hospital resembled Landry's cases: it was that of a man who had been inoculated for hydrophobia, and suggested to Dr Bristowe a comparison with the paralytic form of that disease in dogs and in rabbits.

the proportion in Pellegrino-Levi's cases being twelve to four. Some cases have been ascribed to exposure to cold, or to some of the other supposed causes of myelitis described above (pp. 448–9); some have occurred during convalescence from acute diseases, as enteric fever and diphtheria, a statement that must not be taken as referring to the ordinary diphtheritic paralysis, of which the characters are altogether different.

Rarity.—Landry's paralysis is of exceedingly infrequent occurrence in England, and not a single typical case has been recorded in Guy's Hospital. Dr Wilks ('Lectures,' p. 274) relates seven cases of acute ascending paralysis, and in three of them the cord was found normal. But apart from the difficulty of accepting negative conclusions before modern histological methods were in use, the presence of anæsthesia, of vesical symptoms and of pain in all these cases, brings them under the clinical category of acute ascending diffuse myelitis. A case in a young man which ended favourably is recorded by Dr Ross, and another which proved fatal; but the latter he regards as extremely doubtful. Other authors, Bristowe, Buzzard, Gowers, Bastian, Bramwell describe the disease from French or German accounts, rather than from their own experience. The early cases of Walford ('Brit. Med. Journ.,' November, 1854), Handfield Jones (ibid. October, 1866,) and Harley ('Lancet,' October, 1868), in which last Lockhart Clarke found extensive lesions in the cord, cannot be admitted as genuine cases, and many of those published abroad are either myelitis or subacute atrophic spinal paralysis.

The fully described and probably genuine cases are certainly very few, even abroad, perhaps not thirty. The rapid course, the successive implication of legs, arms, and trunk, and finally of the bulbar nerves, including the portio dura, the absence of marked anæsthesia or pain, the absence of bedsores and rectal or vesical disturbance, the loss of knee-jerk, the absence of atrophy or spasm of the paralysed muscles and their retention of electrical contractility—make up a striking combination. The usually fatal course and negative anatomical result complete the type.

No *treatment* can be said to be indicated, but the application to the spine of ice, of heat, of the actual cautery, and of other counter-irritants, has been recommended, as well as galvanism, ergot, and other drugs.

Alcoholic paraplegia.—Under this name Wilks described a form of partial motor paralysis of the lower limbs not uncommon particularly "in ladies who have given themselves up to brandy-drinking." Pains in the legs and anæsthesia are also present.

These symptoms, with the dragging gait from dropping of the toes, the integrity of the functions of the pelvic organs, and the favourable prognosis under proper treatment, make up a recognisable clinical "disease." Dr Buzzard has suggested that symmetrical and multiple peripheral neuritis is the pathological cause of these symptoms (*vide supra*, p. 432). Dr Gowers takes the same view, and regards as minor cases of the same malady those which have been described as Alcoholic Ataxia and Pseudo-tabes potatorum. This was in fact Wilks's own opinion, for he says that sometimes the symptoms are almost confined to the legs, and resemble in character those of locomotor ataxia.

In Dr Broadbent's remarkable case of alcoholic paralysis ('Med.-Chir. Trans.,' 1884) the cord was found normal, but the nerves were not examined. He compares it with Landry's paralysis. It was in some respects unique;

but his other cases agree with Buzzard's in occurring in women, in being chiefly paraplegic, and in the muscles being tender on handling, with in one case at least sharp pains, dropped-wrist, and sphincters unaffected.

On the whole it seems probable that drink produces paraplegia occasionally by means of myelitis, and more frequently by peripheral neuritis. The latter and more common condition is recognised by the symptoms described in a previous chapter (p. 424) by the absence of pelvic symptoms, and by the frequent implication of the hands.

Syphilitic paraplegia.—That paralysis of the lower limbs may be one of the remote effects of syphilis has for several years been a well-known fact, but morbid anatomy has hitherto done very little to define the exact nature of the changes which occur in such cases. As to their relative frequency there is some discrepancy of opinion. Dr Buzzard has expressed his conviction that, in a person between twenty and forty years of age, a case of paraplegia, when not associated with Bright's disease or embolism, is (like hemiplegia under the same conditions) in nineteen cases out of twenty the result of syphilis. But this statement appears to ignore the important distinction between the pathology of the brain and that of the cord, which is represented by the common occurrence of idiopathic myelitis as compared with the infrequency of primary local encephalitis leading to softening; and probably Heubner is right when he asserts that the *lues venerea* affects the spinal cord far less often than the brain. Buzzard's limits of age appear to be too narrow, for in a case observed by Wilks the patient was a woman of fifty-three, and among the five or six other cases which have been examined in the *post-mortem* room of Guy's Hospital within the last few years, one occurred in a man aged fifty-seven, another in a woman aged forty-seven. But it is probable that syphilitic paraplegia is usually myelitis from diseased arteries. This form of paralysis has hitherto been chiefly studied at the bedside. Many years ago Sir Benjamin Brodie said that he had several times seen paraplegia stopped in its course by bichloride of mercury or by Plummer's pill. The inference has since been drawn that the cases in question were syphilitic, although there is nothing to warrant it in Brodie's brief statements concerning them. A similar conclusion has sometimes been based upon the striking results which now and then follow the administration of iodide of potassium for paraplegia, a wonderful instance of which occurred in the practice of Dr Wilks many years ago.

The frequency with which nodes are seen upon the inner surface of the skull, pushing the dura mater inwards, at one time led to the supposition that a similar affection of the bony walls of the spinal canal might occur. But this is not borne out by observation in the deadhouse. A gumma beginning in the interior of the cord is almost unknown, but it sometimes occurs in the membrane and produces paraplegia by compression.

In certain cases supposed to be examples of syphilitic paraplegia, no obvious morbid change has been discovered, or only a softened state of the cord; and we have seen that syphilis is probably an important cause of myelitis.

A man, aged twenty, was in 1877 under treatment by Mr Davies-Colley for syphilis, when he became paraplegic and was transferred to the care of Dr Wilks. He died two months later. The cord in the mid-dorsal region was flattened and soft for about an inch and a half of its length; the antero-lateral columns and the grey matter were especially affected, the latter being

of a rusty brown colour. To the naked eye there was no obvi
the pia mater. But when a piece of it, corresponding with
part, was placed on a microscopic slide and examined with a l
of the arteries were at once seen to be enormously thickened
rated. By reflected light they looked like solid, opaque, whi
by transmitted light they appeared black. We could not find
the affection was in an earlier stage, so that we might have con
Heubner's description of syphilitic arteritis in the brain. Pr
cases, hitherto regarded as syphilitic myelitis, may depend on de
supply from syphilitic affection of vessels in the spinal pia mat

Reflex paraplegia.—We have passed from myelitis as a cause
paraplegia to other more or less probable anatomical condi
ætiological varieties of paralysis. There remain cases of *functio*

It has long been suspected that a loss of function in the
causing paralysis of the lower limbs, may be an indirect res
visceral diseases. Such cases would be fairly comparable with
mentioned (p. 396), in which paresis of one arm was caused
tooth. Hammond relates the case of a girl who was brong
account of paraplegia which had suddenly developed. He
several doses of santonine, followed by castor-oil; a number of
were passed, and the paralysis disappeared in the night.

Within the last few years there has been much discussion
in which this kind of paralysis is brought about. Mr Stanl
perhaps the first to describe it ('Med.-Chir. Trans.,' 1833), v
to refer it to "irritation, propagated through the sentient
spinal cord," whence he supposed that "the impression" was
through both the motor and sentient spinal nerves to the lim
sioning an impairment both of sensation and of the power
Obviously, however, this explanation goes but a very little w
Dr Brown-Séquard published in the 'Lancet' an elaborate t
ing to which irritation of afferent nerves of a diseased organ
to set up a reflex spasm in the blood-vessels of the spinal
render it anæmic and impair its nutrition and its functions. In
year, however, Sir William Gull refuted this opinion in the 'G
Reports,' and no one now accepts it. Nor has a suggestion of J
accepted that reflex paralysis is due to "exhaustion" of th
the cord upon which fall the stimuli conveyed by the sensitive
irritated part. Thus the field remained clear for the entrance
doctrine of "inhibition," which in theory meets all the difficult
satisfactorily. Its application to paraplegia seems to have be
by Lewisson ('Reichert's Archiv,' 1869). In a series of ex
rabbits, he succeeded in paralysing the lower limbs by forci
between his fingers the kidney, or the uterus, or a large loop
The loss of power is described as lasting only as long as the
continued, or a little longer, and as always disappearing abr
nothing is easier than to refer nervous phenomena of all kinds to
influences; but this very fact makes it essential that the path
never extend the area of inhibition a hair's breadth beyond the li
a strict interpretation of physiological observations would confi
paralytic affections already referred to as being caused by disea
the paraplegia from worms in Hammond's case, were all in p

with Lewisson's experimental results, since they subsided as soon as the source of irritation was removed. Graves placed on record the case of a man who was admitted into the Richmond Hospital in 1835 with partial paraplegia of two weeks' duration, and a tight stricture, which had existed for some months. He had recently been much exposed to cold and wet, and this might have been regarded as the cause of the paralysis. But in a very few days after the first introduction of a catheter a remarkable improvement took place in his legs and in his back, where he had had much pain. In fact, the change was almost sudden; and within a month the power of the lower limbs was completely restored. It would seem that we may fairly regard this as an instance of reflex paraplegia. Cases have been recorded in women in whom the reduction of a displaced uterus has been followed by a marvellously rapid recovery from paralysis; but then it is scarcely possible to exclude the supposition of hysteria from these cases. On the other hand, the immense majority of cases which are supposed to be of reflex origin run a protracted course. It is true that the disease which is supposed to be the starting-point of the inhibitory influence is often itself permanent and incurable; but sometimes it subsides entirely, and yet the paralysis persists.

In such cases it is only reasonable to admit that there is something more than a mere reflex paraplegia; and, indeed, myelitis has sometimes been discovered after death. A particular group, in which the paralysis has appeared to be secondary to some affection of the bladder or of the urethra has been known by the distinctive name of "urinary paraplegia." Three instances of it were recorded by Gull in 1856 ('Med.-Chir. Trans.,' xxxix); in each of them a part of the spinal cord was found softened. Two similar cases have come under the observation of Leyden; the paralysis seemed to have started from a vesical affection, the result of a long-standing stricture; there was a limited inflammation of the upper portion of the lumbar enlargement with granule masses. Leyden remarks that the seat of this lesion, corresponding exactly with the spot at which the nerves of the bladder enter and leave the cord, suggests the conclusion that the morbid action had extended along their fibres. This opinion has since been supported by experiments made by Tiesler and Feinberg and Klemm, each of whom succeeded in generating an inflammation of the lower part of the spinal cord in animals by setting up neuritis of the sciatic nerves. In a dog operated on by Tiesler there was actually a collection of pus in the substance of the cord "at the point of exit of the sciatic plexus." Roesingh, however, has repeated some of these experiments with negative results, so that the question can hardly be regarded as finally settled.

Leyden ascribes most cases of reflex paraplegia to progressive ascending neuritis, and believes that the inflammatory process can be traced step by step along the whole course of the nerves.

Sir William Gull several years ago proposed a different route for the transmission of the morbid action, namely, by the veins. In a man who died of paraplegia, consecutive to a long-standing stricture, he found a small quantity of pus lying outside the sheath of the dorsal part of the cord; and one of the vertebral veins in the lumbar region was full of pus. But it is mentioned that there was a large slough over the sacrum, and, as we now know that a putrid inflammation often invades the lower end of the spinal canal from a bedsore, most probably the suppuration and the venous thrombosis arose in that way shortly before death, and were, in fact, indirect consequences of the paralysis.

There would be no difficulty in collecting from medica journals a large number of cases of supposed reflex paraple would need to be most carefully criticised.* In the 17th c Essay on "Injuries and Diseases of Nerves" Mr Bowlby has a series.

When chronic paraplegia develops itself in a person who had a stricture, or who has recently had dysentery or some tinal affection, or who has a retroflexed or prolapsed uterus, forget that this may be a mere coincidence. Brown-Séquard down a series of fifteen criteria, by which he thought that a refl might be distinguished from one due to an organic lesion in th now that we know that myelitis is present in most of the cases to be of reflex origin, these criteria must be set aside, even if ex not shown that no reliance could be placed on them. The exclude the possibility that the relation between the primary di paralysis is accidental is to show that the cases in question are for mere coincidence. But has this yet been shown? If not congestion of the cord, reflex paraplegia must be at least a rare malady.

Hysterical Paraplegia.—It is a well-recognised fact that not infrequent cause of paraplegia; the proof being that young have previously suffered, or are actually suffering, from oth symptoms are apt to be affected with paralysis of the legs, wh after a time, or sometimes suddenly, under the influence of so moral shock. To define as accurately as possible the characte affection must evidently be of great importance in reference and treatment; for the diagnosis cannot be taken for granted u fact that the patient has hysteria, and (on the other hand) affe hysterical are occasionally met with in persons who do not pr indications of that disease.†

Mere malingering is not difficult to detect, but hysterical p very different thing. Apart from the probabilities afforded by

* In the Guy's Hospital Library copy of the 'Medico-Chirurgical T 1833, containing Mr Stanley's paper, there is a pencil-note by Dr Wilks, t the majority of his cases were wrongly interpreted, and were really exampl inflammatory softening of the cord, attended with secondary cystitis and n are, indeed, two cases to which this remark does not apply: one is th admitted into St Bartholomew's Hospital for retention of urine (attribu gonorrhœa, the discharge of which he had stopped by injections), who be and died in about a fortnight; the other, of a man who was getting well when he was seized with paralysis extending up to the umbilicus, and fell bed sixteen hours afterwards. But we shall find cases in which septicæmi fatal cerebral symptoms for which no adequate explanation could be found and it seems not unlikely that the cases just quoted were really of that nat merely reflex, or due to inhibition of the spinal centres.

† There is a remarkable want of agreement among writers as to the crite paraplegia. Radcliffe and Bastian say that the paralysis is usually incomp completeness is, in a doubtful case, an argument for paraplegia being Duchenne attached much importance to the presence of a diminution of el without loss of electro-contractility; but Reynolds has related two cases sensibility, sensibility to electricity, and electro-contractility were all perfec that although each of these patients could raise her legs from the bed, ye made to attempt to walk, no amount of help could prevent her from tumbling down to within a few inches of the ground, and then she would without assistance. Even as regards micturition, Radcliffe states that " rectum are little, if at all, under control; less so, as a rule, than in common

sex of the patient, the following points are of service:—There is usually retention but seldom or never incontinence of urine; obstinate constipation but never incontinence of fæces; and bed-sores do not form. The motor palsy, though absolute at certain times, is at others slight; sensory palsy, patches of anæsthesia, hyperæsthesia and analgesia are much more marked than in ordinary paraplegia, as much as in total transverse lesions with pelvic symptoms. Analgesia without loss of touch or sense of temperature, and anæsthesia without loss of movement are almost diagnostic of hysteria. Electro-sensibility is often small or absent, but the muscles respond normally to galvanic or faradic stimuli. The paralysed limbs are cold but do not waste. Rigidity and contraction, most frequently flexion, are frequent and early symptoms, but they are completely removed under chloroform and during sleep. The knee-jerk is sometimes absent, sometimes exaggerated.

Wilks remarks that hysterical girls remain plump in spite of their being affected with paralysis. Sir William Gull used to point out the coldness and pallor of the legs in such patients, as contrasting with their condition in cases of organic disease of the cord.

We ought to be extremely careful in diagnosing paraplegia as hysterical, since neurotic girls are not less liable than other people to myelitis, caries of the spine, and other causes of organic paraplegia.

Dr Reynolds has described a closely allied functional paralysis, which he called paraplegia dependent on an idea ('Brit. Med. Journ.,' Nov., 1869).

The *treatment* of hysterical paraplegia is that of hysteria, and electricity should only be used, if at all, for its effect upon the mind.

NEURASTHENIA SPINALIS.—"*Spinal weakness.*"—Among the patients who seek medical advice, believing that they are suffering from spinal disease, there are some—chiefly youths and men not far advanced in adult life—whose principal complaint is of muscular fatigue and weakness. If they attempt to walk far, they experience aching pains or stiffness in their limbs. When they have to stand for more than a short time, their legs tremble and seem to give way under them. They feel tired and weary all day long, even before they get out of bed in the morning. "Spinal Neurasthenia," as a special name for these cases, was originally proposed by Drs Beard and Rockwell in 1871, and is accepted by Erb, Ross, and other systematic writers. The exciting causes appear generally to be either over-exertion of body or mind (especially if carried on during the hours that ought to have been devoted to sleep), or the various forms of sexual excess. There is irritable weakness of the generative organs, and sometimes a little dribbling of the urine after micturition. In some cases dragging or tearing pains in the limbs were complained of; but generally these were not severe. Very often there was a peculiar pain in the back, seated apparently in the muscles, increased by movements of the spine or of the scapulæ. Sometimes local tenderness of certain spinous processes was present, exactly as in the affection known as rachialgia or *spinal irritation;* it may be said that the two neuroses are combined in the same patient. Often there is mental disturbance indicated by sleeplessness, timidity, and depression of spirits. In fact, the condition is that long recognised as a form of sexual hypochondriasis, or, as it used to be called, tabes dorsalis.*

* The cases cited in a work by Surgeon-Major Neale, published under this title in the year 1806, are many of them of this character.

It is supposed by Erb that the state of the nervous el spinal centres in such cases is in fact the same as in the heal exhausted by the discharge of its functions, and that the dif natural period of rest failing to restore them to vigour and a view seems reasonable enough.

The *diagnosis* from myelitis and from other serious spinal be based mainly upon the absence of objective symptoms of a of the cord, in contrast with the vehemence with which the plains of his subjective sensations.

Spinal neurasthenia sometimes develops itself in the co days, but much more frequently its advent is gradual and s reach such an intensity as to compel the sufferer to give up l and to renounce all society. It is often obstinate, lasting for or for years. Relapses sometimes occur. Whether it ever passes on into any organic affection is doubtful.

Treatment should be directed entirely to the general hea from the spinal symptoms. Cold sponging, exercise out of nence from liquor and from sexual indulgence, aided by the steel, strychnia, or cod-liver oil are usually successful. Galv of decided benefit.

RACHIALGIA. "*Spinal irritation.*"—In 1828 Dr Thomas E gow, drew attention to an affection attended with pain and ten or more of the vertebræ, and termed it *spinal irritation*. He by Mr Teale, of Leeds (1829), by the brothers William and of Limerick (1834), and by Stilling, of Cassel (1840), the devoted a volume of 540 pages to the subject. All these ob very wide scope to the definition. They detected tenderne over certain spinous processes in persons suffering from var and other affections, and they maintained that in all such c state of the spine was really the fundamental disease. They their definition, so as to include transitory paralytic affections of other neuroses. In this they were no doubt influenced which they had adopted, for it would obviously be difficu from "spinal irritation" almost any "functional" disorder nervous centres from tetanus to pleurodynia.

It is not surprising that these views met with much oppo were criticised in trenchant style, but very justly, by Romb have never met with general acceptance in this country. Ollivier maintained that "spinal irritation" was due to the the cord (p. 453). More recently Hammond has asserted tha condition is spinal anæmia (p. 451). The boundary lines b irritation and other affections have been repeatedly shifte observers have ignored it altogether. This is to be regretted, undoubtedly some cases which naturally fall under this hea have hitherto found no other place in the nosology. It is clinical importance to recognise an independent neurosis, cha pain and tenderness in the back, but unattended with any organic lesion of the cord, or of the vertebræ. The most of "spinal irritation," may be regarded as a form of neura the posterior branches of the spinal nerves, and perhaps distributed to the meninges of the cord. It is true that the

and of Van der Kolk (p. 386), would explain how pain may be referred to the cutaneous nerves of the back, even though its starting-point is within the spinal cord; and such a view of "spinal irritation" is undoubtedly supported by the occasional transference of impressions from the posterior to the anterior branches of the nerves in cases of this kind. After all, this question of its centric or excentric seat is not peculiar to rachialgia, but concerns every other form of neuralgia likewise.

But even if spinal irritation is a neuralgia, it is most usefully considered in this place, on account of the importance of the diagnosis between it and the more grave diseases of the vertebræ and of the cord—just as it is convenient to classify pleurodynia with affections of the chest, and gastralgia with those of the stomach. Considering the abuse of the term "spinal irritation," it is better to denote this condition by the word *Rachialgia*, which was long ago employed by Joseph Frank, and corresponds with those applied to other local neuralgic affections.

Symptoms.—The severity of the pain varies indefinitely in different cases. Sometimes there is no spontaneous pain at all; but by pressing upon the different spinous processes we may find that some of them are tender; the patient shrinks or complains that we are hurting him, but he may hitherto have had no idea that anything was the matter with his spine. Great stress was laid on cases of this kind by those who pushed the theory of "spinal irritation" furthest. They were in the habit of directing their treatment mainly to the supposed spinal affection, and applied leeches, blisters, or tartar-emetic ointment over the vertebræ. Romberg, however, tells us that very little was effected by these measures, which we can well believe.

In other cases there is a dull aching sensation in the affected part of the spine, or even severe pain. Its development is usually gradual. At first it may be felt only after great fatigue or excitement; but, as time passes, slighter causes excite it and it is less ready to subside, until at last it becomes constant. It is almost always increased by muscular efforts and by movements of the vertebræ, so that the patient is unable to walk far, or to stand upright for more than a short time; even sitting at the piano, the writing desk, or the sewing machine, may be too painful to be borne.

Sometimes the best way of bringing out the increased sensitiveness of the affected nerves is to pass one's fingers along the spine, so as to press upon the vertebræ in succession; sometimes it is more manifest when a sponge wrung out of hot water is drawn down the back. To the patient this seems to scald at the tender spots, while he experiences no discomfort elsewhere.

There is often a close anatomical relation between the seat of rachialgia and that of any other neuralgia which happens to be present in the same case—the one corresponding with the posterior, the other with the anterior main branch of a spinal nerve. This statement is, indeed, almost identical with one which used to be insisted on by Trousseau; namely, that a "*point apophysaire*" is discoverable in most instances of neuralgia. Pressure on the tender vertebra will often bring out or increase the pain in any other part that happens to have its nerves in an irritable condition.

Thus one of the cases related by Dr and Mr Griffin (at p. 19 of their work) is that of a girl who complained of headache and pains in all parts of her body. Her whole spinal column was acutely tender. Pressure upon the

first or second cervical vertebra caused a pain which shot fo occiput to the brow; a little further down, it excited pain over the lowest cervical spine, at the spot where the trach the sternum; and still lower, at the middle of the sternu cartilage, and the pubic region successively. This certainly any direct mechanical impression upon the cord; for whe made behind the trochanter, pain was felt at the iliac crest, the thigh, and even in the opposite hip; while pressure up knee set up pains in the shins and toes.

In some cases there is a fixed spot in the front of the pain is always referred when any one of several tend pressed upon. Thus another of Dr Griffin's patients had g of all the dorsal and lumbar, but not of the cervical spi Pressure on the upper dorsal vertebræ caused pain at the sternum; from the third or fourth dorsal down to the sa pain, not in the corresponding points as usual, but at the ens Pain at this spot was even brought on by pressure behind upon the muscles of the thigh, or over one knee-joint, and the same pain if she chanced to tread on uneven ground, or i beneath her feet in walking. To ascertain whether the sev dorsal vertebræ were, as usual, more affected than other pa firm pressure was made upon them. The result was tha tumbled forwards insensible, and would have struck her f floor had not some one caught her. Such an occurrence mi almost incredible; but the same observers have placed s record.

Another of their patients was a lady who complained of but who "had no conception that her spine was at all affecte second cervical vertebra was touched she sprang up, as i been driven through the cord, and then fell in a state insensibility. Out of this stupor she twice started up in and as often dropped back powerless, her countenance evin terror and agitation. As soon as she could speak, she sai felt a numbness and sensation as of pins and needles in all ensiform cartilage. She would on no account permit her nec again.

A fourth instance was that of a young gentleman, who d as suffering from a chronic liver complaint. Pressure u column was excessively disagreeable to him. When the fi one of the dorsal vertebræ he grew pale and terrified, saying thrill through every nerve in his frame. He had an unp about the part for the remainder of the day, and shuddered allowing the pressure to be repeated. After a few weeks was tried again, and with precisely the same results.

A fifth case was that of a boy, aged twelve, who fell forw as if he had been shot, as soon as slight pressure was made u lumbar spine.

Phenomena somewhat similar, though less marked in uncommon. They undoubtedly lend support to the view t rachialgia is in the cord itself rather than in the nerves. A a case in which pressure on one spot, over the lowest ce caused exquisite pain, a sensation of extreme nausea, and d

the pulse at the right wrist, that on the left side remaining unaltered. The patient, a young lady, was also seen by Dr Walshe and Dr Reynolds. In the case of a child, brought to Dr Frederick Taylor, at Guy's Hospital, it was repeatedly noticed that pressure upon the seventh cervical vertebra at once gave rise to a marked pallor of the left side of the face, which lasted a few minutes.

Ætiology.—Dr Radcliffe suggested that the cause of rachialgia (or, as he terms it, "spinal irritation") is often a strain of the back or a blow, which may have been forgotten by the patient. This supposition is confirmed by the fact that slight railway accidents so often give rise to it. The so-called "Railway-spine" is, in fact, in the great majority of cases an affection of this kind. Anstie's patient, just referred to, had received a very slight contusion in a collision. Her sister, who was severely injured at the same time, nursed her assiduously for three or four months, and then began to be seriously ill herself. Many observers think that no reliance can be placed on the statements of persons who have met with such accidents, so far as their subjective symptoms are concerned. But the author saw several cases in which there was abundant collateral proof of good faith.

A general "nervous susceptibility" certainly plays an important part in the ætiology of the complaint. It is far more common in women than in men, and in persons between fifteen and thirty years of age, than in those at earlier or later periods of life. An inherited "neuropathic tendency" predisposes to it. Like other neuralgiæ, it often affects women who at the same time are obviously suffering from hysteria; and thus it may be associated with any of the varied symptoms of that morbid state.

Among other causes to which rachialgia has been attributed are over-fatigue, exhaustion by night-watching, sexual excesses, onanism, violent mental emotions, and insufficient food; but probably none of these are more than predisposing causes.

Prognosis.—Rachialgia may develop into marked hysteria in women, or into neurasthenia spinalis in men, but the usual course of the affection is towards recovery. Relapses, however, are common, and often the subsidence of pain in a particular spot in the back is followed by the appearance of neuralgia elsewhere, or of some different neurosis.

Treatment.—A most important point to be determined is whether or not rest in the horizontal posture should be insisted on. Some of the patients whom the late Mr Hilton used to keep lying on their backs for months together, with sandbags to prevent any movements of the spine, were (as Dr Fagge believed) sufferers from rachialgia rather than from actual spinal disease; yet undoubtedly good results were obtained. Mr Teale says that he succeeded in curing several persons belonging to the poorer classes while they were still pursuing their laborious vocations; and in many cases the general nervous state renders it exceedingly desirable that the patient should have fresh air and change of scene, and should even be encouraged to take moderate exercise, short of fatigue. An abundant supply of good food is of great importance. Dr Radcliffe advised a somewhat bold use of alcoholic drinks as a cardinal point in the treatment; but Dr Anstie would only allow them in great moderation, and only with the meals. This is the wiser course, or we may have not only rachialgia but a worse disease to deal with. If "spinal irritation" is only a combination of hysteria and neuralgia, it is food, not narcotics, which are useful. Quinine, the tincture of steel, strychnia, and cod-liver oil are each of them valuable

adjuvants. The application of blisters to the spine is reco
some modern authors, and older writers speak no less favourab
and of cupping. These measures are now out of fashion; bu
severe and obstinate case which arose out of the Thorpe rail
and which the author saw with Mr Erichsen and with Mr
Norwich, nothing gave so much relief as the repeated applicati
at intervals of a few weeks, and especially at the catamenial p
patient was stout and florid. Other applications that hav
proved useful are turpentine liniment and unguentum veratr
of hot sand may be placed along the spine, or a galvanic cu
through it in the manner directed for neuralgic affections in ge

HEMIPARAPLEGIA.—There is a curious and rare kind of para
a lesion which is limited to one lateral half of the cord, but
half completely at a certain level. As might be anticipated, t
power in the half of the body which is on the same side as t
the cord, and the upper limit of the paralysis varies with the d
the highest nerves coming off from the part of the cord belo
the disease. Such a condition may conveniently be terme
plegia, whether it is limited to the leg or involves the corres
as well. What could not have been foreseen, until Brown-Séq
demonstrated the fact in animals by dividing one half of the
is that anæsthesia is present, not on the same side as the diseas
opposite side. That half of the body which retains its moto
its sensibility, while the paralysed half has its sensory function
or exalted. The explanation of the crossed anæsthesia is t
the fibres belonging to each sensory nerve-root decussate
stance of the spinal cord itself, immediately above their en
least before they reach the level of the decussation of the
in the pyramids. This applies equally to the fibres which
sorts of impressions,—whether of touch, or pain, or heat an
with the curious exception that the fibres belonging to t
sense run upward without crossing. Thus, there is said to
muscular sense, and with it, of electro-muscular sensibility, on t
and not on the anæsthetic side of the body. Why there shoul
hyperæsthesia of the skin on the paralysed side,—the susc
tactile and to painful impressions and to change of temperatu
increased,—is not so clear. The inflammatory action which de
in the neighbourhood of the lesion may render the fibres undu
or there may be a suppression of some normally controlling inf

Thus it would appear that no disease of the spinal cord pro
rise to paralysis and anæsthesia limited to one and the same
body. If a patient is found to have one of his lower limbs,
affected in this way, we are justified in concluding that the
concern the corresponding half of the nerve-roots which form
equina—unless, indeed, it is seated above the decussation of t
in the opposite half of the encephalon. In other words,
peripheral neuritis or a cerebral lesion.

On the other hand, it must not be supposed that every affe
side of the spinal cord necessarily causes paralysis of the sa
anæsthesia of the opposite side of the body. This effect is ob
when the abolition of function in that half of the cord is compl

so. All morbid changes in the nervous structures produce loss of motor power more constantly than loss of sensation, and unilateral lesions of the cord offer no exception to this rule. It is quite possible for such a lesion to give rise to complete paralysis of one lower limb, or of the arm and leg on one side of the body, without there being any anæsthesia of the limbs on the opposite side. Such cases were formerly known under the name of "spinal hemiplegia." The explanation of them is that the disease, while it is confined to one half of the cord, nevertheless leaves a part of that half functionally active. The best marked cases are those which will be hereafter discussed as essential spinal paralysis in infants and in adults, where the anterior cornua are the seat of the lesion.

The vaso-motor nerves are said to be affected on that side which is the seat of the lesion, especially when this has occurred suddenly or developed itself rapidly. The temperature of the paralysed limb or limbs is at first higher than that of the corresponding parts by a difference amounting to 1° or 2° Fahrenheit or even more. After a time, however, it becomes normal, and sometimes falls to a still lower level.

With regard to the state of the reflex excitability of the lower spinal centres in cases of hemiparaplegia, there is still some uncertainty. On the paralysed side it has in some cases been increased, in others it has been diminished; on the anæsthetic side it has generally appeared to be normal, but occasionally it has been exalted. Erb says that the bladder and rectum have generally been paralysed—always when there has been a sudden traumatic lesion—with either complete retention or complete incontinence of urine, and involuntary passage of fæces. This appears to lend powerful support to the view that the paralysis of these parts in cases of ordinary paraplegia, due to a bilateral lesion limited to the upper part of the cord, depends on an inhibitory influence transmitted downwards from the diseased parts to the healthy centres below. An analogous fact, which has now been noticed in several cases, is that the muscles on the paralysed side in hemiparaplegia sometimes have their faradic contractility lowered in a marked degree; they may also undergo rapid wasting.

Hemiparaplegia may be due to many different *causes*, but not to so many as those which may produce paralysis on both sides of the body alike; and some of the rare lesions of the cord are most apt to give rise to it. Thus it has been observed in patients who have been stabbed in the back by a knife or dagger. It seems strange that a chance wound should make a section of one half of the cord with the accuracy of an incision performed by an experimental physiologist; but the shape and size of the spaces between the arches of the vertebræ are possibly such as to prevent a cutting instrument from passing across the median line within the spinal canal. Now and then a fracture or a dislocation of the spine has been attended with hemiparaplegia, and perhaps an effusion of blood into the membranes on one side may cause it. A tumour outside the cord, compressing one half of it, has more than once caused paraplegia of one leg. In other cases this form of paralysis has been due to a lesion limited to the interior of the cord on one side: an effusion of blood, a patch of sclerosis, or of acute focal myelitis, an intrachordal tumour, or a gumma.

Some of these various unilateral lesions are more apt than others to affect a considerable length of the cord, and at the same time to involve the roots of several spinal nerves. This leads to the development in many cases of hemiparaplegia of a fresh set of symptoms due to interference with nerve-roots.

Thus there is commonly an anæsthetic half-zone of greate passing round the paralysed half of the body from back dividing the hyperæsthetic part of the surface below from t above; and the upper edge of this anæsthetic space is sai present a narrow hyperæsthetic border. The explanation i nerves of the region deprived of sensation have had their roo the disease; those of the region which is over-sensitive have n roots irritated. The other (or anæsthetic) half of the body has a narrow hyperæsthetic half-zone, limiting the anæsthe due to irritation of fibres which have just decussated at the up lesion in the cord. In some cases the patient experiences sense of constriction, or severe burning or shooting pains, rou a level corresponding with that of the nerves whose roots are disease, and such sensations may be limited to one half of th both sides alike. We shall presently see that exact coun these symptoms are met with in certain forms of paraplegi on slow compression of the cord.

The *course* taken by hemiparaplegia varies in different to the nature of the disease that gives rise to it. It very stationary. Most commonly it soon undergoes conversion paraplegia, as the result of the development of myelitis, w whole thickness of the cord around the original lesion. fatally, but sometimes, according to Erb, it subsides and allow of a unilateral lesion to reappear. These may then persist fo change, and in certain cases they have been known to termin According to Brown-Séquard, the power of motion is then and more completely than that of sensation. If this is th opposition to what is observed in all other affections, whethe or of nerve-trunks.

The second group of affections causing Paraplegia (cf. p those in which the primary lesion is *extrinsic*, or outside the co subjecting it to *slow compression*, so as first to produce pain with the posterior roots of the nerves, then to excite sec with softening, and finally to destroy the cord completel affected.

Compression-Paraplegia.—Ollivier and Cruveilhier separate form of paraplegia that which is produced by t pression of the cord." The importance of thus grouping t affections of the parts which surround and support the sp not to have been recognised by the writers who followe recently, several observers have worked out the idea in others Charcot, who, however, includes in his descriptio tubercles lying in the interior of the cord, although, as he some of the most characteristic phenomena are absent in suc

Causes.—In the first place it is to be remarked that some spine never cause paraplegia. Thus the cord seems always to sion in cases of lateral curvature (or *scoliosis*, as it is often c extreme may be the deformity. Leyden, indeed, alludes to which the patient was unable to stand or walk for more tha of time, but such an effect is quite exceptional. Whethe

canal is ever narrowed in cases of *osteo-arthritis* is doubtful. In 1838 Aston Key related in the third volume of the 'Guy's Hospital Reports' two cases of paraplegia in each of which Wilkinson King—a pathologist of deserved reputation—found an intervertebral substance projecting backwards with raised lips upon the edges of the two adjacent vertebræ, and in one instance with an ossified posterior common ligament bridging over the space between them. It was taken for granted that this was the cause of the paralysis, but this may perhaps be doubted. Gowers mentions two cases of intravertebral *exostosis* compressing the cord. Syphilitic *nodes* seem never to grow from the bodies or arches of the vertebra inwards, so as to interfere with the cord.

1. *Caries of the spine. Pott's disease.*—The most frequent, and therefore by far the most important, of all the affections that are really capable of giving rise to a "compression-paraplegia," is that which, from the kind of deformity produced by it, is in this country commonly known as "angular curvature," but which on the Continent is universally called after one of the greatest English surgeons of the last century, Percival Pott, of St Bartholomew's Hospital. It consists in the destruction by caries, with or without necrosis, of the body or bodies of one or more vertebræ, which then fall together beneath the weight of the head and upper part of the trunk. The necessary result is a displacement of the corresponding arches and spinous processes, which become bent into a sharp angle or rounded curve, according to the number of the bones which are diseased.

Opinions differ with regard to the mode of commencement of this morbid process. German pathologists agree in stating that it begins in the bodies of the vertebræ, the discs escaping or being implicated only at a late period; and undoubtedly one may see vertebræ with caseous masses in their interior, or even irregular cavities, which nowhere touch the discs. But, as Wilks long ago pointed out, there is in some cases a source of fallacy in the fact that when a disc is completely destroyed, the adjacent vertebræ often lose each about half of its substance, and the remaining halves, coming together, look exactly like the fragments of a single bone ulcerated through by the disease. The author has repeatedly found intervertebral discs above and below the main seat of mischief presenting early changes of such a kind as to convince him that in the particular case under observation they, rather than the bones, were primarily affected. Thus, in an instance recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856, p. 179), death occurred from an affection of the cord at a time when the only change was softening of three of the discs, with the formation of a cheesy substance in the middle one and a little "absorption" limited to the adjacent part of the bone.

Another question formerly debated, whether caries of the vertebræ is to be regarded as "scrofulous," is now decided—partly by the presence of tubercular lesions elsewhere, partly by detection of the characteristic bacilli of Koch. Cases of tubercular pyelitis or of Addison's disease are now and then accompanied by an affection of the vertebræ immediately adjacent; and a similar affection occurs in conjunction with pulmonary phthisis, or tubercular disease of the testis, or disease of other bones or joints. Like tubercular lesions of joints, caries of the spine can, however, often be traced to accidental injuries.

In most instances of permanently cured angular curvature with ankylosis, the active stage of the disease was passed through in childhood. Of sixteen cases, all causing fatal paraplegia, in fifteen the patients were adults, four between twenty and thirty years of age, three between thirty-one and forty,

seven between forty-one and fifty, and one fifty-six. The only ankylosis existed was one in which the spinal disease beg patient was six years old, and he died at thirty-two.

Considering how great is the deformity in many of these c much it alters the relations of the arches of the vertebræ to one could not be surprised if the displaced bones often co cord directly. That this does sometimes occur appears clea of Brown-Séquard's, in which paraplegia which had set in removed in twenty-five hours by extension of the spine. But logical museum contains specimens which show that the generally remains of its full width, however much its dire altered. Moreover, as Charcot points out, paralysis often oc of vertebral caries in which there is no curvature at all; w cases, in which curvature exists, the patient regains the use although the state of the bones remains unaltered. The im of pressure on the cord when there is caries of the spine is, as in 1856, a mass of cheesy *débris*, which collects between the affe and the dura mater, having, perhaps, been extruded from fibro-cartilages or from the carious bones, as the result of dest posterior common ligament. As Michaud observed, the ou the dura mater become in their turn converted into a thick, yel mass.

2. *Malignant disease of the spine.*—This is the only other f of extrinsic paraplegia. It assumes a variety of forms. is secondary to carcinoma of the breast, or to sarcoma occ lymph-glands of the neck or loins or in one of the long bones of the œsophagus or colon, the uterus or testicle. The nature is sufficiently obvious if there has been a primary affection in parts, especially if a surgical operation has already been perf is otherwise when the growth is seated internally, as, for in mediastinum. Sometimes the vertebræ become affected with mal by direct extension, as from a cancer of the kidney. Sometim lesion is itself a primary new growth. In these cases it is gener of one kind or other; and not infrequently it affects simultaneo of vertebræ in different regions, as well as other bones, such a limbs, the ossa innominata, or the skull. Thus a careful sear may lead to the discovery of a tumour in some distant part and so may clear up a case that would otherwise have remain obscure.

Sometimes there is a distinct projection of one or more cesses, or a mass of the growth can be felt within the sub erector-spinæ muscle. Sometimes a tumour in connection wit the spinal column is discovered when deep pressure is mad cervical vertebræ are diseased, manipulation of the neck m enlargement on one side corresponding with the transverse pr in the great majority of cases one can feel nothing abnormal lies entirely within the spinal canal, except in so far as it bodies or laminæ of the vertebræ.

Of seventeen cases of malignant disease of the spine, colle records of *post-mortem* examinations at Guy's Hospital, tw males, five in females. The patients were of all ages, fr sixty-eight.

In one case in Philip Ward a correct diagnosis was possible, from the age of the patient excluding caries, while the severe pain and its increase on pressure of the spine downwards pointed to the vertebræ. It was confirmed before death by the detection of a tumour in the abdomen, which proved to be a cancerous gland.

In another case, occurring in a man aged 50, the paralysis was very slight, and the pain and other symptoms subsided so much after rest in bed that the patient was allowed to get up. The same day he was attacked with renewed pain, and paraplegia became complete. Death speedily followed, and we found that while secondary cancer of the vertebræ had compressed the cord, the weight of his body in walking had crushed the bones together, and destroyed that segment.

In two of the writer's cases, the primary growth was in the œsophagus. In thirteen other cases at Guy's Hospital it was in the vertebræ in five, in the bladder and prostate in two, in the breast, uterus, colon, or adrenal in the remainder.

3. *Erosion of the vertebræ by aneurysm.*—Common as it is for an aneurysm of the aorta to eat away the bodies of the vertebræ with which it comes into contact, it very rarely penetrates the spinal canal so as to interfere with the cord. We have in Guy's museum specimens from two such cases, in each of which paraplegia developed itself at a period long after the discovery of a pulsating tumour in the back. A third remarkable instance of this kind occurred in 1871. A man, aged thirty, was admitted into the hospital for paraplegia, which had begun three months previously. He had first complained of pain in the shoulders, then his left leg became weak and numb, and afterwards his right leg, and he experienced a pain as though the abdomen were constricted by a cord. He died without our suspecting of the real nature of his disease. When the erector spinæ was cut into, a large mass of laminated clot was found in its substance on the left side. This belonged to an aneurysm of the descending aortic arch, which had destroyed several ribs, and laid open the spinal canal for a space two inches in length. Some of the clot adhered to the outer surface of the dura mater; the cord itself was flattened, white, and very soft.

4. *Erosion of the vertebræ by a hydatid.*—This also is extremely rare. A striking instance is recorded by Cruveilhier. A large echinococcus lay behind the vertebræ, and filled the groove on each side of the spine beneath the lumbar muscles, so as to form two elongated pouches, which were connected together by a narrow neck. A somewhat similar case occurred to Dr Moxon in 1871, a few months before the case of aneurysm just referred to. The parasite was "multilocular," budding externally. It formed a large elastic swelling on the left side of the spine, and made its way into the canal through the second and third lumbar vertebræ. It compressed the cord, but did not penetrate the dura mater. The patient, a woman of fifty-eight, had been paraplegic for six weeks.

5. *Meningeal tumour.*—New growths of various kinds occasionally form in connection with the spinal membranes, and may press on the cord so as to produce paraplegia. Sometimes a lipoma or an enchondroma is formed in the connective tissue outside the dura mater of the cord. More frequently, one of the spaces within the sheath is the seat of the affection. According to Charcot, tumours are particularly apt to grow from the inner face of the dura mater—generally sarcoma or psammoma. New growths

of the spinal membranes, although rare, are much less so th
begin in the cord itself.

Sir William Gull described three cases of this kind in
paraplegia in the 'Guy's Reports' for 1856: one (? a myxo
the inner surface of the dura mater at the first dorsal vertebr
a sarcoma, had the same origin a little lower down; the thi
gumma) grew from the pia mater in the lower dorsal region.

In one more recent case at Guy's Hospital, a tumour of
loped fibrous tissue lay close to the foramen magnum and e
wards for more than two inches, so as to press upon the ba
on the left side. In another case a soft, granular, redc
smooth and lobulated on the surface, lay loosely attached bet
noid membrane and the pia mater, in the dorsal region.
inch and a quarter in length, and consisted partly of fib
spindle-cell tissue.

6. *Meningeal gumma.*—This affection also seems to be
more so than tumour. Virchow has related a case in which
in the lower cervical region was increased to three times its nc
and was bound down to the bodies of the fifth and sixth ce
by a large quantity of firm connective tissue, which was belie
in a gumma. Wilks once found a hard, irregular, yellow mas
of an inch long, and probably syphilitic, lying on the right s
within its sheath, and adherent to the pia mater and to the pc
the spinal nerves, which were compressed by it. In a case
('Guy's Hosp. Rep.,' 1871) there were a number of brown
patches, from the size of a barleycorn to that of a pea, with
centres, penetrating into the substance of the cord from
In one described by Heubner, in 'Ziemssen's Cyclopædia,' a
extended from the floor of the fourth ventricle into the bulb
one twelfth of an inch; while in the cervical region the
the other membranes behind the cord were pressed togethe
mass, which was firmly adherent both to the arch of the a
posterior columns of the cord.

Symptoms of compression-paraplegia.—At the bedside we re
condary paralysis, and distinguish it from primary affections of
not so much by peculiarities in its proper symptoms as by th
are accompanied, and in most cases preceded, by symptoms d
ence with the roots of the nerves coming from the cord at
the level of the lesion. By Charcot these are termed "extrin
while he gives the name of "intrinsic" symptoms to the los
in parts lower down, to the anæsthesia, and to the other effe
upon the cord itself.* Erb describes the former as belongi
stage," the latter to a "second stage" of the disease.

The statement that the earlier phenomena are due to son
nerves having their roots directly involved in the lesion, i
speculative opinion. They have been observed on dissection
and greatly swollen, even when not showing any mark
change; while in advanced cases they have been found transp
and atrophied, with their fibres in a state of fatty degenerati

* The terms extrinsic and intrinsic are also applied to denote lesions
and those which affect it from outside, respectively—intra-medullary an
as they are called by some writers, with reference to the medulla spinalis.

Chief among the early symptoms is *pain;* and few points in clinical medicine are more important than the fact that this is referred by the patient, not to the seat of mischief, but chiefly or solely to those parts to which the affected nerves are distributed. Thus, there is always danger of mistaking cases of spinal disease for various other affections; now for occipital or brachial tic, then for pleurodynia, and again for gastrodynia, for colic, or for sciatica. Very often the pain appears to be fixed in a single spot, or to occupy only a very small part of the whole surface supplied by a single nerve. In other cases it shoots through all the main trunks belonging to a limb. Charcot lays stress on its burning character and on the absence of *points douloureux*, as distinguishing it from (idiopathic) neuralgia; but if we believe with Anstie that the *points* are to be detected only in nerves which have already been the seat of pain for a considerable length of time, the distinction is not of much service.

Associated with the pain there is often an extreme degree of hyperæsthesia. The patient may be unable to bear even the slightest touch without crying out, while every movement is excessively painful. On the other hand, there is sometimes impairment of sensibility or actual anæsthesia over a more or less extensive part of the surface to which the affected nerves are supplied. The muscles which receive branches from them may show tonic or clonic spasms, or become paralysed and even atrophied, with loss of faradic contractility and inability to respond to reflex stimuli. An eruption of herpes zoster has now and then been observed in the course of some of the nerves which are involved in the disease.

It is to be observed that these pains are symptoms of compression-paraplegia only indirectly; they depend on the nerve-roots or branches being pressed upon, and may occur whenever spinal nerves or their roots are involved in disease, though the cord may to the last remain intact. Thus lateral curvature of the spine, which probably never causes paralysis, is frequently accompanied by a fixed pain in one or more of the ribs or intercostal spaces, which seems to be due to pressure upon nerves as they are passing through the intervertebral foramina. In the following remarkable case of medullary sarcoma of the vertebræ there was at no time any well-marked paralysis. The patient was an errand-boy, aged sixteen, who, five months before his death, began to experience pain first in the loins, then in the shoulders, and then in all his limbs. His sufferings became almost unbearable. The whole surface of the body was excessively tender, but especially the lower part of the back. The pain was constant, but it varied in position. There was numbness and a sensation of tingling in each hand; this was first noticed in the fingers supplied by the ulnar nerve on either side. He became wasted to the most extreme degree. The only way in which he got any ease was by lying flat on his back, with his legs drawn up. Every movement of his neck, and of any part below it, caused him severe pain. One day he became insensible and had a series of epileptiform seizures, in one of which he died. A mass of white medullary new growth (with microscopical characters, such as are now known to belong to the sarcomata) occupied the lumbar glands, and spread from them to the intervertebral discs, eating also into the lumbar vertebræ themselves. It passed up in front of the spine into the neck, where it involved part of the brachial plexus on each side. It nowhere penetrated into the vertebral canal or implicated the membranes of the cord.

There seems to be no doubt that pain is more marked as an early symptom

in cases of malignant disease of the spine than in those of compression-paraplegia.

Cruveilhier long ago brought together under the name *douloureuse* certain cases attended with intense lancinating pain of the sciatic and lumbar plexuses, and with paralysis of t branches. Recent writers have remarked that a large majorit in question are instances of malignant disease of the spine, roots of the nerves for the lower limbs in the cauda equina for this distribution of symptoms seems to be that wherea lumbar and lowest dorsal vertebræ frequently become cance not often affected with caries.

On the other hand, in fourteen consecutive cases of p to Pott's disease of the vertebræ, collected from our *post-m* at Guy's Hospital, the caries did not once occupy this the spine. A few other cases in which it did occupy this without paralysis. Thus in one instance the third and vertebræ were found "extensively diseased," and in anothe and twelfth dorsal vertebræ had entirely disappeared, so that o the spine a large space was seen in front, in which the cord was exposed; but in each case the disease had been during life, for the patient had been kept in bed for a consid some other malady, and this no doubt had enabled the spinal being compressed. In the report of the second case it is e that repeated questions failed to elicit any complaint of pain, back or elsewhere.

Pain in the back itself—rachialgia, already described as although not infrequently present as an early symptom of com plegia, is far less constant than one might have expected. different forms. Certain of the spinous processes may be tend or over-sensitive to a slight blow or jar, or to a hot sponge, may be conscious of a feeling of stiffness in moving the bac according to the seat of the mischief. Sometimes—as in a in the cervical vertebræ placed on record by Mr Cæsar Haw a great increase of pain in the neck when the face is turne pillow to one side or the other, so that the hands are used head in every change of posture.

Intrinsic or myelitic symptoms.—After the "extrinsic" s lasted for weeks, months, or even for years—having been perh neuralgic or rheumatic—they are succeeded by others of wh origin is obvious. These "intrinsic" symptoms are by no results of mechanical pressure upon the cord. Very gr pressure by itself may probably alter the shape of the cord, only begin when the pressure interferes with its circulation a

As far back as 1856 Gull discovered in a case of this masses in the tissues of the cord; and more recently th spinal cord in compression-paraplegia has been thoroughl by Michaud and other French observers. The affected pa reduced in size (so that sometimes it is scarcely as thick flattened or distorted in shape. Its tissue may be pale, natural to the naked eye; or it may obviously have lost it ture. It is either softened or (in a more advanced stage) ha Under the microscope the neuroglia is seen to be thicker and

the nerve-tubes have no longer any medullary sheaths, but their axis-cylinders persist, and may even be increased in size. Granule-masses are abundant. The ganglion-cells are swollen, vacuolated, and pigmented, or sometimes they are degenerated and broken down. In other words, there is a chronic transverse myelitis, which extends a little way above and below the spot actually compressed; so that many cases of paraplegia due to disease of the vertebræ or other causes of compression of the cord are really cases of secondary myelitis.

The course of the paraplegia is such as might be inferred from the nature of the lesion causing it. Sometimes its development takes place very rapidly—within two or three days, or even in a few hours—much more often it occupies several weeks, or months. The patient finds his legs more and more heavy, especially in going up and down stairs; he becomes unable to stand; at last he cannot even move his toes while lying in bed. Subjective sensations of numbness, pins-and-needles, &c., often precede the motor symptoms; but, later on, loss of voluntary power over the muscles generally preponderates over loss of sensation. The upper limit of the spinal lesion is commonly well marked, especially if there is much anæsthesia. It corresponds more or less exactly with the seat of the early "extrinsic" pains and tenderness, which, indeed, often persist after paralysis has set in. A point of great importance is that there is little tendency for secondary changes in the cord to spread upwards in the main tracts beyond the point of compression. Sometimes the local myelitis from compression gives rise to ascending sclerosis of the posterior median columns without special symptoms; more frequently to *descending* sclerosis of the lateral columns.*

Reflex contractions in the lower limbs are usually much more readily excited than under normal conditions.†

The state of the bladder is very variable; it often continues to act naturally for some time after the legs have begun to be paralysed, but whenever the paraplegia is complete there is retention of urine.

Charcot lays stress on some forms of *paræsthesia* which, he says, although not peculiar to compression-paraplegia, are observed more frequently in these cases than in other affections of the cord. One is a retardation in the transmission of sensory impressions when there is not absolute anæsthesia; fifteen, twenty, or even thirty seconds may elapse between the application of a stimulus to the cutaneous surface and the perception of it by the patient. This symptom we shall meet with again as a symptom of Tabes dorsalis. Another is a peculiar dysæsthesia, a very painful feeling, which is excited by slightly pinching a limb or touching it with anything cold; this lasts for several minutes, and apparently no more exact description of it can be given than that it is a sort of diffused vibration. Another is an "associated sensation" which is referred to a spot upon the opposite limb corresponding exactly with that to which the stimulus is applied.

Diagnosis.—The determination of compression as the cause of paraplegia may be either very easy or very difficult. The simplest cases of all are those in which there is obvious spinal deformity, for the short round bend or sharp angle produced by Pott's disease is unlike anything else. But often the natural configuration is preserved.

* Erb quotes Michaud as having observed that in some very rare cases a morbid action may ascend along the lateral columns, so that the upper limbs may after a time become paralysed, although the part of the cord which is compressed is in the thoracic region.

† See on this point a recent paper by Dr Bastian, with remarks by Dr H. Jackson, Mr Bowlby, and Dr Buzzard ('Med.-Chir. Proc.,' February 25th, 1890).

Leyden says that if caries occurs in either the lumbar or region there is rarely any projection of the spinous processe being that the natural curve of those parts of the column is in direction; but if in these cases the patient is made to stoop head forward, it is not uncommon for some one of the cervic spines to appear unduly prominent. It is also important to obs the vertebral column retains its flexibility.

In malignant disease, if there is anything to be detected on r it is the presence of a new growth and not a mere displac bones. Cruveilhier, in his hydatid case (p. 473), discovered twelfth dorsal and first lumbar vertebræ a spot the size of a six felt like a depression surrounded by a bony ring, and which the remains of a spina bifida. As he says with regret, if he interpreted what he observed, and had ventured upon making the patient might perhaps have been cured.

Tumours and gummata are of course beyond the reach of r A careful analysis of the symptoms affords the only chance of d these cases from those of disease of the vertebræ unattended wit The peculiar severity of the pain caused by malignant growth a diagnosis to be made between that particular form of comp plegia and the rest.

It is often difficult to distinguish cases of caries of the ve meningeal tumour from those of primary *transverse myelitis*, or affection of the interior of the cord limited to a particular segme is often accompanied by painful sensations referred to various body, and by the peculiar "girdle-feeling"—phenomena whic comparable with the "extrinsic" symptoms of compression-pa which do not always differ from them even in degree. We that in some cases diagnosis between the two forms of dis between primary and secondary myelitis) is impossible.

It is chiefly at an early period that a case of compressi might be mistaken for one of *rachialgia*. The suggestion has, made that, considering the frequency with which this spina associated with hysteria, one ought to be prepared for its be then accompanied by hysterical paraplegia, so as very closely grave disease of the vertebræ. It is probable that in such a c careful examination of the patient would show a want of co between the apparent upper limit of the lesion in the cord, a tenderness and pain in the spinal column; but as a fact we do cases of rachialgia are complicated by hysterical paraplegia.

Prognosis.—Most forms of paraplegia by compression end in the patients, but their duration is very variable.

According to Leyden, *cancer* of the vertebræ commonly of some months, sometimes of more than a year; it kills, cachexia and wasting with dropsy, or more directly by interfe cord. Among fifteen recent cases in Guy's Hospital (1888) from the earliest symptoms till death, was three months in year in eight, and fifteen months in one patient. When the vertebræ are affected, the fatal issue is sometimes preceded by stupor, for which no explanation can be found in the autopsy.

Meningeal *tumours* are described by Erb as slower in their cases end in eight or ten months, others in from two to five

last longer still, even to a period of fifteen years. In one of the two recent cases above given (p. 474) the duration of the disease was sixteen months, and in the other two years, if we reckon from the time when pain in the back was first complained of; and both patients may be said to have died accidentally—the one of pneumonia, the other of renal suppuration.

The only variety of paraplegia from compression in which recovery of power over the limbs appears to be possible, is that due to *caries* of the spine. Of such cases a great many do well; among six which came under Erb's observation during the year before he wrote his article on this subject, only one ended fatally, the remaining five being cured or greatly relieved. Charcot refers to two patients, who had their lower limbs completely paralysed for eighteen months and two years respectively, and who nevertheless recovered perfectly.

One might well have doubted whether in these cases of caries of the vertebræ, in which recovery takes place from secondary compression-paraplegia, inflammation of the substance of the cord has ever occurred, But an observation of Michaud, which is cited by Charcot, shows that this really is so. A woman who had regained the use of her legs for more than two years, died of hip-joint disease. The cord, at the level of the spinal affection, was found reduced to the thickness of a goose-quill, its sectional area being not more than one third of that of a healthy cord in the same region. It was of firm consistence and grey in colour; in other words, it seemed to be affected with sclerosis in an advanced stage. The microscope, however, showed that a considerable number of nerve-fibres possessing medullary sheaths were embedded in the thick dense fibrous material which gave it these appearances. Only one of the grey cornua remained, and this displayed but a small number of uninjured nerve-cells. This case has been already cited in proof of the possibility of recovery from myelitis (p. 445).

Treatment.—The most important means of treatment in cases of paraplegia from angular curvature is complete rest in the recumbent and, as much as possible, in the prone position.

When from any cause this is impracticable, or when it has been continued with good results, and the patient is first allowed to sit up, a valuable means of securing rest for the diseased part is Sayre's method of applying a plaster-of-Paris bandage, rolled round the body while the patient is suspended from the head, chin, and armpits by a suitable apparatus.

Cod-liver oil should be administered, and every effort made by fresh air and good food, particularly of a fatty nature, to combat the tubercular process.

At the present time (March, 1890) the writer has under his care a case of complete motor and sensory paraplegia from vertebral caries in a young woman of twenty-three. She has been the subject of angular dorsal curvature since childhood, with tubercular disease of one hip-joint: and for seven months had been completely paralysed up to the waist, with some vesical symptoms in addition. Following the practice of some American surgeons, large doses of iodides of potassium and sodium were prescribed (half a drachm every two hours), and though much distress from iodism was produced, the treatment was persevered with, and now after several weeks she is taking ten grains of the potash-salt every four hours. The result is that sensation has been completely regained, and that she is able to move her legs freely in bed.

Charcot speaks in decided terms of the value of the app
actual cautery along both sides of the projecting spinous pr
has been used in two ways, by searing the skin on each side
or by making deep incisions and cauterising the wounds.
of each the present writer would dissuade from the latter me

Of late years antiseptic surgery has ventured to deal w
from compression by the products of vertical caries. Seve
been recorded in which the laminæ have been exposed,
resected, and the carious material cleared out: and this se
has occasionally been successful. Mr Lane recently perform
tion on a lad in Guy's Hospital, with the gratifying result of
power of walking ('Brit. Med. Journ.,' April 20th, 1889).

It is probable that mercury and iodide of potassium
curing paraplegia which is due to syphilitic gummata p
growing into, the cord; and this treatment should never
any case which may possibly be of venereal origin.

Mr Horsley has published a remarkable case in which
spine in a patient of Dr Gowers, who had diagnosed the
intra-vertebral tumour pressing on the cord. The tumour
removed, and the patient was not only relieved from excruci
but was restored to power of locomotion. So brilliant a
combination of the skill of the physician in detecting and t
surgeon in removing an organic lesion can scarcely be mor
event; but even one such case encourages the most minute ef
and define organic diseases, and amply justifies the bol
measures when so directed ('Med.-Chir. Trans.,' 1888).

Spastic Paraplegia.*—Rigidity of the paralysed limbs i
feature in the later stage of segmental disease of the cord, w
or due to compression. It is only when the lowest lumb
destroyed that the legs are of necessity flaccid. In parapleg
curvature and in hysterical paraplegia they are often spasmo
more frequently, both in cases due to caries and in those of pr
they are forcibly extended. We shall find that similar contr
of a "tonic" kind, is a late result of lesions of the brain; and
the paralysed limbs become rigid soon after the paralytic str

In the cases we are now concerned with, the toni
("contractures") and rigidity are late, not early. They c
after paraplegia, and they are associated with increased refle
superficial and deep, and with the clonic contractions known
They are no doubt due to increased reflex irritability of the
and this again may be referred to the absence of the normal
fluences from the centres above the seat of lesion. The diffi
plain why their appearance is delayed; but it seems to be alw
with descending sclerosis affecting the deeper part of the mo
lateral columns, either from a focus in the encephalon, as a cl
from one in the cord, as a segmental lesion from compression.

The contraction of the limbs is in most cases obviously
their paralysis, for it generally does not show itself until
already lost power over his legs for a considerable time.

* *Synonyms.*—Spastic spinal paralysis (Erb)—Tabes dorsalis spa
(Charcot)—Primary and secondary lateral sclerosis—Spasmodic paralysis

secondary rigidity and spasm of muscles which have already lost voluntary power are probably of the same pathology, whether paraplegic, hemiplegic, or monoplegic in distribution.

There are, however, other far less common cases in which rigidity is present from the first, and, indeed, constitutes the most marked symptom, so that it may seem to be the only obstacle to the patient standing or walking. Here also the lesion is in all probability a symmetrical *primary* sclerosis of the lateral columns—the crossed pyramidal tract and especially its hinder parts—leaving the anterior grey cornua intact. Satisfactory evidence on this point was afforded by a case of Dr Morgan's, of Manchester, in which Dr Dreschfeld found sclerosis of the crossed pyramidal tracts and no other lesion, spinal or cerebral ('Transactions of the International Medical Congress of 1881;' and also 'British Medical Journal,' vol. i, p. 407). Spastic paraplegia, whether secondary or primary, is therefore anatomically a "systemic" or "columnar" disease (p. 488), the sclerotic process keeping to a definite tract of fibres.

Spasmodic paraplegia is said to occur chiefly in persons between thirty and fifty years of age, and in men more often than in women. Of Dr Morgan's seven cases only one occurred in a woman. Erb speaks of it as very frequent. In this country primary spastic paraplegia is certainly rare, while the secondary form of the affection is common. Its causes in the former case are unknown. Poisoning by syphilis and by lead have been suspected, and also by *Lathyrus cicera* in three cases reported from Italy.

Symptoms.—The lower limbs assume as a rule a position of rigid extension and adduction. The contraction may vary indefinitely in degree. At first it is only occasionally present, and is generally less marked when the patient is lying down than when he stands upright. One of the earliest symptoms of the complaint is a peculiar gait. In walking the foot is lifted with difficulty; the toes are scraped along the floor and catch against every inequality in the surface; the step is short and hesitating, and sometimes there is a peculiar hopping movement, the body being raised upon the toes at each step. Very often the attempt to walk causes a tremor in the foot and leg, which may extend to the trunk also,—a true clonus. In some cases the back is arched, and the head thrown backwards; in others the body is bent forwards over the toes by similar tonic contraction of the flexor muscles of the trunk, so that there is danger of falling, especially in descending stairs. The gait at one stage may be rolling or waddling, as carefully described by Dr Ross. The legs feel weak and heavy and are easily tired. Sometimes, if the patient sits down, his legs are thrust forward, so that his feet do not touch the ground. In more extreme cases he is confined to bed, and is perfectly helpless. His knees are tightly pressed together by spasm of the adductors, and cannot be bent from spasm of the quadriceps. Yet in primary uncomplicated cases there is no impairment of sensibility, pain is entirely absent, and the bladder and rectum perform their functions naturally. The galvanic and faradic contractility of the muscles is slightly lowered, but there is no reaction of degeneration. In cases secondary to myelitis or compression these negative characters are often wanting.

There is exalted susceptibility to "tendon-reflexes." Not only are the common movements induced by percussion of the ligamentum patellæ exaggerated, but movements can be excited through the tendons of the tibialis anticus and posticus, the biceps femoris, and even through aponeurotic structures. Ankle-clonus is present. There is sometimes, but not always,

increase of the superficial reflex movements which are excite impressions.

The *course* of spastic paraplegia is progressive, but very s a period of eight, ten, or fifteen years; sometimes it remain the same point for a long time. It may never extend abo it may at length affect the lumbar and abdominal muscles limbs. The abdomen then feels hard, and is separated from of the chest by a furrow. The fingers are, from time to nently, clenched within the palm; there is extension of the w joints, with pronation of the forearm; the arms may be fixed side of the trunk. One leg is often affected before the contraction may then spread to the arm of the same side volves the opposite leg. Occasionally the upper limbs are attacked. Erb says that it rarely happens—and only at th of the disease—that the lower limbs are rigidly flexed, in extended.

Dr Savage has observed cases of spastic paraplegia going lopment of general paralysis of the insane. We have had case of this sequence in a man under treatment in Phili the greater part of the year 1889.

According to Charcot and Erb the only real difficulty in d disseminated sclerosis in that form in which the symptoms the higher centres happen to be wanting. Charcot adds tha cases with which he used to illustrate his lecture on "spa scattered patches of sclerosis were found at the autopsy as h cerebri.

Spasmodic paralysis in children.—Dr Gee recorded in the sixteenth volumes of the 'St Bartholomew's Hospital Repo cases which he called "spastic paraplegia." They all occur and the complaint was either congenital or began in early i were eight cases, six girls and two boys. The legs were and sometimes the arms. The contractions corresponded in closely with Erb's and Charcot's descriptions. Dr Gee lay fact that handling the limbs increased the rigidity. Chlo it in all cases but one; in that instance the muscles of one c wasted, the disease having lasted eleven years.*

Seeligmüller, Erb, and Gowers have also described spa in children. In some of these cases it was of the variet Charcot as "sclérose latérale amyotrophique," in which affected muscles is present, and the anterior cornua as well columns are affected. In other cases the symptoms were cerebral rather than a spinal lesion.

During the last few years we have had two cases in Guy in a boy of three, the other in a girl of seven, both probably

Mixed forms.—Lateral sclerosis of the cord may be con may complicate, other diseases. It is, as we shall see, a fre the normal, sequel of cerebral hæmorrhage; and, as already more often seen as a sequel to transverse myelitis or segmen of the cord than as a primary affection. But beside these co

* See also Dr Gee's paper on "Hereditary Infantile Spastic Paraplegi in the twenty-fifth volume of the same 'Reports' (Jan., 1890), in which paraplegia with some muscular atrophy of the arms in a father and two

secondary spastic paraplegia, it is not unfrequently also found combined with other columnar diseases.

Thus, it not unfrequently supervenes in the later stages of insular sclerosis (v. *infra*, p. 540); it is sometimes followed by extension of the sclerotic process from the lateral columns to the anterior cornua, or perhaps more often primary atrophy of the anterior cornua is complicated by secondary descending lateral sclerosis. The result is a combination of progressive muscular atrophy with spastic paralysis (*infra*, p. 502).

Prognosis and treatment.—Erb has seen two cases of spastic paraplegia almost cured and several much relieved by galvanic currents applied to the spine. He also speaks favourably of the "cold-water cure," and of the gaseous saline baths (*Sool-baden*) of Rehme and Nauheim. He has found the nitrate of silver sometimes useful as an internal medicine, and Charcot recommends the bromides. Strychnia is likely to do harm rather than good. Conium and belladonna have been given without any good results. In some cases to be presently mentioned, physostigma has been of great service. According to Dr Ross, the galvanic current is by far the most trustworthy remedy, but Dr Gowers believes that faradism is harmful, and that galvanism has no influence in lessening the spasm or improving the strength. As he well puts it, in primary lateral sclerosis the chance of recovery and the danger to life are both small.

Clinical cases of spasmodic paraplegia.—In patients suffering from chronic paraplegia the spasmodic symptoms above described are very frequent in any large hospital; but we have had very few cases at Guy's Hospital which answer to the accounts given of primary spastic paraplegia.

In eight or nine instances contraction of the lower limbs has been apparently the primary disease; but the legs and thighs have been drawn up in a state of flexure instead of being stretched out. Any attempt to straighten the limbs has generally caused great suffering; but in one case—that of a man aged thirty-three, under Dr Wilks's care—the suspension of a weight of fourteen pounds to the right foot led to a marked diminution of the pain; the limb was bent, so that the heel almost touched the buttock, and it could not be put straight even when chloroform had been inhaled. In certain instances the extract of *Calabar bean* appeared to remove the rigidity altogether.*

These cases belong to a different clinical type from those described by Charcot, and have possibly a different pathology. Contraction of the

* The following are the cases referred to:—1. The first case in which I observed this satisfactory result was that of a boy, aged six, who was admitted on August 19th, 1874. He had been well until three weeks previously. He lay on his right side, with his legs drawn up, and unable to move them. They could be forcibly extended, but then became rigid. The expression of his face was indicative of pain, and he had complained of pain in the back of the head and over the spine. Sensation was unimpaired, but the fæces and urine were often passed into the bed. He could close his hands, but with a very feeble grasp, and sometimes they were noticed to be stiff. The diagnosis was chronic spinal meningitis, and hydrarg. c. creta, iodide of potassium, bromide of potassium, and other medicines were given without any result up to the 8th of October. At that time he began to take the extract of physostigma in doses of one sixth of a grain, gradually increased to half a grain, three times daily. Improvement quickly set in; the limbs became less rigid, and he regained the power of moving them. On November 21st cod-liver oil and steel wine were substituted for the Calabar bean. On January 9th, 1875, he left the hospital, able to walk pretty well without help.

2. In 1876 I was asked to see a boy, aged fourteen, who was under Mr Cooper Forster's care, with contraction of the thighs and legs, which had been coming on for six months. He appeared to suffer intensely from pains in the affected limbs, and screamed when they were touched. The legs and thighs were much wasted. Remembering the former case, I

legs in a *flexed* condition, apart from primary disease of the joints, may supervene in the last stage of chronic diffused myelitis, of compression-paraplegia, of infantile atrophic paralysis, and even of primary lateral sclerosis itself. It may also occur in hysterical paraplegia; but the "cadaveric" position with extended hips, knees, and ankles and adducted feet is characteristic of the clinical type of Charcot and Erb.

A well-marked case of the more common secondary form was during four years under the writer's care in Guy's Hospital, and ended in recovery. The patient, a boy of twelve, when first admitted in January, 1885, suffered from caries of the cervical and of the lower dorsal vertebræ, which afterwards produced both lumbar and psoas abscesses. There was no anæsthesia or serious disturbance of the bladder or rectum, or bedsores, or pain. The muscles were not atrophied except from want of use. When first admitted there was tonic contraction of both arms and both legs, the former flexed, with *main en griffe*, the latter extended in the cadaveric position. Knee-jerks and plantar reflexes were exaggerated, and ankle-clonus was readily elicited. Faradic contractility was unaffected. Physostigma was pushed without benefit; but after many months, as the result apparently of improvement of the vertebral disease by surgical treatment, the power of his arms gradually became almost normal, and contraction disappeared; he could move his legs well, although clonus and exaggerated reflexes remained,

prescribed the Calabar bean in doses at first moderate, but quickly increased. The symptoms at once began to subside. He was after a time transferred to my charge, and left the hospital cured.

3. On October 31st, 1876, a woman, aged forty-one, was admitted under my care with a spinal affection which had come on suddenly two months before. She first complained of pains in the leg-muscles and then of stiffness in the joints. This was followed by great weakness and wasting of the lower limbs; and then they slowly began to contract. She lay in bed with her legs strongly flexed; when they were forcibly straightened it gave her great pain. Faradic contractility was found to be much diminished, and she had partial loss of sensation; she could not pass her water. She took at first half a grain of extract of physostigma three times daily; after a week this dose was doubled, and four days later it was trebled. By this time she could move her legs to some extent. The medicine was continued for a month, and then some tincture of iron was prescribed. By December 23rd she could straighten her legs perfectly well. In the month of February, 1877, she became able to stand and to walk without help, and on March 1st she was discharged cured.

4. In 1878 a sailor, aged twenty-eight, came into the Clinical Ward under my care on May 29th. Fourteen months previously he had been on a river in South America loading timber, and had repeatedly got wet through and allowed his clothes to dry on him; he said he was well used to getting wet with salt water but not with fresh water. Four or five days after he left off this work, he one morning on waking found that his left leg was stiff and painful in the ham. As the rigidity gradually got worse he went into hospital at New Orleans, and after a time it passed off. He returned to work, but two months later, the weather having been bad, he again began to suffer from stiffness and pain in the left leg. Presently the right one also was attacked. They both became weak, and for six weeks before admission he could not walk without a stick. At times he had short attacks of convulsive twitchings in the left leg. I found the muscles of the thigh decidedly rigid on both sides, especially the left. After leaving him for a few days without treatment, I prescribed the extract of Calabar bean in doses of a quarter of a grain, afterwards increased to one grain. Improvement was not at first very striking, but at the end of thirty-four days there was hardly any rigidity left.

5. The only case that I know of in which the extract of physostigma produced any unpleasant effects is one recorded in vol. xviii of the 'Guy's Hosp. Rep.' A boy, aged ten, had been suffering for some months with paraplegia before rigidity of the legs set in. A quarter of a grain of the extract was ordered, and after a week it was increased to half a grain three times daily. By mistake he took a grain in one dose, and an hour afterwards he was blue in the face but perfectly conscious, perspiring profusely, a clear froth coming from the mouth, the pupils of natural size, the hands cold, numb, and almost powerless. He had an emetic, and in three hours he was as well as before.—C. H. F.

and his general condition greatly improved. Finally he was discharged in 1889, able to walk and use all his limbs, free from spasm, and only showing the angular curvature of his spine, and the scars of healed abscesses.

Of *primary* spastic paraplegia the writer has only seen two cases. Both patients happened to be clergymen. The one, a remarkably healthy, well-developed man of about forty, complained of gradually increasing difficulty in walking owing to "stiffness" of the legs. He could not bend his knees, and found it almost impossible to ascend a broad and shallow flight of stairs. When lying down his legs were rigidly extended. Ankle-clonus was present and the knee-jerks were increased. There was no pain except during occasional cramps or spasms in the legs. The pelvic organs were unaffected. There was no true paraplegia, for the lower limbs could be moved readily until the check of stiffness came, and the skin was normally sensitive. The muscles were of good size. Physostigma was prescribed, and he thought there was some improvement after several months, but if any it was slight. He afterwards tried massage and other plans of treatment, but the local disease went slowly on until his death by an acute attack of inflammation of the lungs.

The other case occurred in an older man—one who had been an Alpine climber and enjoyed robust health. In his case also the loss of locomotive power only resulted from stiffness of the legs. The knees were bent, the body was bowed, as if with the "chronic rheumatism" of an aged peasant, so that it was almost impossible for him to walk or to stand upright. The pelvic organs were unaffected; knee-jerks were readily produced and clonus also. As in the previous patient, the upper limbs were free. In this case also physostigma was fully tried with only doubtful benefit, but under repeated courses of nitrate of silver decided improvement took place, whether in consequence or not must be doubtful. At present he enjoys good health, but the spastic paraplegia remains.

AFFECTIONS OF THE SPINAL MEMBRANES.—This is a convenient place for a brief mention of certain lesions of the meninges of the cord which do not produce by the local action any constant sequence of symptoms so as to enable us to class them with other causes of "compression-paraplegia" or of atrophic paralysis. They are not segmental, but indiscriminate or diffuse, in their localisation.

Spinal meningeal hæmorrhage.—Except as the result of severe injuries, which come under the care of the surgeon, this is a very rare lesion. The causes assigned to it by authors are violent bodily efforts, convulsions, purpura, and the suppression of a hæmorrhoidal or a menstrual flux. If a diagnosis is to be made during life, it must be based upon the sudden development of the symptoms, and upon the co-existence of symptoms of irritation of the cord and its nerves with those of compression of these structures. Certain cases, in which there have been severe pains in the back and limbs, rigidity of the spine, and partial paraplegia, have been attributed to the effusion of blood into the spinal membranes; and this explanation has not been regarded as invalidated when recovery has slowly taken place. But it would seem that in other cases in which meningeal hæmorrhage has proved fatal, so that it has been demonstrated in the deadhouse, the symptoms have not been equally characteristic.

Tumours of the spinal meninges have been already described in their clinical place as causes of paraplegia by compression of the cord (p. 474).

Acute spinal meningitis.—Inflammation of the vertebral dura mater or spinal pachymeningitis is a frequent result of caries of the vertebræ, and forms part of the anatomy of paraplegia from compression.

Secondary inflammation of the spinal membranes may result from the invasion of a deep bedsore over the sacrum. In some cases the vertebral canal is actually opened by sloughing of the fibrous membrane which closes its lower end; in others, perhaps, the morbid process extends inwards along the posterior sacral foramina. At least four instances of this kind have been observed at Guy's Hospital, and probably several have been overlooked. Pus has generally been diffused among the nerves of the cauda equina, and beneath the spinal arachnoid, up to the base of the brain. In one case the inflammatory products lay outside the theca vertebralis. No distinctive symptoms seem to have been noted in the cases in question.

Other forms of acute spinal meningitis accompany myelitis and traumatic, tubercular, epidemic, and other forms of cerebral meningitis, which will be fully described hereafter. Hæmorrhagic spinal pachymeningitis is also occasionally found in association with a similar affection of the cerebral dura mater. Primary acute inflammation of the spinal membranes does not appear to exist.

Chronic spinal meningitis.—Certain local forms of meningitis, occurring chiefly in association with syphilitic gummata, or with caries of the vertebræ, have been already mentioned; but it sometimes happens that the whole of the spinal membranes are found matted together and thickened round a cord which appears to be perfectly healthy. In 1878 a case of this kind was found in an old woman of seventy, who had for about a year been suffering from slowly advancing paraplegia. She had no spasms or rigidity of the legs; she lay helpless in her bed, but the lower limbs still retained some power of sensation, so that she could distinguish heat from cold. She complained of pains in the legs, apparently not severe, and in the lower part of the abdomen. After death the visceral arachnoid, when it had been stripped off the inner surface of the dura mater, was found to be as thick as a sheet of writing-paper. In the lower part of the dorsal region the adhesion of the membranes to the cord was unusually close for about an inch and a half of its length. The thickening extended upwards around the pons, and to the under surface of the cerebellum, so that the cerebro-spinal aperture was closed. Dr Frederick Taylor (under whose care the patient was during life, could detect no morbid change in the cord itself except that the fibrous septa entering it from the surface appeared thicker than usual.

A similar case was recorded by Gull in his well-known paper in the 'Guy's Hospital Reports' for 1856 (Case 7). In that instance rigid flexion of the legs was a marked symptom, and they were also affected with frequent spasms. But may not this have been a case of transverse myelitis in the upper dorsal region, with exalted excitability of the cord below from secondary descending lateral sclerosis?

Charcot first described a form of primary inflammation of the spinal dura mater, producing pain and partial paralysis, in one or the other arm—*Pachymeningitis cervicalis hypertrophica.* The origin of the ulnar nerve seems most often to suffer; and, after paralysis with pain and atrophy of the arms, there follows secondary spastic paraplegia of the legs. We shall meet this affection again in the next chapter (p. 503).

Some modern German writers describe chronic diffused spinal meningitis as of frequent occurrence, and as being often recognised during life without

much difficulty. It is characterised, says Erb, at first by pain and stiffness in the back, a troublesome "girdle sensation," a feeling of weight in the legs, numbness, tingling, tearing, or dragging pains in them, and cutaneous hyperæsthesia. After a time partial paraplegia develops itself, which varies in degree from day to day. Erb supposes that such fluctuations depend upon alterations in the quantity of fluid effusion within the spinal canal, or perhaps in the amount of blood in the vessels about the cord. There is generally but little impairment of sensibility; and, according to Braun, the sphincters retain their functions. Erb speaks of the morbid change in the membranes as being usually but little marked; they are more or less thickened, opaque, and adherent to one another; and there is an excess of fluid round the cord. Cold is said to be the chief exciting cause of this affection; it has often been observed in soldiers after severe fatigue and exposure during a campaign. It commonly begins acutely, and afterwards passes into a chronic form. In many cases it becomes complicated with myelitis, and the peripheral part of the cord beneath the pia mater sometimes undergoes sclerosis in an annular form.

How far one is justified in maintaining an anatomical diagnosis in cases of this kind is doubtful. But the clinical differentiation of them appears to be important, as regards prognosis and treatment.

Erb says that many prove fatal by bedsores, cystitis, or exhaustion; but he admits that others recover more or less completely; and Braun declares that the prognosis is favourable if the acute stage, in spite of the increasing paralysis, be treated at first with local bleeding and cold applications. Afterwards, when paralytic symptoms alone remain, the patient should begin a steady course of hot brine baths, those of Rehme or Nauheim being preferred, which closely resemble the saline waters of Droitwich. Erb endorses Braun's statements, and declares that when there is no myelitis, baths at high temperatures (98°—108° Fahr.) are well borne, and that the reputation of curing spinal paralysis in general which is possessed by hot springs is really derived from their success in this particular class of cases. Hitzig and Erb have found benefit from passing galvanic currents along the spine. Iodide of potassium is given; but good food, wine, and tonics are recommended as important auxiliaries in the treatment.

PARALYSIS ATTENDED WITH MUSCULAR WASTING

> Weak shoulders, overcome with burdening grief,
> And pithless arms, like to a withered vine,
> That droops his sapless branches to the ground.—K. HENRY VI, part i.

General characters of this group of diseases of the cord.
SUDDEN ATROPHIC SPINAL PARALYSIS—*In infants—Its course, sequelæ, anatomy, and treatment—In adults.*
SUBACUTE ATROPHIC SPINAL PARALYSIS—*Its course and anatomy.*
CHRONIC ATROPHIC PARALYSIS—PROGRESSIVE MUSCULAR ATROPHY—*Symptoms—Pathology—Primary and secondary forms—Spastic atrophic paralysis—Pachymeningitis cervicalis—Diagnosis—Treatment.*
Bulbar paralysis—Progressive muscular atrophy in children—Hereditary form.
PSEUDO-HYPERTROPHIC PARALYSIS—*Primary (myopathic) muscular atrophy.*
LEAD PALSY—*Distribution—Diagnosis—Tremors—Treatment.*
Summary of other results of plumbism—Effects upon the nervous system of other metallic poisons—Mercurial tremors, brassfounders' ague, &c.

THE affections discussed in the last chapter are all, we found, accompanied by paraplegia; and when their anatomical seat is ascertained, it is a lesion of the cord which is "indiscriminate," *i. e.* whether it affects the whole organ, as diffuse myelitis, or one or more segments only, as transverse myelitis and paraplegia from compression, or the surface, as meningo-myelitis, or the deepest parts, as peri-ependymal myelitis—in all these varieties of disease, the cord is affected indiscriminately throughout the diseased segments.

The only exception was spastic paraplegia, where the lesion is in the most typical cases limited to the crossed pyramidal tracts of the cord.

There are several other spinal diseases which agree with spastic paraplegia in this particular: they affect particular tracts of the cord, and as a rule do not transgress these anatomical limits. Moreover, they are chronic in their course; they are much less dangerous than acute myelitis or paraplegia from compression; and the anatomical process which presumably causes their symptoms is the same—chronic interstitial myelitis, *i. e.* grey degeneration or sclerosis. They are called by German pathologists "system-diseases," an uncouth and obscure phrase in English. "*Longitudinal*, as opposed to *transverse*, chronic myelitis" would express their range. Perhaps the term "columnar" sclerosis may serve to denote the characteristic limitation of the lesion to the longitudinal columns or tracts of the cord.*

* The recognition of the physiological division of the white substance of the cord into distinct tracts of ascending and descending fibres is due to Türck, of Vienna, who in 1851 and 1853 described them as regions of secondary degeneration after lesions of the brain and cord. Flechsig subsequently showed that the same distinction is observable in development, the fibres of the anterior column and anterior root-zone acquiring their myelin first, those of Goll's tract and the direct cerebellar tract next, and the crossed pyramidal tracts last, not indeed until some weeks after birth. (See Dr Bramwell's excellent plates, 19, 20, 21.)

The longitudinal, columnar, or systemic scleroses which have been as yet observed are the following:

1. Of the lateral columns (crossed pyramidal tract) rarely primary, often secondary; descending from a primary lesion of the motor tract in the brain or cord. The corresponding disease is that described in the last chapter as Spastic Paraplegia.

2. Of the antero-median column of Türck. This is always secondary and descending. It does not correspond to any known clinical group of symptoms.

3. Of the posterior columns of Burdach (*fasciculi cuneati*). This is usually primary, and corresponds with the clinical malady which will be described in the following chapter as Tabes Dorsalis.

4. Of the postero-median columns of Goll. This is almost always a secondary lesion, often combined with the last in tabes, sometimes appearing as an ascending degeneration from a transverse lesion. No constant symptoms are known to answer to it.

5. Of the direct cerebellar tract. This is always secondary and ascending. It also does not give rise to any ascertained malady. Secondary sclerosis of the "mixed tract" outside the anterior root-zone (antero-lateral column) has been observed by Dr Gowers and by Dr Tooth. It occurred as an ascending degeneration after fracture of the spine. Its pathological significance is as yet unknown ('St Barth. Hosp. Reports,' vol. xxi).

6. Of the large motor and trophic ganglion-cells of the anterior cornua. This is sometimes an acute, sometimes a chronic process, and is often confined to a single segment or to some part of the anterior cornu in a single segment. The cells which form the posterior vesicular column of Lockhart Clarke appear also to be subject to a destructive process which affects them alone.

In the present chapter we deal with affections for the most part identified with the last of these "systemic" scleroses. But they are also united by a common symptom, that of marked and rapid atrophy of the paralysed muscles, as well as negatively by the absence of anæsthesia, trophic changes in the skin, or affections of the bladder and other pelvic organs.

They may for the present be called in common Atrophic Spinal Paralyses.

ACUTE ATROPHIC PARALYSIS.*—In the last century this affection was described by Underwood in his treatise on the 'Diseases of Children' (1784). Jacob von Heine published a monograph on it in 1840, and this writer and afterwards Duchenne expressed the opinion that the cause of infantile paralysis was a lesion of the spinal cord. When autopsies failed to reveal any morbid change, MM. Rilliet and Barthez proposed the name of "paralysie essentielle;" but since 1863 more refined histological methods have shown that the anterior cornua of the cord are diseased. Accordingly the affection is now known as "poliomyelitis † anterior acuta." The old name "infantile paralysis" is not wide enough, for it is now ascertained that the same disease occasionally occurs in adults.

Onset and course.—A child who is about to suffer from acute atrophic

* *Syn.*—Acute amyotrophic spinal paralysis—Poliomyelitis anterior acutissima (Kussmaul)—Anterior cornual myelitis (Gowers)—Infantile paralysis—Wasting palsy of children.

† *I. e.* myelitis affecting the *grey* matter (πολιός). Charcot proposed Tephromyelitis, τεφρός (ash-coloured, cinereous); and Vulpian, Spodiomyelitis (σπόδιος, also ash-coloured, like the last, but an epithet only used in poetry).

paralysis is sometimes apparently well until the loss of power appears. But more often he falls ill with febrile disturbance, oppression, or drowsiness. Sometimes the earliest symptom is an epileptiform convulsion, or there may be spasmodic twitchings of the face or limbs, grinding of the teeth, and rolling of the eyes. The fever usually lasts a day or two, but it may pass off in a few hours, or may continue for a week or longer. Convulsions may be repeated during twenty-four or forty-eight hours.

When the child seems to be getting better, and is being washed or dressed—or perhaps when it first attempts to stand—the mother or nurse finds that one or more of the limbs is powerless. He has gone to sleep at night with full use of his arms and legs, and in the morning a limb may hang flaccid and motionless. The affection may be a monoplegia, the paralysed limb being most often the right leg, but sometimes an arm; or it may be a paraplegia, or a hemiplegia, or a crossed paralysis of one arm and the opposite leg. Sometimes there is loss of power of the trunk or of all four limbs, and sometimes they are attacked in succession at intervals of a few hours.

Occasionally the initial fever is very slight; it may perhaps be absent, and the paralysis be the first symptom that the child is unwell. But, as might be anticipated, there is no thermometric evidence of this.

It is characteristic of acute atrophic paralysis that the affected parts are completely relaxed. No reflex movements can be elicited, whether by stimulation of the skin or of the tendons. On testing the muscles with faradic currents, one finds that their contractility becomes markedly diminished within the first four or five days, and that it is entirely extinguished at the end of a week or a fortnight, but the susceptibility to galvanic currents is increased. In other words, the reaction of degeneration is present. Rapid and early wasting of the muscles takes place. Formerly they were said to undergo fatty degeneration, but the histological changes are really of a different kind, and correspond exactly with those described at p. 401 as following lesions of the nerve-trunks. The surface of the paralysed limbs is cold, and they are pale or of a livid bluish tint; the volume of the pulse in them is said by Volkmann to be diminished, and Charcot states that after death the main blood-vessels are found remarkably reduced in size.

As a rule, no impairment of sensation can be detected: the child cries lustily as soon as the poles of a galvanic battery are applied. The functions of the bladder and rectum are unaffected, and bed-sores do not appear.

Unlike most forms of paralysis, that now under consideration shows no tendency to gradual increase. The limbs are often attacked one after another, but the loss of power in each of them, although not absolutely sudden, becomes complete within a few hours. Erb says that the whole development of the disease, in successive outbreaks, may be protracted over a week, but it generally occupies much less time.

No long period elapses before recovery begins to take place; sometimes this is observed within a few days, more often in the course of two or three weeks. Even muscles which have to some extent undergone wasting regain their size and strength. If the arms and the upper part of the trunk are affected they are particularly likely to get quickly well. In the course of a few months the disease may entirely disappear, especially when it is confined to a single limb. Some writers have disputed the claim of such a "temporary paralysis" to be classed with those forms in which there is perma-

nent loss of power; but most likely it is the same disease. These favourable cases are, however, very exceptional. Usually the recovery is incomplete. It goes on for a month or eight weeks, so that the parents of the child cherish the most pleasing hopes; but after this the progress becomes slower, and at the end of six or nine months it ceases for ever.

The condition of the little patient at this time is very variable. One limb or two may be powerless and shrunken throughout, or the affection may have become limited to certain parts of an arm or of a leg. As a rule, the distal parts are more apt to suffer than those nearer the trunk. Volkmann, however, speaks of one patient as having a muscular forearm attached to a humerus like a stick, and Erb says that the deltoid sometimes suffers alone. Nay, the acromial part may escape, while the rest wastes away. Certain muscles exhibit a peculiar independence, some by remaining paralysed when those near them get well, some by recovering while the others are undergoing atrophy. The following usually escape: the *supinator longus* of the forearm, the *tensor fasciæ latæ* and *sartorius* of the thigh; while the *deltoid*, *serratus magnus*, *quadriceps extensor*, *gastrocnemius*, and *peronæi* are apt to suffer; or the last-mentioned may suffer alone, and the extensors in front of the leg escape.* When a muscle wastes, the destruction of its fibres is not necessarily made manifest by a corresponding diminution of its mass, for interstitial development of adipose tissue often takes place, which may entirely conceal it. Erb says that this is particularly apt to occur in young children, and that a muscle may actually appear bigger than in the opposite healthy limb, although it scarcely contains any contractile fibres.

Another peculiarity of parts affected with this disease is that they do not grow at the natural rate. Hence a paralysed limb is commonly shortened to the extent of an inch or an inch and a half, and sometimes by as much as eight inches. Even the corresponding half of the pelvis may remain undeveloped. The bones are thinner and more spongy and their processes smaller than on the healthy side. Remembering Hilton's observations with regard to the arrest of growth in the feet of patients suffering from disuse of the hip or knee-joint, one might be disposed to refer all these conditions to disuse, but this is certainly not the case. The disused muscles of hemiplegia are small and flaccid, but they do not atrophy like these. Moreover, the muscles waste even when the paralysis is partial, so that the child halts very little and is on its legs all day. Volkmann saw persistent failure of development follow four or five cases of "temporary paralysis," in which all the muscles recovered.

But the most important remote effects of acute atrophic paralysis in children are the contractions and deformities which are so commonly observed. Twenty years ago, Dr Wilks insisted on the fact that club-foot, when not congenital, is the result of paralysis, and not primarily a spasmodic affection; and this view, which was opposed by "orthopædic" surgeons, is now generally accepted by pathologists. Until recently, the accepted explanation was that contraction was generally the result of the unopposed action of the antagonists of the muscles paralysed. But Hüter and Volkmann have shown that when there is loss of power in several

* On this curious distribution of paralysis, seen also in the chronic form of atrophic paralysis and in lead palsy, light is thrown by the interesting experimental evidence, obtained by Professors Ferrier and Yeo, on the motor roots of the nerves of the extremities in the ape ('Proc. Royal Soc.,' March, 1881).

groups of muscles, it not infrequently happens that those on which the paralysis is most marked lie *within* the open angle formed by the displacement of the bones at a joint. For example, a *genu recurvatum* with the concavity directed forwards may appear when the extensor muscle of the knee is powerless, or a *talipes equino-varus* when no contractions can be obtained by galvanising the calf of the leg. Volkmann explains these facts by showing that they are in part due to the influence of gravitation; thus the weight of the foot causes the toe to point downwards when the limb hangs in the air. But another important factor is the gradual strain on a joint, in efforts made by the patient to employ the weight of the body as a propelling force. Volkmann points out that when a person whose thigh-muscles are paralysed learns to walk without crutches, he swings the trunk forwards so as to extend the knee as fully as possible. The result is that after a time the ligaments behind the joint yield, and the knee becomes bent the wrong way. The articulations in such paralysed limbs often become exceedingly loose. Lastly, a subordinate element in the causation of deformities is the contraction of the new connective tissue developed in the interstices of the wasted muscles, a result which has been compared to "sclerosis" of the cord and "cirrhosis" of the liver.

Distribution.—Putting together sixty-two cases collected by M. Duchenne, fils, thirty-two of Dr West's patients, and sixteen of Dr Goodhart's, we find that of the total 110 patients, the right leg only was paralysed in thirty-six (a third), the left only in twelve, both legs in nineteen (paraplegia), one or other arm in seventeen (making sixty-five cases of monoplegia or more than half); hemiplegia occurred in twelve (right five, left five, crossed two), facial palsy in seven (all observed by Dr West); while in five cases, three of the limbs, all four, or the muscles of the trunk were paralysed.

Morbid anatomy.—Cornil, in 1863, was the first to record an autopsy in which changes were found in the cord; but the body was that of a woman who had been paralysed when a child, nearly half a century before.

The earliest autopsy after the attack which has hitherto been made was by Royer and Damaschino, in the case of a boy who died of scarlet fever within two months from the onset of the paralysis. The results of this, and of a few other observations made within two years of the attack, are summed up by Erb as follows:—To the naked eye there is generally no perceptible alteration. Sometimes the cord seems rather tough, and its antero-lateral columns are a little shrunken at the level of the lumbar or of the cervical enlargement. Its structure may be slightly blurred. The forepart of the grey matter may be whitish or pink and softened, or even reduced in size; the corresponding motor nerve-roots may be atrophic, grey, and translucent. The microscope reveals an extensive area of morbid change, perhaps an inch or more in length, in this region. It is sometimes confined to one anterior grey cornu, sometimes it involves both of them, according to the distribution of the paralytic symptoms during life. Granule-masses are generally present in abundance; there is nuclear overgrowth, the vessels are dilated and full of blood—in other words, there is an *inflammatory softening*. But the most striking change is a more or less complete disappearance of the large multipolar nerve-cells, many of them that still remain being in various stages of atrophy. The nerve-fibres also have undergone destruction, even to their axis-cylinders. At the periphery of the affected area the nuclei are often massed together in large numbers, so as to form a kind of capsule, but slight diffused lesions are discoverable

throughout the grey matter of a large part of the cord, generally in the whole length of the dorsal region. In the anterior and lateral white columns there is little change to be seen; sometimes a slight degree of sclerosis, thickening of the trabeculæ, atrophy of a few of the nerve-fibres, or a few scattered granule-masses. The anterior roots display all the appearances of "degenerative atrophy."

When many years have elapsed since the onset of the paralysis the appearances are far more marked. The shrinking and atrophy of the anterior part of the cord is now evident on the cut surface, especially when the affection is one-sided, in which case the symmetry of the two halves is lost. The anterior and lateral columns may be obviously grey in tint, and translucent as compared with the posterior columns. The anterior cornu is less deeply coloured by carmine, because the protoplasm and nuclei of the motor cells are gone, but the adjacent white substance is more so because the myelin is gone, and Deiters' cells and even fibrous tissue stain better than normal nerve-fibres. Under the microscope the diseased area in the lumbar or the cervical enlargement is seen to consist almost entirely of a delicate connective tissue, containing an immense number of corpora amylacea, but now no granule-masses. No normal ganglion-cells or nerve-fibres are at this stage to be seen; any remnants of cells are shrunken and pigmented. The anterior cornu often contains the small branched spider-cells of Deiters.

That a local affection of the spinal grey matter should set up pyrexia is remarkable. There may be a question as to whether the myelitis is not at first diffused, and whether it does not clear up and subside throughout the greater part of the cord, while one particular region, to which it becomes limited, undergoes entire destruction.

It has not yet been decided whether the starting-point of the affection is in the multipolar cells or in the neuroglia of the anterior cornua. Charcot insists that the fact of its strict limitation to certain regions of the cord is strongly in favour of the former view; he also says that at some points the cells may be found diseased while the connective tissue remains all but normal. Erb thinks that perhaps both tissues become simultaneously diseased. The muscles are diminished in volume, and their fibres are replaced by fatty or by fibrous tissue.

Ætiology.—With regard to the *causes* of acute atrophic paralysis very little is as yet known. It is far more common in children between one and three years of age than in those who are older, but it sometimes develops itself in infants from six to twelve months old. Duchenne has recorded cases occurring in children of twelve days and one month, and the disorder may occur at any age up to puberty, so that it is not separated by any absolute line of age from the atrophic spinal paralysis of adults.

The two sexes appear to be equally liable to infantile palsy. Whether teething plays any part in its ætiology is very doubtful. Wharton Sinkler, an American observer, met with it much more often between the months of May and September than during the rest of the year, the proportion being forty-seven cases to ten; and this is confirmed by Ross, Gowers, and other observers. In July and August Dr Sinkler recorded seventy-seven cases out of 149, and in the same months Dr W. H. Barlow, of Manchester, forty-eight out of 111. The so-called neuropathic family tendency seems seldom to be present. Duchenne says that he has never seen a case of this affection befalling two children in the same family, and the few cases that have

been published are probably mere coincidences. Those who are attacked are commonly neither rachitic nor scrofulous nor syphilitic, but robust and healthy. Sometimes it is directly set up by a chill, at least Erb says that this has been demonstrated beyond dispute (?). Sometimes it arises during convalescence from some acute disease, such as scarlet fever, modified smallpox, or typhoid fever.

The *diagnosis* is seldom difficult, if one keeps in mind the clinical history and features of the disease. One must remember that other forms of paralysis may occur in childhood; perhaps that which is most likely to cause a mistake is a peripheral paralysis of the brachial plexus from sleeping on the side, or from pressure by a band fastened tightly round the arm. In the case of an infant brought by a person ignorant of the circumstances it may be impossible to come to a positive conclusion.

Prognosis.—So far as is known, anterior cornual myelitis is never fatal, whether in a child or in an adult; but complete recovery is rare. When a child first comes under observation at an advanced stage, with the muscles already wasted, one is apt to think that the issue would have been better had treatment been begun earlier. But if it is placed under treatment from the commencement there is great difficulty in saying how far any improvement that occurs is attributable to the remedial measures that may have been adopted.

Treatment.—Next to keeping the palsied limbs warm and promoting their circulation, the application of galvanism to the spine seems to be the most important thing. The poles must be large, and a current of moderate strength may be passed for one or two minutes at a time. Afterwards the paralysed muscles may be galvanised in turn. Faradisation may also sometimes be useful if the muscles have not lost their susceptibility to induced currents. Even when several years have elapsed one must never hastily conclude that electrical treatment will be fruitless. It now and then happens that a few applications produce extraordinary effects, and often, by persevering for several weeks or even for months, one at length attains considerable success. In one case of the author's the internal administration of strychnia was, beyond dispute, the means of restoring to a child the power of standing. Shampooing and friction with stimulating liniments may sometimes be of service. Dr Angel Money observes that massage well carried out is far more useful than electricity. Nourishing food, cod-liver oil, and tonics are matters of course. For the avoidance of the different forms of contracture recourse must be had to mechanical apparatus. Volkmann says that drawing up of the limb may be prevented by fastening the feet every night on a splint with a flannel roller, and carrying a strip of plaster across to the leg.

Acute atrophic paralysis in an adult develops itself in most respects as in infants. Meyer, of Berlin, is said to have been the first who, in 1868, recorded two cases of this kind after measles. Duchenne, Erb, and many others have since written upon the subject. The disease seems never to set in with epileptiform convulsions; but there is fever of greater or less intensity, with headache, drowsiness, and occasionally delirium. After a single night, or at the end of a few days, one or more of the limbs become paralysed, and sometimes there is a transitory failure of the bladder. There is not the slightest impairment of sensibility, the muscles are perfectly relaxed, and their susceptibility to reflex stimuli is lost. Their

electrical reactions are like those of the same disease in children. The ætiology is equally unknown, but it is much more rare in women than in men. Recovery may either be complete or partial; in the latter case the affected parts become contracted and deformed, but not to the same extent as at an early period of life. Of course, there cannot afterwards be any difference in the length of the limbs on the two sides of the body. In the only marked case which has come under the writer's care, the patient, a healthy railway servant, aged about twenty-five, gradually but completely recovered.

CHRONIC AND SUBACUTE ATROPHIC SPINAL PARALYSIS.*—Within the last few years writers on spinal diseases have recognised as a distinct affection a form of paralysis which was first described by Duchenne in 1849 and 1853 under the name of *paralysie générale spinale antérieure subaigue.* He thought that it was probably due to an atrophy of the cells of the anterior grey cornua; and as recent observations have shown that there is a chronic inflammatory change in this part of the cord, Erb proposes to call the disease *poliomyelitis anterior subacuta et chronica.* Perhaps the most suitable designation is "chronic diffused atrophic paralysis," which at once distinguishes it from the "*acute* atrophic paralysis" already described, and also from the *limited* forms of paralysis which are usually known by the title of "progressive muscular atrophy."

Course.—This disorder generally begins in the lower limbs. The patient first experiences a sense of weakness in one or both of his legs, especially in going upstairs, or if he attempts to walk far. At the end of a few days, or in the course of some weeks, this develops itself into actual paralysis, so that he is obliged to keep his bed. Before long the upper limbs are affected, especially the fingers and wrists. The muscles of the trunk also become powerless, so that he cannot sit up, and has difficulty in coughing or sneezing, and in defæcation.

The paralysed muscles are absolutely lax and flaccid. No reflex movements can be excited in them by irritation of the skin or of the tendons. They very rapidly waste, so that with a measuring tape one can follow the loss of substance in the calves or in the thighs from week to week, until the bones seem to be covered only by the integuments. When electrical tests are applied, typical reaction of degeneration is found to be present. Duchenne long ago discovered that susceptibility to faradic currents is extinguished. Others have shown that they react well to interrupted galvanism, that A.C.C. is more marked than C.C.C., and that the contractions are slow and tonic in character.

So far there is nothing in the symptoms of chronic diffused atrophic paralysis which is distinctive of this affection rather than of myelitis, spreading upwards through the whole substance of the cord from its lower end. What shows that the lesion is limited to the anterior cornua is the absence of anæsthesia, the retention of power over the bladder and rectum, and the fact that there is no tendency to the formation of bedsores. Erb, indeed, says that pains in the back and slight paræsthesia in the limbs are not uncommonly present, and that even before the paralysis has developed itself; but on testing the cutaneous sensibility one finds it either perfect, or only very slightly blunted. At the commencement there may be a little febrile disturbance, with headache and digestive disorder, but these quickly

* Chronic myelopathic atrophic paralysis of adults—Poliomyelitis anterior chronica.

pass off and the patient afterwards feels quite well, and eats, drinks, and sleeps as usual.

The further progress of the disease varies in different cases. Generally it remains for a certain time stationary; that is, it ceases to spread upwards, or to extend to fresh sets of muscles, although those already affected go on wasting. Erb says that sometimes at this period the joints become slightly contracted. But at last, after several weeks, a gradual improvement begins to be observed; the arms regain movement, and afterwards the legs. All this takes place very slowly; months pass before the patient can feed himself, or write, or walk. Sometimes he ultimately becomes as muscular and as active as before, but sometimes some particular muscles remain wasted for the rest of his life; according to Erb this is especially apt to be the case with the peronei. As recovery goes on, the electric contractility of the muscles gradually reappears.

On the other hand, there are cases in which no change for the better occurs, but in which the paralysis extends upwards to affect the face and the tongue and to interfere with deglutition and with respiration. In this way the disease may terminate fatally from one to four years after it commencement. It does not appear that there are any special indications to direct a prognosis at an early period of the case.

Histology.—Up to the present time only two autopsies appear to have been made in which the cord was examined by the modern methods. One was recorded by MM. Cornil and Lépine, the other by Dr Webber.* In each instance the multipolar nerve-cells in the anterior grey cornua had undergone a more or less complete destruction; there were evidences of inflammatory changes in the presence of compound granule-masses, the proliferation of the cells of the neuroglia, the accumulation of nuclei round the blood-vessels, and the like. In the antero-lateral columns there was a certain degree of sclerosis or degeneration of nerve-fibres. The motor nerve-roots and the muscles were in a state of extreme atrophy, so that the morbid anatomy of the disease appears to correspond exactly with what might have been anticipated on theoretical grounds.

With regard to the *ætiology* of chronic diffused atrophic paralysis nothing is as yet known. It is a rare affection, most apt to occur in persons between the ages of thirty and fifty; but in this, while differing from acute atrophic paralysis, it resembles several other spinal affections. It has sometimes been ascribed to cold or to an injury of the back, sometimes to intemperance or to sexual excesses.

The *diagnosis* is, in well-marked cases, easy. It is separated from ascending myelitis by the absence of anæsthesia and of pelvic symptoms; from acute atrophic paralysis of adults by its gradual instead of sudden advent; from progressive muscular atrophy by the paralysis preceding instead of resulting from the muscular atrophy, and by its better prognosis; from multiple peripheral neuritis by the absence of tenderness and also of anæsthesia, and of the peculiar distribution of that affection. Chronic atrophic paralysis closely resembles that which occurs in persons poisoned by lead. Indeed, if the latter is dependent on the same change in the anterior cornua, we ought to enumerate chronic plumbism among the causes of the disease now under consideration. But, as we shall see, there are reasons for believing that lead palsy is due to peripheral neuritis.

The *treatment* of the disease appears to be that of chronic myelitis in

* 'Trans. American Neurological Association,' vol. i, p. 45.

general; the chief remedies being iodide of potassium, ergot, and the application of galvanism to the spine. The paralysis is not always progressive, and recovery has been observed.

PROGRESSIVE MUSCULAR ATROPHY.*—In 1850 a French physician, Aran, described under the name of "*atrophie musculaire progressive*," an affection which he believed had been overlooked by previous writers. Only a few instances had previously been recorded by Sir Charles Bell, Abercrombie, and Romberg. Three years later Cruveilhier wrote upon the same subject, in 1853, when he published his classical case of the athlete Lecomte, whose arms wasted muscle by muscle, then his legs, and finally his intercostal muscles, so that he died from want of power to breathe. The preparation from this case is preserved in the Musée Dupuytren. Sir Wm. Roberts, of Manchester, published a monograph in 1858. Next to infantile palsy, it is the most common of all the diseases described in this chapter, but even this is comparatively rare.

Distribution.—In the large majority of instances the upper limbs are first attacked. Aran found that this was the case in nine among eleven patients, and that in seven of them the right arm was that in which the disease began. The proportion cannot be fixed with accuracy, because it is doubtful whether all the cases in which the wasting is first observed in the legs really belong to the same category. Thus, whereas Duchenne says that he met with only two such cases out of 159, Hammond, of New York, speaks of having had eight in a total of twenty-five. Duchenne himself saw one patient in whom the disease began in the *sacro-lumbales*, and one in whom the *pectorales*, the *trapezii*, and *latissimi dorsi* were wasted before the arms became involved. The apparently capricious way in which the atrophy is sometimes distributed is shown by another of this writer's cases, in which, even at an advanced stage, no muscles of the upper limbs except the *supinatores longi* were attacked. Conversely, Trousseau mentions a patient of Bretonneau's, an old lady, who retained power over none of her muscles save those of the right index finger. She could not speak, but with her finger she picked out of a heap of letters those which she required to form words and sentences, and in this way she made her will.

As a rule progressive muscular atrophy begins in the small muscles of the hand, and especially in the ball of the thumb; before passing to the forearm it commonly spreads from the thenar to the hypothenar muscles, and also to the *lumbricales* and *interossei*. Sometimes it appears first of all in the *deltoid* and other scapular muscles. It is remarkable that when the disease attacks the *trapezius* it always leaves the anterior part of the muscle uninjured; and that the *triceps* usually escapes, even when all the other muscles in the arm have wasted away. The patient's arm then hangs helplessly by his side, he cannot raise it by any muscular effort; if he wishes to lay hold of anything he has to swing it forward with a jerk until his fingers are brought into contact with it. His hand often presents the "griffin" or "bird-claw" deformity (p. 398) from paralysis of the *interossei*. His power of prehension is greatly diminished, especially when the *opponens pollicis* is atrophied. Instead of being able to grasp an object between

* *Synonyms.*—Cruveilhier's paralysis—Wasting palsy—Amyotrophie essentielle or protopathique of Charcot (as distinguished from amyotrophic lateral sclerosis, p. 503)—Poliomyelitis anterior progressiva longissima—Chronic spinal (as distinguished from myopathic) muscular atrophy.

the fingers and the thumb, he has to fix it as well as he can within the hook-like concavity formed by the fingers alone.

Whatever may be the starting-point of the disease, it in most cases soon afterwards shows itself in the corresponding muscles on the other side of the body. But sometimes the symmetry of its distribution is very incomplete; so that, for instance, one forearm and the opposite upper arm may be principally affected. Having spread more or less generally over the upper limbs it at length passes to the neck and to the trunk; the lower limbs almost always remain intact until a very late period. But we have seen that exceptional cases are on record of the atrophy beginning in the legs. The face is seldom or never affected.

Symptoms.—The loss of substance in the muscles is in most cases visible through the integuments, and all the more because the subcutaneous fat disappears, and the skin itself becomes thin. The ball of the thumb is flat, and there is an obvious hollowing of the metacarpal spaces, especially of that which lies between the thumb and the forefinger; or the shoulder has lost its fulness, the bones forming sharp angular prominences; or the normal rounded curves of the forearm are replaced by shallow depressions. But in stout persons the wasting is not always thus easily to be detected by the eye. It is not merely concealed by the overlying fat; there is often an actual new growth of adipose tissue in the interstices of the affected muscles, as in infantile paralysis; and since at the same time a tough fibrous material is present, the part sometimes feels as firm and fleshy as in the natural state. The application of a faradic current may at once clear up all doubt, if the disease is in a sufficiently advanced stage; or a minute portion of muscular tissue may be removed for microscopical examination by Middeldorpff's "harpoon," or by Duchenne's "emporte-pièce."

The weakness in the affected muscles is increased by fatigue, and to a remarkable extent by cold. Some patients tell us that when warm in bed after a night's rest, their hands felt as strong as ever. During the day the hands and fingers often look pale and blue, and they are cold to the touch. Jaccoud and others have taken thermometric observations, from which it appears that the temperature is notably lower on the side on which the muscles are the more wasted; it is said that a difference can sometimes be detected even between the two axillæ. Sometimes there is an excessive secretion of sweat. All these symptoms are referred by Leyden to disturbance of the vaso-motor nerves. In several instances a contracted state of the pupils is recorded as having been noticed.

A symptom on which some writers have laid great stress is the occurrence of transient "fibrillary tremors," or slight quivering movements limited to particular fasciculi of the muscles involved in the disease. They may either be spontaneous, occurring when the part is perfectly at rest, or one may elicit them by giving the skin a smart tap with the finger. All observers now admit that so far from being pathognomonic of any one affection, they may be seen under various circumstances, and even in healthy persons, if carefully looked for. Those which follow taps upon the surface of the muscle are particularly well marked in patients who are emaciated by pulmonary phthisis or any similar disease. But in progressive muscular atrophy, the spontaneous fibrillary contractions sometimes far exceed those which occur under other conditions, so as to produce perceptible movements of the limbs and to attract the notice of the patient, although they are

attended with no pain. It is said, too, that they sometimes afford the earliest indication of the extension of the disease to a fresh group of muscles. Duchenne found fibrillary contractions absent in about a fifth of the cases that came under his observation.

The cutaneous sensibility is altogether intact; there is usually no pain in the affected parts, nor are any sensations of numbness experienced. This, at least, is what Charcot says of that form of the disease which he terms "protopathic," and which alone is now under consideration. Leyden and other writers, while admitting that impairment is slight in comparison with the muscular wasting, have yet described partial anæsthesia as of rather frequent occurrence. But they doubtless included certain other affections which Charcot has since shown to be both clinically and pathologically distinct, and which will be mentioned further on in this chapter. Occasionally severe shooting pains are felt, especially in the early stages of the disease.

Course and event.—It is not in every case that progressive muscular atrophy manifests a tendency to spread from one group of muscles to another. On the contrary, it may remain for many years limited to those muscles which are first attacked by it. But, as we have seen, it often extends from the limbs to the trunk; and then it is very apt to destroy life by interfering with the respiratory movements. One should always carefully observe the condition of the intercostals, and also that of the diaphragm. If the former muscles fail to contract, the upper two thirds of the chest cease to expand, and the act of expiration is proportionately shortened. If the latter is atrophied the epigastric and hypochondriac regions are drawn in during inspiration. In either case the patient has frequently to stop between his words to take breath. He has difficulty in crying out and in singing, and the attempt to cough or to sneeze causes him great distress. An attack of bronchitis is almost inevitably fatal, so that the greatest care must be taken to protect him from exposure to cold. Not infrequently the intercostal muscles and the diaphragm fail simultaneously; and then, as the disease advances, suffocation becomes inevitable. Sometimes, on the other hand, the disease proves fatal by spreading to the muscles of the throat and of the pharynx; the symptoms which are then observed will be presently described under the head of "Progressive Bulbar Paralysis." In not a few instances death occurs from phthisis or pneumonia, or from an accidental attack of enteric fever. The duration of progressive muscular atrophy varies from two or three to as many as twenty years.

Ætiology.—The causes of progressive muscular atrophy are quite uncertain. It occurs chiefly in young adults, seldom or never under its typical form in children (cf. *infra*, p. 514); according to Leyden, the mean age is about thirty-two years. It is far more common in men than in women. Friedreich found only thirty-three women among 209 patients, and Sir Wm. Roberts only fifteen among ninety-nine.

Charcot and many other writers think that the disease is frequently transmitted by inheritance. That such transmission sometimes occurs is indisputable; but it would seem that the majority of the supposed examples have really a different pathology (*infra*, p. 515).

Over-use of the muscles, leading to exhaustion (of them or of the corresponding spinal centres), is often regarded as the cause of the disease, as when it occurs in a bank clerk, in a cobbler, or in a saddler. This would naturally be more common in men, although washerwomen are mentioned

as furnishing examples of the same ætiology. But here, again, confusion may have arisen from the admixture of cases of "writers' cramp" with those of the affection now under consideration, since until recently the two diseases were often included under the vague designation of "scriveners' palsy." Progressive muscular atrophy often occurs in persons who have in no way over-fatigued their muscles, and the relation between the supposed cause and its effect is probably that of accidental coincidence. A certain number of cases follow closely upon measles and other acute diseases, and others again after a longer interval upon syphilis.

Pathology and histology.—The true nature of this affection has only gradually been cleared up. Both Aran and Cruveilhier originally supposed that it was a primary atrophy of the muscles, but the latter altered his opinion when in 1853 he found, in the celebrated case of the rope-dancer Lecomte, that the anterior roots of the spinal nerves were grey and atrophied. The central origin of the disease in the destruction of the large motor ganglion-cells of the anterior cornu of the cord was demonstrated by Lockhart Clarke. In 1873, Friedreich, of Heidelberg, devoted a quarto volume to the support of the myopathic view, and proposed to term the affection "Polymyositis chronica progressiva." His chief arguments were that the spinal lesions which had in certain cases been found were very variable in their nature and in their exact position, and that they might fairly be attributed to the extension of an inflammatory process upwards along the nerves. But about the same time Charcot devoted five of his lectures to this subject, and he has shown beyond dispute not only that the most typical form of progressive muscular atrophy in one or both upper limbs is due to a particular change in the corresponding anterior grey horn or horns, but also that other clinical varieties of the complaint can be assigned to separate and distinct affections of the spinal cord.

Duchenne asserted that in the disease under discussion there is *atrophy without paralysis*. Although he abandoned the myopathic theory of its origin, he continued to maintain that there was no failure of "nervous *motor* action." He thought that the multipolar cells of the anterior cornua did not lose their motor, but only their *trophic* function, and asserted that the loss of power was always proportionate to the degree of wasting, and that any muscular fibres remaining at a particular time would always obey the will, even though they might not succeed in overcoming resistance so as to effect a visible movement. In proof of this doctrine he appealed to the fact that faradisation of the affected muscles would cause contractions after they had undergone extreme wasting, so long as any fibres remained. But Erb subsequently investigated with great care the action of galvanic as well as of faradic currents, and he was always able to detect, in at least some of the affected muscles, a modified (or "middle") form of the reaction of degeneration: the fibres contracted slowly under a powerful galvanic current when they were no longer sensitive to the faradic current, and A.C.C. was greater than C.C.C. The facts that this change cannot be detected in other muscles, and that it is easily overlooked where present, he attributes to the partial and gradual character of the atrophy. The fibres which have undergone degeneration are concealed by the still healthy fibres which lie beside them. So far as a theory can go this seems satisfactory; and it allows us to admit nervous motor paralysis. We may therefore conclude that the most common and typical form of Cruveilhier's palsy is truly an atrophic spinal paralysis,

like infantile palsy, but exceedingly chronic instead of exceedingly acute; and it picks out, not certain muscles of a limb, but certain fasciculi, or perhaps certain fibres of a muscle.

Histologically the morbid process in the paralysed muscles is far from being as simple as it was at one time supposed to be. By the earlier writers on the subject they were described as being in a state of "fatty degeneration," having lost their transverse striation, and being full of drops and granules of oil. Robin, however, found that the granules were often dissolved by acetic acid, and therefore could not be all of a fatty nature; and it is now known that while many of the fibres undergo fatty degeneration like that to which the heart is subject, others become merely reduced in size and thickness while retaining their striation, that others pass into the condition known as "cloudy swelling" and that others again undergo "waxy" degeneration, such as may occur in enteric fever. Another part of the process consists in an increase in the number of the sarcolemma-nuclei; and at the same time the perimysium becomes the seat of a cell-growth, from which are developed the adipose and connective tissue that sometimes gives to the muscles a deceptive appearance of being still well nourished. The several fibres undergo destruction not simultaneously, but in succession. Thus there are all possible degrees of wasting, down to a point at which the microscope reveals not a vestige of the normal striation, nor even an empty sarcolemma-sheath. Muscles so affected, instead of red, are pale grey or yellowish, and at length become converted into mere bands of white fibrous tissue, or shapeless masses of fat lying between their tendons of attachment.

The histological condition of the cord in progressive muscular atrophy is essentially the same as we have seen to exist in the acute atrophic paralysis of children and in the acute or chronic atrophic paralysis of adults. The large motor ganglion-cells of the anterior cornua are found shrunken and often much pigmented; or they have disappeared, and Deiters' cells or corpora amylacea take their place, along with fibrous tissue made up of neuroglia, atrophied nerve-fibres, and obsolete blood-vessels. The blood-vessels become enlarged and thickened, and in extreme cases the whole anterior cornu is obviously shrunken to the naked eye. The pathological process differs from that of other forms of cornual myelitis chiefly by its exceeding chronicity.

It is right to add that the anterior horns are said to have been found unaffected in certain cases which during life answered to the type of progressive muscular atrophy, so that many good authorities still admit a primary atrophy of muscular fasciculi, which leads to loss of power without precedent lesion of the anterior cornua or motor roots. This primary or myopathic atrophy will be described separately (*infra*, pp. 514, 519).

In a large number of cases, sclerosis of the crossed pyramidal tracts is also found.

According to Charcot, distinct lesions of the cord have been included under the designation of progressive muscular atrophy.

1. *Primary progressive muscular atrophy — Primary poliomyelitis anterior longissima.*—In the typical affection of Aran and Cruveilhier, as just described, which Charcot proposes to call *amyotrophie progressive protopathique*, the primary morbid change is atrophy of the multipolar cells of one or both of the anterior grey cornua. The resulting impairment of muscular power

presents those characters of sharp and apparently capricious limitation which have already been detailed, and is accompanied neither by disturbances of sensation nor by spasmodic contractions. Charcot's account of this form of the disease was based upon six or seven autopsies, and it has been since fully confirmed.

2. *Progressive muscular atrophy combined with spastic paraplegia.*—Contrasting with primary muscular atrophy there are other cases which Charcot terms *amyotrophies deutéropathiques*, because in them the change in the anterior cornua is secondary to a lesion of some other part of the cord. Of these he describes the commonest as *sclérose latérale amyotrophique*, consisting in a chronic inflammatory process which begins in the lateral white columns (occupying them symmetrically on each side), and then spreads into the grey matter, principally in the cervical region. Türck, in 1856, appears to have been the first to notice this morbid change occurring independently of any disease in the brain. Charcot says that it is characterised clinically by the presence of paralysis with rigidity of the lower limbs, in addition to the atrophy of muscles in the upper limbs. The spasm in the legs is at first transitory, but afterwards permanent; they generally assume a *position of flexion*, and remain but little wasted. In the arms and hands the muscles undergo excessive atrophy, not one by one, as in primary progressive muscular atrophy, but several at once; the elbow assumes a position of semiflexion and pronation; the wrist is bent; the fingers are closed into the palm of the hand. Charcot further says that the morbid change in the muscles presents inflammatory or irritative characters in a more marked degree than in the "protopathic" form; the connective tissue between the fibres undergoes a still more decided overgrowth, and the nuclei with which it is infiltrated are more numerous. Another peculiarity is that the course of the disease is more rapid; all four limbs become quickly involved; the patient is confined to his bed in a few months and does not live more than from one to three years, the usual cause of death being an extension of the morbid process to the medulla oblongata, with the symptoms of *bulbar paralysis*. This form of progressive muscular atrophy is said by Charcot to be always incurable; those affected by it have been from twenty-six to fifty years old; exposure to cold and damp has sometimes been assigned as its cause. At the Salpêtrière five cases had occurred in which autopsies were made when Charcot wrote.

There is no doubt of the accuracy of these clinical observations of Charcot, and we have already stated that lateral sclerosis affecting the crossed pyramidal tracts in the dorso-lumbar region may often be discovered after death. But it may be that this is a descending secondary sclerosis, and that the primary lesion is the poliomyelitis of the cervical enlargement which produces the atrophic palsy of the arms. This view was taken by Leyden. Dr Gowers believes that lateral sclerosis is frequently, perhaps usually, present when there are spastic symptoms during life, and that it is not secondary to the cornual lesion nor (as Charcot supposed) its cause, but part of the same process. He therefore would draw no sharp line between this second and the first variety, either clinically or pathologically.* Erb and Eichhorst recognise the validity of Charcot's striking account of the affection, and place it provisionally with other forms of spastic paralysis.

* See also cases by Dr Ferrier in the 'Lancet' for 1881, p. 822, and a paper by Friedenreich, of Copenhagen, and Roth, of Moscow, in the 'Transactions of the International Medical Congress' for 1884, vol. iv, pp. 100, 105.

It has been mentioned above as one of the forms of secondary spasmodic paraplegia (483).

3. *Hypertrophic cervical pachymeningitis*, with secondary spastic paralysis and atrophy.—Charcot regards this remarkable affection as another form of *amyotrophie deutéropathique*. As its name implies, it is a chronic thickening of the dura mater, which presents a number of concentric layers, and may fill up the whole vertebral canal. It and the arachnoid adhere firmly to the cord, so as to compress and flatten it, and necessarily surround and press upon those nerve-roots which come off at the level of the lesion, generally belonging to the brachial plexus on each side. This affection, like that last mentioned, is attended with progressive wasting of the muscles of the upper limbs, and with rigidity of the lower limbs. It is distinguished by the circumstance that the ulnar and median nerves are especially involved (the musculo-spiral nerve escaping), so that the wrist assumes a position of extension instead of being flexed. Charcot has observed that the skin often becomes anæsthetic, not only in the arms, but in the upper part of the trunk; and that when the lower limbs become rigid they do not seem to waste. But what is especially characteristic of cervical pachymeningitis is the occurrence of an early stage, lasting two or three months, and accompanied by severe pains in the neck and back of the head, by a sort of rigidity of the cervical muscles, by sensations of numbness and tingling in the upper limbs, and sometimes by bullous eruptions. All of these symptoms are of course due to irritation of the nerve-roots at the seat of the disease. The affection seems to stand to the other atrophic spinal paralyses in the same relation as "compression-paraplegia" to the common forms of myelitis. Perhaps the most important point of all is that it is not always incurable. A woman under Charcot's care, after an illness of five or six years, during which she was for a long time perfectly helpless and confined to her bed, ultimately became able to walk, and could also to some extent make use of her hands. The lesion does not tend to spread to the medulla oblongata, so that the symptoms are seldom complicated by the supervention of bulbar paralysis; but there may at last be loss of power over the bladder and the rectum, and bedsores may form, symptoms which are not observed in other forms of wasting palsy.*

We have had what is probably a case of this form of paralysis in a young man who has lain for nearly three years in Philip Ward. His symptoms were at first those of acute cervical meningitis, perhaps of traumatic origin: these were followed by a period of paralysis with wasting of the arms, and

* In 1868 I recorded in the 'Practitioner' a series of cases of progressive muscular atrophy, one of which would seem to have been an example of hypertrophic cervical pachymeningitis, if reliance can be placed on the facts that rigidity of the lower limbs constituted a prominent symptom, and that the patient, a woman aged thirty-two, recovered. At that time I spoke of the affection as altogether exceptional. It is interesting now to refer to the clinical report, and to find that at an early stage she had complained of severe pains about the elbows, which were doubtless due to irritation of nerve-roots. Moreover, three years previously she had for a time lost power in the second finger of the right hand. I have also reason to believe that a second example of the same affection is afforded by another of my cases, that of G. P. H—, aged twenty-seven, who is recorded as having recovered from a well-marked form of progressive muscular atrophy under treatment by the continuous current. This man was readmitted into the hospital in 1869 with severe cramps and numbness in the arms, tonic extension of the legs, rigidity of the recti muscles of the abdomen, and a sensation of tightness round the waist, as if constricted by a cord. On referring to the notes taken in 1867, I find that at that time he had tingling sensations in the arms and weakness of the lower limbs, so that even then the case deviated to some extent from the typical "Cruveilhier's paralysis."—C. H. F.

then of spastic paraplegia with pelvic symptoms. At the present time (March, 1890) the disease has been long in a chronic state.

4. *Hydromyelia.*—In some instances of what may be called a secondary form of atrophic paralysis, a very obvious lesion has been found, a hollow space occupying the centre of the cord. The first case in which atrophic paralysis of the arms was observed in connection with this curious lesion was recorded by Gull in the 'Guy's Hospital Reports' for 1862. The patient, a journeyman tailor, aged forty-four, had suffered for thirteen months from loss of power in the little and ring fingers of the right hand, and for a shorter time from a similar affection of the corresponding fingers of the left hand. The muscles of the hands had undergone extreme wasting. He caught typhus in the hospital, and died of that disease. In the cervical and upper dorsal regions of the cord the place of the grey matter was taken by a large quadrilateral cavity which was supposed to be a dilated part of the central canal. Since *hydromyelus* has been met with without any spinal symptoms during life, we might doubt whether its presence was not a mere accident. In children suffering from spina bifida, hydromyelus is not uncommon. Since the publication of Gull's case, however, similar ones, attended with like symptoms, have been met with by Schüppel and Hallopeau and Westphal; and it now seems impossible to explain them away. Th. Simon has shown that in reality the cavity is often unconnected with the central canal, which lies in front of it and is no larger than natural. He proposes to apply to such cases, instead of hydromyelus, the term *syringomyelus*, originally invented by Ollivier, though not with the object of conveying this distinction. We have already seen that the development of a solid new growth in the cord may be attended with the formation of a cyst in its interior (p. 451); but the cases now under discussion have not that origin. Clinically, the progressive muscular atrophy due to syringomyelus does not appear to differ from the ordinary protopathic form of the disease.*

5. Secondary (or deuteropathic) progressive muscular atrophy may occur as a complication in cases of *Tabes dorsalis* or *locomotor ataxy;* and also in those of *multiple insular sclerosis*, if the morbid change should happen to involve the anterior cornua and destroy the multipolar cells. It may be associated with bulbar paralysis, but the latter affection is usually secondary.

A *tumour* growing in the substance of the cord, or in the membranes, may sometimes be attended with symptoms of a similar kind. As Erb remarks, it is sometimes impossible to distinguish between a meningeal new growth and cervical pachymeningitis. A woman was admitted to Guy's Hospital in 1878 with atrophic paralysis of certain muscles in the left hand; and she also was liable to exceedingly violent paroxysms of pain in the right shoulder and arm, attended with sudden redness and swelling of the tender parts, evidently due to disturbance of the vaso-motor nerves. She had traces of iritis, and the author inclined to the diagnosis of a syphilitic gumma, growing from the membranes and pressing on the cord in the cervical region. She improved under iodide of potassium.

Diagnosis.—Progressive muscular atrophy must be carefully distinguished from other diseases attended with wasting of muscles.

First among them should perhaps be mentioned various forms of *peripheral paralysis* due to neuritis of the several branches of the brachial plexus, such

* Dr Frederick Taylor collected ten cases, and in his papers in the 'Pathological Transactions' (vols. xxix, p. 21, and xxxv, p. 36) gave a complete account of this singular lesion.

as have been described at p. 396. There is no doubt that cases of this kind have often been mistaken for those of Cruveilhier's palsy; but a careful observer will generally recognise the limitation of the affection to the area of distribution of particular nerves; and all doubt will be removed by the application of galvanic and faradic currents, which will reveal a far more marked alteration of the normal electrical reactions than is ever seen in progressive muscular atrophy. Moreover, in peripheral neuritis there is almost always anæsthesia, and usually local tenderness.

The same tests will distinguish ordinary cases of *lead paralysis*, even when the history of the patient, the line on the gums, and the other poisonous effects of the metal are not of themselves sufficient to prevent hesitation as to the diagnosis. For the peculiarities of the reaction of degeneration are well marked; whereas in progressive muscular atrophy they are either imperfect (p. 500) or the muscles continue to respond to faradism as well as to galvanism so long as there is any muscle left to contract; the reason probably being that the motor nerves, trophic cells, and muscle-fibres with their end plates all undergo very slow and equal atrophy, and healthy muscle is not left with damaged anterior cornua and degenerated nerves, as is the case in lead palsy.*

The diagnosis from *chronic atrophic spinal paralysis* apart from lead (p. 495) rests on the facts that, in the latter, paralysis precedes atrophy, instead of going with it, and that instead of the reaction of degeneration occurring only in the "middle" or qualitative form, or only towards the close of the disease, or not at all, it is present in its typical form just as in infantile palsy.

Another possible error is to mistake for progressive muscular atrophy the diffused wasting of the muscles, attended with great helplessness and loss of power, which is a result of protracted *osteo-arthritis*. The hollowing of the interosseous spaces and of the ball of the thumb may be very striking in cases of this kind.

French writers describe under the name of *marasme essentiel*, an atrophic affection which they say occurs in hypochondriacal patients, who gradually assume the appearance of living skeletons. It corresponds with the remarkable case of emaciation in a man who was cured by Weir Mitchell's massage treatment, published by Drs Playfair and Brunton. There is, however, no true paralysis in these cases.

Prognosis.—The natural course of progressive muscular atrophy is gradual but fatal. Its tendency is slowly but surely to spread from one muscle to another until those of respiration are involved. Occasionally its course is arrested without apparent cause, and the atrophied muscles remain like those of a withered limb after infantile palsy.

Treatment.—Experience has shown that drugs are of little value in this disease, though Dr Gowers thinks that arsenic and strychnia are sometimes of service. Neither nitrate of silver, phosphorus, nor iodide of potassium can restore the wasted muscles, or prevent further extension of the disease.

* I have seen a few instances in which, although it was known that the poison had been absorbed to some extent, opinions yet differed as to whether this fact would satisfactorily account for the symptoms, which seemed to be too severe, or too little amenable to treatment, to be caused by lead. Two such cases I published in the 'Practitioner' (1868), under the name of progressive muscular atrophy. I am bound to say that now, on reading them over, I think that the view then taken was wrong: they were cases of plumbism.—C. H. F.

There is considerable evidence of the value of electricity. Faradisation of the affected parts was strongly recommended by Duchenne. He advised the application of currents of moderate intensity, with not too frequent intermissions, and for a few minutes only at a time, so as not to fatigue the fibres remaining undestroyed; he particularly insisted on the importance of including in the treatment any important muscles, such as the diaphragm, the intercostals, and the deltoids when they are first threatened by the disease, and before they are wasted. In the case of a man named Bonnard, who had lost many muscles of his trunk, and who was beginning to suffer from dyspnœa, so that he could scarcely walk a few steps without stopping to take breath, faradisation of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances and to go upstairs without fatigue. A similar treatment, applied to certain muscles of the arms which were wasted, restored their functions, so that at the end of six months he was again able to support his family by his exertions. And he went on for some years without the disease advancing further.

Remak advocated the use of the galvanic current. His method was to place the positive pole in front of one mastoid process, and the negative pole on the opposite side of the neck, near the spinous processes of the vertebræ, not higher than the fifth cervical. This, he found, would often produce contractions in the fingers or other paralysed parts.

All observers admit that in not a few cases both these plans of treatment utterly fail. They may, however, be tried in succession; and it is worth while to persevere with them, even when they seem at first to be doing no good, because sometimes good results have been attained after several months of electrical treatment.

Dr Ross believes that all drugs are useless, and that galvanism is the most efficient remedy for progressive muscular atrophy. But Dr Gowers finds that "the most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce any effect on the course of the disease." He is equally incredulous of the asserted benefits of *massage*, which are "usually inappreciable," and of *balnéothérapie*. Even when the disease follows syphilis he finds mercury useless. "The disease is one in which it is not easy to do good and not difficult to do harm."

Early in the disease the affected parts should be rested as much as possible. In one of the author's cases the forearms were wrapped in cotton-wool and placed in splints, and after a week the patient was found to have much more power in his hands. But in the more advanced stages methodical exercise of the wasted muscles appears to be sometimes useful.

PROGRESSIVE BULBAR PARALYSIS.*—In 1860, Duchenne gave a clinical description of a form of progressive paralysis of the tongue, palate, and lips, which had previously received no systematic recognition from writers in medicine, although Trousseau, as far back as 1841, had noted the peculiar symptoms presented by a well-marked case occurring in the Prince de la Moskowa. The disease has now become well known in England, and is often termed labio-glosso-laryngeal paralysis, after Trousseau and Duchenne;

* *Synonyms.*—Labio-glosso-laryngeal paralysis — Glosso-labio-pharyngeal paralysis—Duchenne's paralysis—Progressive muscular atrophy of the tongue, palate and lips—Atrophic bulbar paralysis (Leyden).

but a shorter and better name is that of "progressive bulbar paralysis," which was originally suggested by Wachsmuth in 1864. As Kussmaul has remarked, it might be called "progressive paralysis of the bulbar nuclei;" for the morbid change does not involve that part of the bulb (*i. e.* medulla oblongata) which transmits the motor and sensory strands for the limbs and the body generally, but is confined to the grey centres for certain of the cranial nerves on the floor of the fourth ventricle.

Course and symptoms.—As a rule, bulbar paralysis begins insidiously. The earliest symptoms are commonly subjective,—a feeling of pressure at the back of the neck and head, a little giddiness, a sense of constriction round the throat or chest, slight discomfort in talking, as if the tongue were heavy and its movements laboured, or a tired feeling after speaking. Krishaber has stated that in two cases he discovered a loss of reflex irritability in the pharynx and larynx some months before any signs of paralysis made their appearance. Sometimes the palate is affected before the tongue, or the disease begins in the lips: in the former case the speech acquires a "nasal" quality, and swallowing seems to require an unusual effort; in the latter the expression of the face about the mouth becomes altered, or there may be difficulty in the utterance of certain letters, as though (to use a phrase of Duchenne's) the lips were half paralysed by cold.

Sometimes, however, the commencement of the disease appears to be sudden. Kussmaul, in one of the clinical lectures published by Volkmann, relates that a patient of his, a Catholic priest, found one day, while preaching, that his mouth was distorted and that he had a difficulty in speaking. He was able to finish his sermon, but from that time he felt "a heaviness of the tongue." For a week previously he had suffered from pains in the back, but he had had no giddiness. Dysphagia soon set in, and within six months the case became one of confirmed bulbar paralysis, with loss of power in the arms and wasting of the small muscles of the hands. It was ascertained at the autopsy that there was no hæmorrhage into the pons or the medulla oblongata; and this, as well as the progressive character of the complaint, distinguishes it from certain cases of Wilks, of hæmorrhage in the bulb producing symptoms of paralysis to which we shall presently refer (p. 513). In a woman whose case is recorded by Leyden the first symptom was a sudden attack of dyspnœa, lasting five minutes; a few days afterwards she noticed a difficulty in moving the tongue when she spoke or ate.

In fully developed cases the tongue is generally the part in which the loss of power is most obvious. It lies flaccid in the floor of the mouth; the patient can neither bend it laterally, nor raise it against the palate, nor hollow its centre; and he may be unable to protrude it beyond the teeth. Scarcely less marked is the paralysis of the lips. The mouth remains open, and with its angles drawn wide apart; the naso-labial furrows are deepened, the lower lip hangs away from the gum. The patient cannot whistle or blow out a candle, or kiss. What is most remarkable is that although facial muscles supplied by the lower branches of the *portio dura* are thus affected on both sides in every case, those to which the upper branches of the same nerve are distributed as constantly escape. The orbicularis palpebrarum and the occipito-frontalis act as well as ever. The countenance thus acquires a curiously contrasted expression: the eyes are full of life, while the mouth is fixed, sad, and gloomy.

On the other hand, the paralysis of the palate is not indicated by any obvious change in its form. The uvula hangs in its ordinary place; and

Duchenne says that he always found that touching the fauces caused the usual reflex movements. With the laryngoscope the vocal cords may in advanced cases be seen to be more or less completely paralysed.

Several important functions are impaired in ways which are somewhat complicated, and demand careful study. Thus the defect of speech appears to vary with the part which becomes earliest paralysed. According to Kussmaul, if the lips suffer first, O and U are the vowels which the patient experiences most difficulty in uttering; if the tongue, I is sooner lost. A is always retained longer than any of the others.* Among consonants, loss of power in the tongue renders the patient first unable to utter TH, R and SH; next S, L, K, G, T; afterwards D and N. Paralysis of the lips prevents the formation of P and F, then of B and M, ultimately of V. Paralysis of the palate gives to the speech a nasal twang, and it specially prevents the formation of the lip-sounds B and P, because it allows so much of the air to escape through the nose; the proof of this being, as Duchenne pointed out, that closing the nostrils may enable these letters to be sounded. So far the affection of speech is one which merely concerns articulation, and may be called "anarthria," in distinction from the "aphasia" that depends upon lesions in the left side of the brain, and the "aphonia" that is caused by loss of power in the larynx. But in bulbar paralysis, after a time, the vocal cords also lose their functions; and the voice becomes extinguished.

Another set of movements which are interfered with in progressive bulbar paralysis are those which are concerned in the reception of food into the mouth, and its transmission backwards into the œsophagus. During mastication it collects inside the cheek, not only when the buccinator is paralysed, but (even apart from this) because the tongue cannot properly dislodge it. Very often the patient helps himself with the fingers of both hands, supporting the floor of the mouth or the cheeks, or pushing the half-chewed food into the proper position. Then, again, the tongue cannot roll up the softened pulpy material into morsels for swallowing, nor carry them into the pharynx. Loose fragments are constantly dropping out of the open mouth into the plate or upon the patient's clothes. Other pieces collect about the root of the tongue, or in the grooves by the side of the epiglottis. The attempts to swallow them succeed very imperfectly—some pass up into the pharynx or through the nose, others enter the larynx and set up a choking cough; they may even accumulate in the fauces to such an extent as to interfere with the passage of air and to produce sudden death. It depends upon circumstances whether the patient finds most difficulty in dealing with solids or with liquids. If the principal defect is a weakness in the tongue and in the muscles of mastication, he requires to have all his food reduced to a semi-fluid state; but when there is paralysis of the parts concerned in closing the larynx during deglutition, he can often dispose of solid masses better than of liquids, being unable to prevent the latter from trickling down into the air-passages. The inability to swallow gives to the disease another peculiarity in the circumstance that the saliva keeps running out of the mouth; such patients keep a handkerchief constantly held below

* These vowels must be pronounced in the Italian way: U=OO (in *fool*), I=E (in *feel*), A=Ah (in *father*). The two English sounds represented by the letters Th are among the first to disappear in cases in which the tongue is affected. Kussmaul's statements correspond closely with what might have been anticipated from theoretical considerations, of which an admirable account will be found in a paper by Dr Bristowe in the first volume of the 'St Thomas's Hospital Reports.'

the chin, and, as Wilks remarks, this often at the first glance enables one to guess what is the matter with them. The secretion itself is sometimes viscid, sometimes watery. It seems so abundant as naturally to suggest that it is formed in excess, and in one instance Schultz is said to have estimated that there were six or eight times as much of it as would have been poured out under normal conditions. Kussmaul, however, found no such increase in a case in which he determined its quantity. Sometimes the masticatory muscles become at length involved in the paralysis; the patient is then unable to move the lower jaw from side to side, nor can he close the mouth firmly.

Generally the respiration is not obviously affected at an early stage of the disease. Later on dyspnœa often becomes a marked symptom, and on stripping the patient one may find that the respiratory movements of the chest walls are very shallow. In some cases, however, distress of breathing and a constant craving for air are said to have been present, although the diaphragm and the thoracic muscles were still vigorous. Kussmaul cites the written statement of a woman under his care who was in great dread of gaping, because "she was obliged to groan and strain in order to get rid of the air and then breathe more freely." He follows Duchenne in attributing such symptoms to paralysis of the bronchial muscles. There is generally inability to sneeze, to cough, to hawk up phlegm, and to blow the nose; the patient sometimes complains that he is no longer able to smoke. Towards the last, paroxysms often occur, attended with a rapid pulse; these are supposed to depend upon paralysis of the vagi.

Among the symptoms which have been noticed in some exceptional cases may be mentioned slight deafness, noises in the ears, ptosis, paralytic affections of the ocular muscles, numbness and anæsthesia of the face or tongue. Physiological considerations have led naturally to a search for sugar in the urine; but this appears to be always found normal.

The higher cerebral functions remain undisturbed, the patient sleeps well, his intelligence and memory are perfect. The movements of the body and limbs are generally free and active, except towards the last, when there is emaciation from want of food and extreme weakness.

Pathology.—Although, as just stated, the great motor paths through the bulb and the pons escape, it nevertheless frequently happens that there is associated with bulbar paralysis an affection of the upper (or more rarely of the lower) limbs, which agrees in all respects with what has already been described as Progressive Muscular Atrophy. Sometimes one, sometimes the other of these two diseases is the first to develop itself. Duchenne drew a sharp distinction between the two diseases. In labio-glosso-laryngeal paralysis he said there was paralysis without atrophy; in progressive muscular atrophy there was atrophy without paralysis, and certain cases in which he found the tongue wasted were regarded by him as examples of a separate malady, a true progressive muscular atrophy involving that organ. But we have seen that the tendency of recent investigations into "Cruveilhier's palsy" is to show that it is a true spinal paralysis. Moveover, there seems to be no satisfactory proof that, after death from progressive bulbar paralysis, the muscles have ever been examined microscopically and found to be all in a healthy state. Charcot once discovered degenerative atrophy in a tongue which during life had appeared smooth and of natural size, so that it is clear that nothing but pathological evidence can be conclusive. In many cases the fleshy substance of the tongue, and even that

of the lips and palate, is obviously pale, of a yellow or greyish-red colour; and it is streaked with fat or more or less completely converted into a mass of fatty connective tissue.

Thus at the present time the opinion of all the most competent authorities is that progressive bulbar paralysis is pathologically identical with progressive muscular atrophy, differing merely in the circumstance that it affects a particular set of nerve-nuclei in the bulb instead of those in the cord. It is admitted that in the earlier stages the tongue is often not obviously reduced in size, but in advanced cases it is described as being soft, small, wrinkled on the surface, and incessantly agitated by a fibrillary tremor. The lips also become thin and sharp edged; their muscular substance quivers, and the skin over them is marked with minute furrows. In the palate, as might be anticipated, wasting of the muscles is not discoverable, at least during life. It is allowed that there is no absolute correspondence between the degree of the paralysis and that of the atrophy.

The results which have been attained by testing the affected muscles with electricity appear to accord with these statements. The earlier investigators described the faradic contractility as normal; but several German observers have since found it lowered or even extinguished; and with galvanic currents, Erb (as well as Kussmaul) has recently detected the reaction of degeneration in a perfectly characteristic form. The mistake seems to have arisen from the stimulus having been applied to the motor nerves instead of to the muscles themselves. Lastly, atrophy of the motor cells which form the nuclei of the hypoglossal and other motor nerves in the bulb has now been repeatedly observed after death from bulbar paralysis; and this agrees histologically with the lesions of the anterior cornua seen in Cruveilhier's palsy. The bulb after all is but the *medulla spinalis oblongata*, and the grey matter of the floor of the fourth ventricle is formed by the anterior cornua laid open and separated, so that the seat of each disease is serially homologous.

Histology.—Kussmaul cites eight cases of progressive bulbar paralysis, in each of which a complete microscopical examination was made. In general no marked change is seen by the naked eye in the fresh medulla oblongata; but it is said that sometimes a little want of symmetry in the two halves of the floor of the fourth ventricle has been detected, or a slight shrinking; or that this part has appeared discoloured and reddish grey; or that its texture, when cut into, has looked blurred; or that its consistency has been greater or less than natural. One thing, however, is very obvious, even to the naked eye, namely, an extreme degree of atrophy of the nerve-roots arising from this part of the cerebro-spinal axis. It is especially conspicuous in the hypoglossal and the facial nerves; but it is generally plainly visible also in the three divisions of the eighth nerve on each side, and sometimes in the sixth, and in the motor portion of the fifth. The roots in question are grey and transparent, and exceedingly reduced in size.

In stained sections of the hardened medulla oblongata the multipolar cells in certain nuclei are uniformly found to have undergone degenerative changes. Their colour is darker than natural, being deep yellow or yellowish brown; they are often shrunken; their prolongations may be indistinct or may have quite disappeared. They seem to be reduced in number. In one case Charcot and Joffroy came to the conclusion that the hypoglossal nucleus contained only one tenth or one twelfth part of the cells which would have been present in it in a healthy bulb. French histologists appear

not to have recognised any marked change in the neuroglia, but in Germany Leyden in 1870, and Maier, and other observers since, have found it increased in quantity, filamentous, and containing stellate cells; in other words, presenting appearances like those which characterise myelitis going on to sclerosis. The nerve-fibres undergo atrophy. The parts in which these changes are most constantly seen are the nuclei of the hypoglossal, the vagus, and the facial nerves. As regards the last-mentioned nerve, indeed, the symptoms of bulbar paralysis point to the conclusion that only a part of its nucleus is affected; and this accords well with Lockhart Clarke's statement that its roots arise in two separate masses of grey matter; but it does not appear that the obvious pathological application of his observations has been yet traced out in detail. The nucleus of the glosso-pharyngeal nerve escaped the morbid process in a case recorded by Duchenne and Joffroy. The sensory nucleus of the fifth and that of the auditory nerve have been constantly found intact. The olivary bodies have sometimes presented degenerative changes, but more often they have been in a normal state.

Ætiology.—With regard to the causes of bulbar paralysis nothing is certainly known.* It scarcely ever occurs in persons under thirty, and appears absolutely to increase in frequency as age advances up to the seventieth year. It is more common in men than in women—thirty-four cases in fifty-three collected by Kussmaul.

The few cases that have been recorded in young persons have presented peculiar features, and their pathology is still undetermined.

Diagnosis.—This at the commencement of the disease requires considerable acumen; one might easily make light of the early symptoms, and so justly forfeit the confidence of the patient. Even when they are fully developed, one must avoid a hasty conclusion, for the lips and the tongue may be paralysed by various affections, beside that which constitutes the disease described by Duchenne. What characterises it is the absence of additional symptoms which belong to them.

Wilks has pointed out that a condition precisely like that which belongs to progressive bulbar paralysis may be suddenly developed as the result of a circumscribed effusion of blood into the lower part of the pons. He relates in the 'Guy's Hospital Reports' two cases, in one of which an old brownish cyst was found at that spot after death. The same author has seen the disease simulated by hysteria; other observers have recorded cases due to syphilis which have been cured by iodide of potassium; and tumours growing near the bulb may cause similar symptoms.

Prognosis.—It seems probable that the very few recorded instances in which recovery has taken place from "bulbar paralysis" have been essentially different from those described by Duchenne.

Genuine cases which depend upon a progressive change in the bulbar nuclei appear always to end fatally. This was Trousseau's verdict, and all later experience confirms it. Their duration is generally from one to three years; but it may be as long as five years. Sometimes the patient is choked unexpectedly by a mass of food which cannot be propelled beyond

* Erb speaks confidently of its being sometimes caused by cold, and Kussmaul relates a case in which the patient traced it to a cold caught while he was at work in the fields; he was attacked with headache and pain in the neck, and a week later he was conscious of difficulty in swallowing and in speaking. Among other conditions which have been supposed to give rise to it are excessive smoking, over-exertion in playing wind-instruments, syphilis, and falls producing concussion of the medulla oblongata. In one of Trousseau's cases it began during convalescence from a febrile attack.

the entrance into the larynx; of this an instance occurred at Guy's Hospital in 1865. Sometimes death is brought about by an attack of syncope, sometimes by a paroxysm of dyspnœa. Kussmaul remarks that such seizures are particularly apt to happen after exertion of some kind, but that they not infrequently occur at night when the patient is in bed. In other cases exhaustion and emaciation gradually lead to a fatal termination; or pulmonary phthisis may develop itself; or acute pneumonia, consequent upon the admission of food into the air-passages during the act of deglutition.

Treatment.—It is doubtful whether any benefit results from the therapeutic measures yet tried. Kussmaul recommends dry cupping at the nape of the neck in early cases, and the use of shower baths. Among drugs he thinks the nitrate of silver most likely to be serviceable. He has seen temporary benefit from faradisation of the palate and tongue; and he mentions two cases in which striking results were for a time attained by the use of strong galvanic currents passed through the neck and spine. One of his patients, the priest, mentioned above, who had been unable to get his tongue out between his teeth, could, after being galvanised, protrude it beyond his lips; his speech became more distinct and his deglutition easier. After four or five weeks, however, the improvement ceased. Specially applicable to progressive bulbar paralysis is, perhaps, Schulze's method of inducing the act of deglutition by galvanism. It consists in fixing the positive pole upon the nape of the neck, and then rapidly moving the negative pole downwards over the side of the larynx. This procedure may be repeated at short intervals several times during four or five minutes. In the first instance a current from six to eight cells should be tried; but to produce the desired result a large number of elements are often required.

When the patient cannot swallow food at all, or seems likely to be choked in the attempt, a tube must be passed into the stomach; but this procedure often has to be abandoned on account of the irritation excited by it. Nutrient enemata then afford the only means of sustaining life for a time, unless recourse is had to gastrostomy. In one case at Guy's Hospital the trachea was opened when danger was apprehended from frequent attacks of dyspnœa; and the result was that they ceased, although of course the other symptoms remained.

Acute bulbar paralysis—myelitis bulbi acuta focalis.—The remarkable malady just described is pathologically an example of atrophy of the ganglion-cells of the motor tract; comparable with the similar anatomical condition which precedes progressive muscular atrophy, the latter affects the anterior cornua of the medulla spinalis, the former the corresponding grey nuclei in the medulla oblongata. A few cases have also been recorded of acute paralysis of the muscles, supplied from the bulb which similarly corresponds to acute atrophic spinal paralysis from acute anterior cornual myelitis. Three examples are cited by Erb, all of which were observed by Leyden. In one case a patch of softening with numerous capillary extravasations of blood was plainly visible in the pyramids and in the olivary bodies at the autopsy; in the others no lesion was discovered until the parts had been hardened, when the microscope revealed inflammatory changes of the most marked kind. Each patient had been taken ill rather suddenly, and had died in from four to ten days. The symptoms varied considerably, since they depended upon the exact seat of the morbid process in a part of the

nervous system where so many important centres are gathered into a narrow space. Chief among them were dysphagia, headache, giddiness, vomiting or severe hiccough, more or less impairment of speech, irregular, rapid, or interrupted breathing, a quick, feeble and intermittent pulse, partial or complete paralysis of the tongue, formication and pains in the limbs. There was no failure of consciousness, nothing at all resembling an apoplectic seizure. Depression and collapse quickly set in, and the fatal issue was brought about by paralysis of the respiratory muscles, with distress of breathing and lividity.

The *diagnosis* of the seat of the disease seems not to be difficult; but it is not easy to exclude the possibility of embolism of the basilar artery or of the vertebral arteries or their branches, or of a minute spot of hæmorrhage, or even of a rapidly-developed compression of the medulla oblongata by some disease in its neighbourhood. Possibly such cases need not invariably prove fatal, since recovery from ordinary myelitis is not unknown.

A case which seems to have been of this kind occurred in Guy's Hospital in 1874, in a man aged forty-five. It proved fatal in about ten days. The central part of the pons appeared softened; but after preparation in chromic acid no decided morbid changes were made out.

Hæmorrhage, embolism, tubercular and other tumours of the bulb, have been recorded, with symptoms resembling those of focal myelitis affecting the same region.

PROGRESSIVE MUSCULAR ATROPHY OF YOUTH.—*a. The early form of Duchenne.*—According to Duchenne, progressive muscular atrophy is not so rare as is supposed in children, and when it occurs presents certain peculiarities in its symptoms and course. It begins in the lips, which become thick and cannot be brought together. If the child smiles, the angles of the mouth are drawn far apart, and the cheeks are flattened by the action of the buccinator muscles. The articulation of labial letters and of the vowel *o* is impaired. It is almost always between the fifth and the seventh year that this form of the affection first appears; but at that time it often attracts little notice from the parents, although the reality of the morbid change is at once made apparent if the orbicularis oris and the other muscles are tested by faradic currents, when they are found to have lost their contractility. About the eleventh or twelfth year the muscles of the shoulders and arms begin to waste, and then medical advice is first sought. Later still the muscles of the trunk and those of the lower limbs are attacked in their turn, but the hands escape.

This form of disease is extremely rare; but Duchenne, whose first cases were recorded in 1855, said in 1872 that he had observed no fewer than twenty. One fact which would tend to show that the progressive muscular atrophy of childhood is distinct from the most common forms of wasting paralysis in adults is that it very often occurs in two or more brothers and sisters; but a curious circumstance is that the father of the first two children in whom Duchenne recognised its peculiar characters was afterwards, at the age of forty-eight, attacked with typical "Cruveilhier's paralysis," affecting first the shoulders and arms, but ultimately the lower limbs; he also stated that his father had died of a similar complaint. Whether this was anything more than an accidental coincidence it is difficult to say.

Duchenne's description was written in 1855, but no one appears to have observed similar cases until Landouzy and Déjérine published several in 1874 and one, with an autopsy, in 1885. Barsickow's series of cases of hereditary muscular atrophy, published in 1872, are of a similar clinical type. Westphal, E. Remak, and other writers in France and Germany, have published a few cases since. The muscles of the face are not exempt, and girls as well as boys are affected. The cord and bulb have been found unaffected. If the seat of the disease is in the nerves, it is a form of Peripheral Neuritis affecting the facial and other motor nerves; but in most cases it appears to be of primary muscular origin.

β. *Erb's juvenile hereditary form.**—A clinically distinct "juvenile form" of progressive muscular atrophy has been described by Erb ('Deutsches Archiv f. klin. Med.,' 1884). It usually begins at puberty. The muscles of the shoulder are more affected than those of the hands, and the legs are not infrequently attacked. The face and hands escape. These cases, like Duchenne's above noted, are marked by a *hereditary* character. There are no fibrillary contractions, no reaction of degeneration even of the "middle" or imperfect kind, and *post mortem* the anterior cornua have been found intact in two cases by Friedreich. Occasionally the deltoid or other paralysed muscles hypertrophy—which connects this form with the pseudo-hypertrophic atrophy to be next described.

γ. Dr Tooth has collected the previously published cases of hereditary atrophic paralysis, and added four new ones (Thesis, 1886). Of 30 in all, he finds that 8 occurred between one and five years of age, 10 between five and ten, and 18 between ten and twenty, while only 3 patients were older—twenty-five, thirty-seven,and forty-six; 28 were males and 16 females, so that the disproportion between the sexes was much less than in Cruveilhier's muscular atrophy. Grouped in families, there were 17 instances of more than one case in a family, and only 12 isolated.

In one group the lower extremities were first affected, and particularly the *peroneal* muscles. The extensors and gastrocnemii shared in the paralysis, and the hands and forearms followed. Fibrillary tremors and marked reaction of degeneration were usually present. ('St Barth. Hosp. Rep.,' 1889; see also 'Lond. Med. Rec.' for October of the same year, p. 430.)

It appears from the clinical features, as well as from the anatomical results of Friedreich's two cases, that the lesion is not central, but either a Peripheral Neuritis or primary (myopathic) muscular atrophy. In a case in Virchow's ('Archiv,' 1855) there was sclerosis of the posterior cornua and root-zones.

δ. Meryon's remarkable cases of *hereditary muscular atrophy* described in 1852, may not improbably belong to this same clinical group. They have, however, been generally appropriated as examples of the following disease, and will be again referred to (p. 517).

PSEUDO-HYPERTROPHIC PARALYSIS.†—In 1861, Duchenne recorded in the second edition of his 'Electrisation Localisée' the case of a boy whose legs were so weak that he could scarcely stand, while the muscles of his calves and hips looked like those of an athlete. Seven years later the same fertile author wrote a detailed paper on the disease in question.

* *Syn.*—Dystrophia musculorum progressiva juvenilis (Erb).—Myopathic atrophy of adolescents.

† *Syn.*—Myopathic atrophy with fatty overgrowth—Myosclerosis—Progressive muscular sclerosis—Lipomatosis musculorum luxurians.

In the meantime cases had been noticed by a few German observers. There was one in 1863 in Oppolzer's wards at Vienna; and another, which came under Griesinger's notice in 1864, gave Billroth the opportunity of excising a piece of the deltoid muscle and of proving that there was no real but only an apparent hypertrophy; for it consisted almost entirely of adipose and fibrous tissue, while the muscular fibres themselves were unaltered. Duchenne, in 1865, made similar observations in one of his cases with the aid of his "emporte-pièce." He therefore proposed to term the disease *paralysie myosclérosique*. Dr Gowers' monograph, published in 1879, contained all the information obtained up to that date, and little has been added since.

Histology.—The substance of the enlarged muscles has a whitish-yellow colour, with only a faint reddish tint. There is still a linear arrangement of the fibres, and in extreme cases this alone distinguishes it from the subcutaneous adipose tissue. The fat sometimes extends into the tendons, so that during life they seem to have been encroached upon by the fleshy bellies of the muscles. Occasionally, on the other hand, fibrous tissue only and no fat has been found between the muscular fibres; and in the earlier stages of the disease, this new tissue has been found full of nuclei or spindle-cells. The muscular fibres seem to become greatly reduced in number by a process of simple atrophy, which at last leaves only the collapsed sarcolemma-sheaths. Those fibres which remain are not always completely unaltered, as described by Griesinger. Duchenne described their transverse striation as unusually faint. Other observers have seen some which were striated longitudinally, some which were translucent or "waxy," and some which were in a state of granular or fatty degeneration. Some fibres again are truly hypertrophied, so as to be two or three times as thick as normal; a fact first observed by Cohnheim.

True muscular hypertrophy.—Apart from the physiological hypertrophy of health, a few cases have been described—by Auerbach, Berger, and Friedreich—in which the muscles of the limbs, particularly the arms, have greatly increased in size without increase or with diminution of contractile power. When a minute portion is extracted it is found that the individual fibres are increased in diameter, without interstitial fibrous or fatty overgrowth. Though it is nearly twenty years since the first case was recorded, the pathology of this rare affection is quite unknown.

Course.—The enlargement of the muscles is not present at all stages of pseudo-hypertrophic paralysis. There is an early period during which the only symptom is an impairment of power in the lower limbs. The child—for the affection almost always begins in childhood—is noticed to totter in walking, and to be apt to fall; he has difficulty in getting on to his feet, and is particularly awkward in going upstairs; when he tries to sit down he falls into the chair. In many cases the commencement of the disease occurs before the little patient has learned to walk. At first the parents think that the child is only backward, as so many rachitic children are, but at length they see that there is something more seriously wrong.

When, at the end of a few months or a year from the beginning of the paralysis, the change in the muscles is discoverable, its extent and degree vary widely in different cases. The calves are often affected alone; next in liability to undergo enlargement are the masses of the *erector spinæ* in the loins, the *glutæi*, the *deltoids*, and the *infra spinati*. The *glutæus medius* usually escapes. The muscles of the thigh, the *latissimus dorsi*, the lower

part of the *pectoralis* and *serratus magnus* are more often reduced in size, so as to afford a strange contrast. But sometimes the whole of the trunk and every part of the limbs displays an exaggeration of contour which leaves far behind that of the Farnese Hercules, as Duchenne shows by comparing a sketch of that ancient statue with portraits of a patient of his, a boy ten years old. The enlarged muscles are generally firm and elastic, and when they are brought into action they harden so that it is difficult to believe that they do not consist wholly of contractile elements.

When a child affected with pseudo-hypertrophic paralysis stands up, his attitude is characteristic. The abdomen is pushed forwards, and the hollow of the lumbar vertebræ is greatly exaggerated. The nates project backwards; but the shoulders are thrown further back still, so that a line dropt from the upper dorsal spines falls behind the sacrum. The legs are separated widely from one another. If an attempt is made to straighten the back the child at once falls down. When he walks, he balances the body from side to side at every step, with a "waddling" gait. He cannot rise from the sitting posture without the use of his hands, and the way in which he uses them is very characteristic. He places both hands upon his knees, and gradually pushes up his body into the erect posture by moving his hands from his knees to his groin. This manœuvre, which has been not inaptly called "climbing up the thighs," is well represented by sketches in Dr Gowers' work on 'Diseases of the Cord' (p. 391), and by photographs in Dr Ross's work (vol. i, p. 996).

The susceptibility of the affected muscles to faradic currents is sometimes normal, but in other cases it is considerably lowered. This appears to depend solely on the degree of atrophy of muscular fibres. There is no reaction of degeneration. As with all the atrophic disorders of muscles described in this chapter, pelvic symptoms are absent, and the sphincters act naturally throughout the whole course of the disease. The knee-jerk gradually disappears, as in other atrophic forms of paralysis. Fibrillary tremors, such as are seen in progressive muscular atrophy, are not generally to be noticed; but they have been observed in a few instances. The cutaneous sensibility is unimpaired. The legs and feet are often cold and damp and bluish. The heels are usually drawn up to a greater or less extent, by contraction of the *tendo Achillis;* and there may be a well-marked club-foot. Sometimes the knees are rigidly flexed. The intelligence may be perfect; but it is often defective, and in some of the cases recorded the patients have been idiots.

When the disease has developed itself to a certain point, it is described by Duchenne as remaining stationary for two or three years, or even longer. But at length a further advance takes place. If the legs only were affected, the arms are now involved. The muscles, however, which now lose their functions, do not show even an apparent hypertrophy, but are always reduced in size, although the histological change is exactly the same as in those which were earlier affected. Even those muscles which were at first enlarged ultimately shrink, until they too are obviously atrophied. The patient, who has generally now reached adolescence, becomes altogether unable to stand, or even to sit up; he is a prisoner upon his couch. Ultimately he dies of exhaustion, or is carried off by phthisis, or some other intercurrent complaint. Friedreich, however, remarks that a large majority of the cases hitherto recorded have been lost sight of, so that it is not known how they ended.

Ætiology.—Nothing is known as to the cause of pseudo-hypertrophic paralysis. As occasional exciting causes are mentioned unhealthy conditions of life, chills, over-exertion of the muscles, and the occurrence of convulsions or of an exanthem such as measles.

It often appears in succession in two, or three, or even four children of the same family, especially in the boys, and at the same age in all of them. As might be expected, the parents themselves have always been free from it; but its hereditary origin has often been traceable by its having occurred in brothers or other relations of the father or mother. Transmission is almost always through mothers to sons.*

Boys are far more subject to this malady than girls, the proportion, among seventy-seven cases collected by Friedreich, being sixty-four to thirteen. Of Duchenne's original fifteen cases only two were girls; of forty-one collected by Webber of Boston and quoted by Ross, only five; of thirty-three by Gowers, ten. In a remarkable series of cases recorded by Lutz it appeared in two successive generations in the female line only, affecting five girls. More often it attacks several sons of the same parents, and spares all the daughters.

Out of 75 cases in which the date of its commencement was ascertained, Friedreich found 45 in which it began under five years of age, 17 between six and ten, 8 between eleven and sixteen. In the remaining 5 cases the patients were adults; and 2 of them were aged forty and forty-one respectively when the disease began. There was in 1887 a patient in Guy's Hospital under Dr Frederick Taylor, a youth of eighteen, in whom this disease had developed within only a few months.

Dr Port, of the German Hospital, showed two cases at the Hunterian Society (1887–8), in brothers aged fourteen and nine years. Their four sisters were healthy and one brother; but three other brothers had died after showing symptoms of the same disease.

In eight cases at Guy's Hospital six were in male and two in female patients; two were under ten, and four between ten and twenty.

Diagnosis.—This is very easy in well-marked cases. One morbid state, which must be thought of in connection with it, is that which Brodie and Hilton both believed to depend on the hip-joints being congenitally placed too far backwards; the drawings given in Hilton's work show a compensatory curvature of the spine very like that which is seen in pseudo-hypertrophic paralysis, so that one is inclined to wonder whether his cases could possibly, after all, have been examples of that disease in a slight form. Dr Leech's "harpoon" enables one to examine the muscular tissue during life, and is said to be more useful than Duchenne's *emporte-pièce*. That enlargement of the muscles is not always a conspicuous symptom is certain; unless it should prove that Duchenne and Friedreich were wrong in including under the present head the cases described by Dr Meryon in 1852 under the name of "granular and fatty degeneration of the voluntary muscles," which occurred at about the same age in several boys belonging to two families. Meryon found a different morbid change in the muscles; but it must be remembered that his investigations were made after death had occurred at a very advanced period of the disease, and not upon portions of muscle removed during life. Charcot and Sir William Roberts, however, regard the cases in question as examples of progressive muscular atrophy; and it is to be noted that Friedreich himself relates as instances of the latter disease several cases

* In this character of chiefly affecting males but being chiefly transmitted through females, this curious disease resembles Hæmophilia.

which seem to belong to the same class as Meryon's (cf. p. 514). They occurred in three families residing in or near Heidelberg; and what is very curious is that Hemptenbacher, a pupil of Friedreich's, succeeded in tracing all three families to a single pair of ancestors a century and a half back. A similar malady is said by Eichhorst to have appeared in six successive generations in a family at Königsberg.

Pathology.—Charcot and most other writers at the present time hold that this disease is primarily an affection of the muscles themselves, and thus differs absolutely from progressive muscular atrophy, which has been traced to a lesion in the grey matter of the cord. But we have seen that in progressive muscular atrophy, and even in the atrophic paralysis of children, the wasted muscles often have their bulk made up to the natural standard by an interstitial development of adipose and fibrous tissues, so that if, on the other hand, an apparent overgrowth of the muscles is not an essential feature of pseudo-hypertrophic paralysis, one cannot help suspecting that it must, after all, have close relation to the other atrophic diseases. Indeed, although Pierret and Charcot studied with great care the histology of the cord in a fatal case of M. Bergeron's, and detected no morbid change in it, afterwards Lockhart Clarke and Gowers recorded a case in which they discovered extensive areas of disintegration in various parts, in some of which the nerve-cells were in a state of atrophy. Dr Fagge was strongly inclined to the opinion that pseudo-hypertrophic paralysis will ultimately be found to be a spinal affection, and since the first edition of this work was published Dr Ross and Dr Bramwell have each found changes in the spinal cord. The former observer, however, has in a second case found the cord and nerves of the paralysed muscles unaffected, and the lesions recorded by the latter were either congenital or secondary and recent. Accordingly Dr Ross has, in the second edition of his treatise, abandoned the theory of a spinal origin, and come to the conclusion that pseudo-hypertrophic paralysis is a primary and idiopathic atrophy of the muscles. Dr Gowers also holds that "the essential lesion is the growth of connective tissue by which the muscular fibres are damaged, whether fatty tissue is found or not."

The nearest allies of this curious disease are the hereditary juvenile and probably myopathic forms of progressive muscular atrophy last described.*

Treatment.—There is unfortunately very little to be said on this point. Duchenne believed that he cured two cases (the details of which are given in the 'Archives Générales' for 1868) by faradisation of the affected muscles, with the aid of hydrotherapeutics and massage; they were both in an early stage, with but slight enlargement of the gastrocnemii. At a later period of the disease he found treatment of no use. Benedikt has recorded three cases in which he obtained results by "galvanising the sympathetic," but in the hands of Erb this procedure has since signally failed. Meryon believed that the administration of arsenic retarded the progress of his cases. When contractures have occurred, so as to produce talipes, division of the tendo Achillis has sometimes been useful, by enabling the patient to walk until the muscular atrophy has advanced to a further stage.

* Is it not possible that the apparently inconstant changes in the cord may be the result and not the cause of the muscular atrophy, like the atrophy of motor nerve-cells found by Dr Dickinson and other observers as the result of amputation? ('Journ. of Anat.,' 1869, p. 88; see also Dr Dreschfield's case, *ibid.* 1879, p. 424, and Mr. Bowlby's 'Injuries and Diseases of Nerves,' p. 27, with reference to Vulpian's experimental results.)

Primary multiple muscular atrophy is a condition closely allied to the preceding, but differing in the fact that there is no stage of apparent hypertrophy. Cases will be found mentioned in Dr Poore's edition of Duchenne's great work, and in Dr Gowers' treatise. They occur in families, and sometimes reappear for several generations. Thus Barsickow, in a thesis on the subject, quoted by Gowers, found twenty-four cases in five generations. The affection is not much, if at all, more frequent in men than in women, and usually appears, not in childhood, but in early adult life. It often begins in the muscles of the face, giving what has been called the "myopathic expression," or rather want of expression. When fully developed the condition is much like that of advanced progressive muscular atrophy, and probably some of the cases might take their place as examples of Duchenne's "juvenile" type referred to above (pp. 513, 514).

ATROPHIC PARALYSIS FROM LEAD.*—The most common effect of chronic poisoning by lead upon the nervous system is a peculiar form of paralysis affecting the upper limbs, and known as "the dropped wrist."

This attacks certain groups of muscles much more often than others. Generally the extensors of the hand are the chief ones to suffer; the patient's hand hangs powerless from his wrist, and his fingers are more or less forcibly flexed. When the forearm is laid prone upon a table he is unable to turn it round so as to bring the palm uppermost, but the supinator longus generally escapes. The muscles which form the ball of the thumb are affected very frequently and sometimes before any others; also those of the little finger, the lumbricales and the interossei. In some cases the deltoid muscles are first attacked, and with them the lower part of the trapezii† and the muscles which cover the dorsal surface of the scapulæ. The affected muscles always become atrophied. Thus the ball of the thumb, instead of being rounded, is sunken; the loss of substance in the lumbricales causes the flexor tendons to be visible in the palm of the hand; the neighbourhood of the external condyle of the humerus and the back of the forearm are hollowed out and flaccid. If the shoulder is attacked, it loses its roundness, and the outlines of the bones can be felt much more plainly than is natural. In some cases the whole upper limb may be weak and all its muscles more or less wasted. But the biceps and triceps in the arm and the flexor muscles in the forearm are never so much affected as the extensors.

Most frequently both upper limbs are attacked, but one much more than the other. Sometimes the paralysis is limited to one arm and hand. Perhaps the difference is due to the fact that one arm has been used more than the other in the patient's daily work.

The paralysis which occurs in chronic plumbism is not always confined to the upper limbs. In the legs the peronei are first attacked. In two instances at Guy's Hospital the whole body was affected. The patient could not walk nor raise himself in bed.

* *Syn.* Saturnine palsy.—This term and the corresponding one of Saturnine gout are survivals of the astrological chemistry which connected the seven principal metals with the "seven stars"—gold with the sun, silver with the moon (witness "lunar-caustic"), iron with Mars, quicksilver with "Mercury," tin with Jupiter, copper with Venus (*diva potens Cypri*), and lead with Saturn.

† This curious immunity of the upper part of the trapezius also obtains in progressive muscular atrophy. Compare the muscles most often affected in that disease (p. 497), and in infantile palsy (p. 491).

Pathology.—It was long a question whether lead paralysis is due to an affection of the nervous centres in the cord, or to the local action of lead circulating in the blood upon the muscles themselves. John Hunter long ago took advantage of a fatal accident which had happened to a painter to examine the tissues of his paralysed hand and arm, and he found that the muscles were cream-coloured. Some years ago a man died in Guy's Hospital who had been attacked with lead paralysis seven years before, and had never completely recovered from it. Dr Moxon found that the affected muscles—especially the deep extensors of the forearm—were represented only by loose watery connective tissue.

The tenderness of the muscles, and the marked electrical reaction of degeneration shown by the paralysed muscles, make it probable that lead palsy is due to peripheral neuritis (perhaps affecting the anterior roots of the nerves) rather than to disease of the cord, or to primary atrophy of the muscles.

Diagnosis.—We have already seen the close relation between the symptoms of lead-palsy and those of chronic atrophic spinal paralysis (p. 496). The only other disease for which saturnine paralysis is likely to be mistaken is progressive muscular atrophy. One distinction between them—on which Sir William Gull used to lay stress—is that in chronic plumbism the patient complains of pain when one grasps the affected muscles with one's hand. Moreover, it is not attended with the fibrillary tremors which accompany progressive muscular atrophy. But the most remarkable distinction is afforded by the application of galvanism to the skin over the affected muscles. In lead paralysis faradism causes but slight contraction or none at all, whereas interrupted galvanism gives rise to movements more readily than in health, that is, a smaller number of cells is required to excite the muscles to contract, and the other peculiarities of the reaction of degeneration are well marked. This is absent or ill-developed in progressive muscular atrophy.

The exemption of the supinator longus is so constant as to be often of help in diagnosis. The gums must be examined for the *blue line* which is practically pathognomonic; its peculiar characters will be described in the chapter on Lead-colic. One must ask whether the patient has suffered from pain in the abdomen with constipation; but it is to be borne in mind that when a person has been slowly absorbing the metal in minute quantities for a long time, paralysis often occurs without having been preceded by any pain in the bowels, and these are precisely the cases which are difficult of diagnosis. Sometimes the recognition of chronic plumbism is materially facilitated by the peculiarly sallow and anæmic aspect of the patient.

Another nervous affection which is said to be an occasional result of lead-poisoning is a *tremor*, resembling that caused by mercury. It has hitherto only been observed, by Brockmann, among the miners on the Harts. It is generally limited to the arms and hands, but it very often attacks also the lips and the angles of the mouth. Very rarely it attacks the legs and the muscles of the head and of the trunk.

Treatment.—The medicine most useful in lead palsy is iodide of potassium. Dr Anstie lays stress on the importance of a nutritious diet, and on the value of cod-liver oil in such cases. Sulphur baths have been supposed to be serviceable. They lead to the excretion of some of the lead which has been accumulated in the body; a blackish discoloration of the skin, and still more of the nails, is observed which does not at once rub off, and which evidently is due to the conversion of some of the lead present on the body into a sulphide.

Locally the application of a continuous current, just sufficiently powerful to excite contraction of the affected muscles, is very useful. As soon as they will react to faradism, that form of stimulation may be substituted.

Other effects of plumbic intoxication will be referred to under the headings of Epilepsy and Insanity, Lead-colic, Chronic Interstitial Nephritis, Saturnine Gout, and Anæmia. It is also a cause of abortion and myalgia.

It will be convenient here to refer briefly to the effect of other metallic poisons on the nervous system.

MERCURIAL TREMOR.*—This disorder, once common among looking-glass makers and other workers in quicksilver, has now happily become very rare.

The first thing which a man notices when he is beginning to suffer from mercurial tremor is that he is no longer sure of his arms and hands. So soon as he attempts to take hold of anything they shake. After a time every effort leads to jerking movements. When his arm is bent it is not by a continuous motion, but by fits and starts. His hand cannot be directed with precision to any object, but is thrown to one or the other side of it; when he has even grasped a thing he is often unable to let it go. Presently the lower limbs are affected; he may have to be led, and when he walks his limbs may tremble and dance about, so that (as Sir Thomas Watson says) they look as if hung upon wires. His tongue may become tremulous, his speech may be hurried, jerking (or *staccato*), and at length unintelligible; this last symptom constitutes what has been termed *psellismus mercurialis*. When the tremor has once been excited by exertion or emotion the man himself is unable for a time to stop it, but if he remains quiet for a little while it passes off, and does not return until he has to make some fresh effort. Getting some other person to grasp the affected limbs sometimes controls their movements.

The commencement of mercurial tremor is generally gradual, but sometimes it is sudden, and a fit of passion has seemed to act as an immediate exciting cause of it. It is said to be more apt to occur in the cold and damp weather of winter than at other seasons.

As might be expected, salivation often accompanies the more severe forms of mercurial tremor. The teeth become loose, the gums are ulcerated, the breath is fœtid. The patient grows weak, anæmic, and thin. His teeth are said to become black, and his skin has been described as acquiring a remarkable brown hue. After a time he may begin to suffer from headache and loss of memory, he may become delirious, and at length comatose, and in this way the disease may terminate fatally.

The disease, in fact, tends to advance so long as the patient remains exposed to the influence of the poison, but when he is withdrawn from it the tremor commonly subsides in two or three months. Sometimes, however, the recovery is incomplete, the upper limbs being those which are most apt to remain unsteady. Relapses are frequent, and each successive attack is commonly more severe than the previous ones.

* *Syn.*—Mercurial Shaking Palsy—The "trembles"—*Fr.* Tremblement des Doreurs (Mérat), 1812—*Germ.* Mercurialzittern.

The following is De Haen's account of the disease in one of the miners of Idria more than a century ago, as cited by the late Dr Sanders, of Edinburgh:—"Deaurator, quinque et viginti annorum, horrendo artuum omnium maxime superiorum vexatus, ita ut nec comedere, bibereve solus, neque loquens amplius intelligere potuerit. Nutriendus, vestiendus, et (infantis instar) alvum urinamque positurus, adjuvandus erat: dolorum cæterum immunis."

The workers in quicksilver mines—in which the ore is exposed to heat—are said to suffer severely from mercurial tremor. Formerly it was common enough in this country among "water-gilders," who employ for gilding an amalgam of mercury and gold, from which the volatile metal is expelled by the action of a charcoal fire. But water-gilding is now almost superseded by electro-plating. Another occupation in which mercury is used extensively is that of "silvering" mirrors; this, however, comparatively seldom gives rise to the tremor, probably because the quantity of metal volatilised is but small, since heat is not employed. But mercury does give off vapour to some extent, even at 68° or 70°, so that it is not impossible for the disease to show itself in those who have to deal with the metal at ordinary temperatures. In 1864 a man, suffering from an extreme form of mercurial tremor, was admitted into Guy's Hospital under Dr Rees. He had merely been engaged in packing up skins which had been previously washed with an acid solution of mercury. He said that he had at one time been salivated, but some doubt would perhaps have remained as to the cause of his symptoms, which terminated fatally, had not Dr Swayne Taylor succeeded in obtaining mercury from the kidneys, and (in smaller quantities) from the brain and liver ('Guy's Hospital Reports,' Third Series, vol. x, p. 176). This man had worked at his occupation for three years before he began to suffer to any considerable extent. It is generally said that a time varying from two to twenty-five years is required for the production of mercurial tremor. Sir Thomas Watson relates the case of a man who had been a water-gilder for seventeen years before he was attacked.

There is reason to believe that the disease is sometimes due, not to the inhalation of mercury, but to its absorption by the skin. Dr Mapother states that the anatomy porter of the Irish College of Surgeons, who at one time rubbed enormous quantities of mercury into the bodies of persons affected with syphilis, was for thirty years subject to mercurial stammering. Even the internal administration of this remedy has been known to cause tremor in those who have taken it for a very long time.

The *diagnosis* of mercurial tremor is very easy, except in cases such as that which proved fatal in 1864 in Guy's Hospital, in which the patient might be unaware that he was exposed to the action of the metal. In its earlier stages it is liable to be mistaken for paralysis agitans; in its later periods for chorea. Anstie ('Lancet,' 1872, i, p. 734) mentions the case of a man who was supposed to have died of delirium tremens, until it was afterwards discovered that in his trade he had been inhaling the fumes of the acid nitrate of mercury.

Treatment.—Much has been done to prevent the occurrence of this disease in those whose occupations would otherwise expose them to it. The workshops are freely ventilated and provided with flues or chimneys. In some places it has been found practicable to interpose a glass screen between the upper part of the workman's body and the fire by which the mercurial vapour is set free. Personal cleanliness, frequent ablutions, and avoiding to take food in the room where the work is carried on, are regarded as very important, and it is said that abstemiousness in the use of alcoholic stimulants exerts a beneficial influence, though there may be a doubt whether the effects of intemperance have been isolated from those of other careless habits, which would be apt to prevail in conjunction with it.

Like other forms of tremor, that which is caused by mercury is often diminished for the time by a glass of wine or of some other stimulant. Sir

Thomas Watson's patient said that when the disease was first coming on he found himself unable to get upstairs to his work until he had taken half a quartern of gin.

Among medicines iodide of potassium appears to be the most useful. It was suggested by Melsens on the same theory as for chronic lead-poisoning, and mercury has been said to have been detected in the urine while it was being taken. Sudorific remedies have been recommended, but Sir Thomas Watson says that he found tonics, and particularly iron, more useful than any other drugs in the case which he relates. Anstie found cod-liver oil very useful in several cases. Sir William Gull some years ago recorded an instance in which electricity appeared to be beneficial, sparks being taken from the spine. It was thus also that De Haen's patient (whose case was quoted above in a note) was cured.

BRASS-FOUNDERS' AGUE.—*Tremors from zinc-poisoning.*—There is a curious disorder to which brass-founders are liable; it was studied by Dr Greenhow in Birmingham, in 1858, and named by him "brass-founders' ague," on account of the resemblance between its symptoms and those of a paroxysm of intermittent fever ('Med.-Chir. Trans.,' vol. xlv). It is in no way dependent upon malaria, but is caused by the dense white fumes of oxide of zinc, the result of deflagration of the metal, which, at a certain stage in the formation of brass, fill the casting-shop, unless it is very well ventilated. Exposure to these fumes is followed by malaise and weariness, by a sense of constriction at the chest, and sometimes by nausea. Afterwards shivering comes on, with chattering of the teeth; this is succeeded by a more or less marked hot stage, and the attack ends in very profuse sweating. Next day the man feels out of sorts, but not enough to be unable to work. There is no regularity in the recurrence of the attacks, which come on from time to time under the operation of their exciting cause, but especially when the patient has been off work for a few days. They seem not to impair the health, and it is not known that they shorten the life of the patient. Drinking milk is said to be preventive of the brass-founders' disease.

Dr Greenhow reported that, beside this effect, brass-workers at Birmingham are very liable to shaking palsy. He could only find, however, one case in an old man. Dr Gee has lately published in the 'St Barth. Hosp. Rep.' for 1889, p. 28, a case of a brass-founder, aged thirty-three, who suffered from muscular paresis, trembling tongue, and ankle-clonus, which symptoms were ascribed to zinc-poisoning. See also a good account of the so-called "ague," the bronchitis (coniosis), and the nervous symptoms to which the Birmingham brass-founders are liable, by Dr Robert M. Simon ('Brit. Med. Journ.,' April 28th, 1888). He is inclined, with Dr Hogben, to attribute part of the poisonous effects to the copper, rather than the zinc of the compound metal.

Arsenic is an occasional cause of paralysis, affecting the lower extremities more frequently than the arms. This, like lead-palsy, is believed to depend on peripheral neuritis.

TABES*

'Η νωτιὰς φθίσις ἀπὸ τοῦ μυελοῦ γίνεται
HIPPOCRATES: de morbis, lib. ii, cap. 19.

Histology and nomenclature—Ataxic symptoms—Anæsthesia—Paraplegic symptoms—Lightning-pains—Visceral pains—Loss of knee-jerk—Disorders of the eyes—Atrophy of joints—Perforating ulcer—Other complications—Course and event—Anatomical seat and histology of tabes—Relation between the ataxy, dysæsthesia, and other symptoms—Ætiology—Diagnosis—Prognosis and treatment.

Hereditary tabes: Friedreich's disease, its symptoms and pathology.

History and nomenclature.—Among the chronic affections of the spinal cord there is one which is characterised by an impairment of the power of co-ordinating or combining the actions of muscles in the execution of movements. That it is not an ordinary paralysis is evident from the fact that in some cases the contractile force of the muscles is undiminished. It was carefully described by Duchenne, of Boulogne, in 1858, and he named it *ataxie locomotrice progressive.* But there is no doubt that Romberg had described under the title *tabes dorsalis* most of the principal symptoms of ataxy in 1851, before Duchenne wrote on the subject; and the French observer seems to have somewhat underrated the value of the work of those who had preceded him, although it is incorrect to say that he altogether ignored it.

A series of cases was described by Gull in 1856–7. But earlier still Todd, in 1847 ('Cyclop. of Anat. and Phys.,' iii, p. 721), pointed out the distinction between paralysis and loss of the power of co-ordinating movements, and mentioned the difficulty of walking and the tottering uncertain gait which result from ataxia, while considerable voluntary power may remain. He did not name the latter affection, but his knowledge went beyond that of Romberg and Duchenne in assigning its seat to the posterior columns of the cord.

We now know that though locomotor ataxia is a frequent, it is not the earliest, and not even a constant, symptom of this remarkable disease; so that *tabes* is a far better word. The adjective dorsalis or spinalis is seldom needed, for *tabes mesenterica* is a term now seldom used.†

* *Synonyms.*—Tabes dorsalis *v.* dorsualis—Spinal tabes—Locomotor ataxia—Ataxie locomotrice progressive (Duchenne)—Asynergie locomotrice (Trousseau)—Posterior spinal sclerosis—Chronic posterior leucomyelitis.—*Fr.* Ataxie locomotrice—*Germ.* Tabes dorsalis, Hinterstrangsclerose.

† The original meaning of Tabes dorsalis was purely clinical, and had reference to the weakness and pains in the back, the malnutrition and other disorders which follow excessive sexual indulgence. In fact it nearly answered to what is now called Neurasthenia spinalis. *Tabes dorsalis* from this cause is regarded by Cullen in his nosology as a species of atrophy, distinguished from that of phthisis, of mesenteric tabes, and of chronic suppuration, only by the absence of fever. Celsus has no dorsal variety of Tabes.

Now, however, the word *tabes* is not used, any more than its Greek equivalent *phthisis*, to denote generally wasting, atrophy, or marasmus. "Tabes mesenterica," is tubercular enteritis and peritonitis, with swelling (probably always secondary) of the mesenteric lymph-glands. It is so different from Tabes dorsalis in its clinical features and associations, that confusion can scarcely arise. There is therefore no reason against the term

Clinical characters.—The onset of tabes is always insidious and its progress gradual. Ataxic symptoms may only appear after months or years marked by pains, by loss of knee-jerk, or by contracted pupils. Nevertheless they are the most constant and characteristic, and may properly be first considered.

Motor symptoms.—Ataxia begins almost invariably in the lower limbs. In walking the patient lifts his foot high in the air and throws it outwards, and then he brings the heel to the ground with a stamp. If the case is a slight one, it is at first only in starting that he experiences a difficulty. Wilks relates how a gentleman whom he knew, if he stopped to look in at a shop-window, had to ask someone near to give him a push before he could set off again. Once fairly started, he did pretty well, but found himself unable to turn sharply round without stumbling. Another patient, when he had once walked some distance along a road, wanted to go back, but could only do so by guiding himself up against a bank, when he fell down. A third, an out-patient, one day apologised for being late at the hospital by saying that his friends had sent him off in a wrong direction, and that he went on until he fortunately met an acquaintance who turned him round.

Some writers describe ataxic patients as rolling and staggering like persons who are intoxicated, but Wilks says that the gait rather resembles that of a man walking on a ledge who is anxiously balancing himself. The distinction is a real one, but many persons in the early stage of the disease have been condemned by their neighbours as drunkards. The attempt to run, or to hop, or even to ascend stairs, makes the patient's incapacity still more manifest, he is unable to stand on one foot, or with the two feet close together, without tottering or falling. If told to walk in a straight line—as along one particular board in a floor—his course is sinuous and irregular. The use of a stick, or leaning upon the arm of a friend, has an extraordinary effect in steadying his movements. In more advanced cases, however, as soon as he attempts to stand, his legs are violently jerked in all directions, and this, even though he may be held up on each side by an attendant. When lying on a couch he cannot carry the foot straight towards an object so as to touch it, nor raise the leg up in the air with an even movement. Yet he can exert considerable power in moving his legs against pressure, as in lifting a weight upon his knees.

The disorder may last for years without extending beyond the lower limbs, but in many cases it sooner or later affects the arms. The patient then becomes incapacitated for actions requiring delicate manipulation, such as writing or playing the piano; he cannot fasten his necktie or button his clothes. If he is told to bring the two forefingers into contact from a distance, they miss one another repeatedly. He cannot touch his own nose or rub his own eyes with certainty. He finds a difficulty in taking hold of anything held up before him, and when he attempts to draw or write in the air with his finger, the fingers are moved zigzag in all directions.

Sensory symptoms.—Feeling is more or less impaired in cases of tabes. Subjective feelings of numbness, or of formication, are very often complained of. The patient says that when he stands his feet seem to be covered with thick woollen stockings, or to be treading upon a water-bed, or upon india-

Tabes (or, when needful for distinction from Tabes mesenterica, Tabes dorsalis) being used. It has every character of a good name. It is short, classical, unmeaning, distinctive, and capable of forming an adjective. This adjective is ready made: *tabidus*—not *tabeticus*.

rubber. The cutaneous sensibility may be found diminished in the feet, the legs, or even the thighs, when carefully tested as described at p. 403; but there is rarely any approach to complete anæsthesia. Ability to feel pain is often absent (*analgesia*), while tactile impressions are readily perceived, and patients generally retain the power of appreciating differences of temperature long after they have lost common sensation. That curious symptom, the retarded transmission of impressions, is almost peculiar to locomotor ataxy. It especially concerns the sense of pain, but Hertzberg believed that tactile impressions and those of heat and cold may also be delayed. If there is no anæsthesia, the prick of a needle may first be only felt as a touch, and then, after an interval, a sensation of pain may follow. Erb says that the interval may be one of some minutes; Cruveilhier, who seems to have been the first to draw attention to the symptom in question, noted it at twenty or thirty seconds. Another point is that the pain produced by a slight injury may last longer than under normal circumstances and gradually culminate, and, as a consequence, that the patient becomes unable to count a series of similar impressions if they succeed one another at all quickly.

These paræsthesiæ are by no means limited to the lower limbs. Sensations of numbness are often felt in the parts of the hands supplied by the ulnar nerves, even at the commencement of the disease. Trousseau found in some cases that the mucous membrane of the mouth is anæsthetic, the patient not being able to feel the food between his lips, nor to appreciate its temperature.

A symptom on which French writers lay great stress, as indicative of locomotor ataxy, is the failure to execute movements as well with the eyes shut as when they are open; and there is no doubt that this symptom is often marked. Many a patient who can stand quite steadily so long as he is looking down at his feet totters, and falls when he is made to close his eyes. It may be the first indication that anything is amiss; thus, in a case recorded by the late Dr Bazire, a man believed himself to be quite well until he noticed that he could not wash his face in the morning unless he could lean against a wall; for as soon as he shut his eyes, he lost his balance and staggered.*

Erb has found that this symptom of inability to execute movements accurately with the eyes closed is wanting in certain cases of tabes, those, namely, in which sensation is absolutely unimpaired. On the other hand, it is invariably present when there is much anæsthesia. He admits, indeed, that it may often be noticed in cases in which there is no loss of sensation in the skin; but he supposes that there is failure of the so-called muscular sense. A patient suffering from any spinal disease may be expected to totter in attempting to stand with his eyes shut, if he has imperfect sensation in his legs and feet, although the want of stability is exaggerated and made more manifest by defect of co-ordinating power. Another view first put forth by Leyden is that the want of co-ordination or ataxy is itself the direct result

* According to Jaccoud, the effect of keeping the eyes open is in part due to the influence of light; for he says that a person who has ataxy cannot walk so well when they are closed as when they are open, even when something is held in front of his chest so as to prevent his seeing his feet. But, as Dr Bazire has remarked, this is no doubt due to the feeling of confidence inspired by looking about one, for nervousness has a marked tendency to augment the symptoms of the disease: a patient who is conscious of being observed by others cannot walk nearly so well as when he supposes himself to be unnoticed. A similar explanation may apply to the fact noticed by Benedikt and Friedreich, that some ataxic patients who are blind totter more when they are made to shut their eyes.

of disturbance of tactile and muscular sense. In any case the test of walking with the eyes shut, though practically useful in the diagnosis of tabes from rheumatism, neuralgia, or other peripheral affections of the limbs, does not distinguish one lesion of the spinal cord from the rest.

Paraplegic symptoms.—In some cases of tabes the power of the muscles is undiminished. Duchenne invented the dynamometer for the purpose of demonstrating this fact. In the case of certain muscles, a rough estimate of their strength can easily be made. For example, one can measure that of the quadriceps extensor by getting an assistant to fix the lower part of the thigh, and then telling the patient to keep the knee straight, while one forcibly flexes it. Or one can test the force of the psoas and iliacus muscles by placing him in a chair and making him lift his knee, while at the same time one resists this movement by pressing with both hands upon the knee. Or, following Trousseau's plan, one may determine what weight the patient is able to bear upon his shoulders while standing with the support of a friend's arm or leaning against the wall. A young man under his care could support a hundred and sixty pounds in this way, and another patient carry on his back a doctor who had supposed him to be paraplegic. But, while the theoretical importance of these observations is very great, as showing that locomotor ataxy may be altogether independent of paralysis, it is a great mistake to suppose that all, or nearly all, of the patients who exhibit a want of muscular co-ordination have their muscular power undiminished. On the contrary, one often finds it much enfeebled, and some of the earliest symptoms in these cases are commonly inability to continue standing for long, and fatigue after walking a short distance. It is true that the suggestion has been made that such sensations are really muscular paræsthesia, and do not prove a loss of motor power; and Duchenne records their presence in the patients in whom he demonstrated that the force of the muscles was unimpaired. But our experience is in accordance with that of Erb, who found evident paresis in at least half the cases in which other characteristic symptoms of tabes existed.

Possibly loss of motor power may be due to a complication of posterior sclerosis, the special lesion of tabes, with chronic and more diffuse myelitis. This is confirmed by the not very infrequent existence of the paraplegic symptom described as "girdle-feeling." Trousseau speaks of some of his patients feeling as if the chest or the legs were compressed by an india-rubber cuirass or stocking. Or the sensation may be that of a belt round the waist, of a garter tied below the knee, or of a tight shoe on the foot.

Again, if we inquire how the functions of the *pelvic viscera* are carried on in ataxy, we shall find but little to distinguish tabes from the slighter forms of paraplegia. Thus the bladder is apt to be irritable, and its muscular power may to some extent be diminished, so that the urine no longer flows in a good stream, or is from time to time passed into the bed. Erb notes that the fæces are sometimes discharged without the patient's knowledge, and attributes this to a loss of sensibility in the mucous membrane of the sphincter. The sexual functions are almost always impaired. At an early stage the genital organs are often extremely irritable. It is said that this condition may be accompanied with an increased virile power, but much more frequently there is marked weakness. As the disease advances the patient generally becomes altogether impotent, although there seems no doubt that some persons have been able to beget children after having

symptoms of tabes for years. Bedsores do not appear, at least until the last stage of the disease.

One of the most constant of all symptoms of tabes, first pointed out by Westphal, is *loss of the knee-jerk*. This begins early, and probably continues unchanged throughout the disease. The true superficial reflexes are usually and progressively diminished in proportion to the anæsthesia, but are never absent as in atrophic paraplegia; they are sometimes greatly exaggerated in the early stages, as much so as in many cases of spastic paralysis. Of the visceral reflexes, that of the cremaster is usually impaired, along with loss of sexual power.

The *electrical reaction* of the muscles is often perfect, but sometimes slightly augmented or impaired. They remain well nourished until the disease has reached an advanced stage; but this is likewise the case in paraplegia, unless the anterior cornua of the lumbar cord are affected.

Ultimately many tabid patients pass into a condition of complete paraplegia with atrophy of the muscles, bedsores, paralysis of the bladder, and cystitis.

In exceptional cases of ataxy paraplegia is so marked a symptom that they must be described separately as Ataxic Paraplegia. In most cases this is of the spastic kind, and depends on lateral sclerosis complicating that of the posterior columns. Occasionally also we find atrophy of the muscles set in during cases of tabes, and conclude that the chronic interstitial inflammation has extended to the anterior cornua.*

Pains.—Tabes frequently is attended with certain remarkable early symptoms, warnings, or *prodroma*, which belong to no other spinal affection. Chief among these are sudden pains in the lower limbs or elsewhere. These may either be of a stabbing or boring character, as though a sharp instrument were thrust into the tissues at the spot, which is generally near a joint; or they may be like flashes of lightning or electric shocks shooting down along the course of a nerve. Charcot says that they are worse at night. They last but an instant and return again and again during a period of from four to eight days, after which they may disappear altogether for a fortnight, or even for some months. Their occurrence seems to be favoured by changes of weather. Erb remarks that he has often been struck by the unanimity with which different patients have complained of them on some particular day when there has been much wind, or a fall of snow or of rain. It is also said that they are especially liable to return at spring and autumn, and that they may be brought on by over-exertion of body, mental emotions, or the excitement of coitus. They are sometimes of the most agonising character, sometimes so slight that careful inquiries are needed to elicit the fact of their presence. They are often called "neuralgic" or "rheumatic." They frequently spread from the legs to the trunk, much more rarely to the arms. They may go on for five, ten, or fifteen years, before any other symptoms show themselves, and they often persist throughout the whole course of the disease. As to the frequency of their occurrence, Topinard found that they were wanting in only 22 cases out of 104, Erb in only 8 out of 60, Cyon that among 203 patients there were 138 in whom they were said to have been present, but no more than 8 in whom they were expressly stated to have been absent.

* Dr Gowers has described Westphal's "Ataxic Paraplegia," with lateral and posterior sclerosis, which he recognises in his 'Dis. of Nervous System,' vol. i, p. 341, as a separate affection; see also 'Lancet,' July 3rd, 1886.

Visceral pains.—Other early symptoms mentioned by Charcot are pain in the bladder or in the urethra, with a constant desire to micturate, and a sudden pain in the rectum. Certain strange attacks of pain in the stomach, *crises gastriques*, which were first noticed by Delamare in 1866, consist of lightning-like pains, which start from the groins and pass up both sides of the abdomen towards the shoulders. They are generally accompanied by palpitation of the heart and a quick pulse, by vomiting (of a liquid at first clear but afterwards stained by bile or blood), and by giddiness and malaise. They commonly last for two or three days at a time, and then pass off entirely. They must be carefully distinguished from the gastrodynia of dyspepsia, to which tabid patients are as liable as other people.

Ocular disorders.—Of the symptoms of tabes, which Charcot calls *cephalic*, affections of the ocular muscles, of the iris, and of the optic nerve are the most important.

The functions of one or other of the branches of the third nerve on one side, or of the sixth nerve, may sometimes be annulled, so as to cause diplopia, strabismus, ptosis, and various other disorders, such as have been described in a previous chapter (p. 413). Von Graefe is said to have remarked that in tabid patients, when they have double vision, there is little tendency to fusion of the images, and that this points to a central origin. The ocular paralysis may be transitory, or may last a few days, or may persist for weeks or months; it may return again and again; and may at last become permanent.

Another common symptom is inequality of the pupils, one of them being constantly smaller than the other. Still more frequently both irides are symmetrically contracted to an extreme degree; this *myosis* is often among the earliest warnings. Moreover, the pupils are insensible to the stimulus of light, but contract with accommodation for near objects; the recognition of this important and nearly constant symptom of tabes is due to Dr Argyll Robertson, of Edinburgh.

Impairment of vision is frequent. This is said to develop itself as a progressive narrowing of the visual field, generally from without inwards, until the only part of the retina that remains sensitive to light may be a small patch to the inner side of the blind spots. It is accompanied by achromatopsy; the perception of green being generally the first to disappear, then that of red, and lastly that of yellow and blue. The defects of vision are commonly worse in bright light, so that the patient sees better after sunset. The pupils are often permanently contracted when there is nearly complete blindness. The ophthalmoscope in many cases reveals atrophy of the disc, which in advanced stages is perfectly white, with sharply outlined edge, and very small arteries, whereas at an earlier period it may be greyish in colour. The two retinæ generally suffer together; the affection is a progressive one, and commonly goes on until there is complete amaurosis.

The frequency of these ocular symptoms is very considerable. Erb estimates that, if slight and transitory phenomena are reckoned up with the rest, paresis of the ocular *muscles* is present in more than half of all the cases of ataxy; and that it is persistent in one third or one fifth. How often the optic discs undergo atrophy cannot yet be stated accurately; ophthalmologists are apt to rate it too high, physicians too low; Erb observed it only 8 times in about 70 cases. Topinard found visual disturbances in 49 of 102 cases; and Cyon, amblyopia or amaurosis in 60 of 203

cases. Optic neuritis often occurs at a very early period; according to Charcot, it may precede all other symptoms of ataxy by ten years.

The contracted pupils and the peculiar reflex which is named after Dr A. Robertson are the most frequent of all cephalic symptoms.

Arthropathy.—Another most curious occasional complication of tabes—and often a very early one—is an *affection of the joints*, which has been specially studied by Charcot. Its most common seat is the knee, next in frequency comes the shoulder, and then the elbow, the hip, the wrist. It is not traceable to any blow or other injury. It sets in suddenly with extreme swelling, due not only to effusion into the synovial cavity, but also to infiltration of the parts around. Yet there is generally neither pain, nor heat, nor redness. In the course of a few months the swelling may disappear, but more often the process goes on to destruction of the articular cartilages, with erosion of the ends of the bones and partial dislocation. Charcot says that it never occurs at an advanced period of the disease, except in the upper limbs, when they are beginning to be affected in their turn. This remarkable morbid process will be again considered along with Osteo-arthritis, to which it bears some resemblance.

Other trophic lesions occasionally seen in the course of tabes are: pigmentation or leucodermia in the course of the nerves, like the effects observed in ordinary neuralgia; herpetic eruptions like those of zona; painless ulceration about the fingers and toes which makes the nails fall out; painless loss of teeth, of which we lately had a striking case in Philip Ward; and, most remarkable of all, ulceration of the sole of the foot, which, still without notable pain, gradually bores deeper and deeper until it makes a sinus through to the dorsum. This *perforating ulcer of the foot* has been long known to surgeons, and is now believed to be most frequently a symptom of tabes.

Complications.—These are early and complete loss of motor power causing ataxic paraplegia, described by some authors as a separate disease; secondly, muscular atrophy, either from invasion of the anterior cornua, or according to Déjerine from peripheral neuritis; thirdly, cerebral symptoms, such as epileptiform fits, and occasionally hemiplegic attacks; and fourthly, general paralysis with insanity. Certain tabid symptoms, particularly loss of knee-jerk, are exceedingly common in asylums, often without locomotor ataxia. Cystitis, with other pelvic symptoms succeeding the slight affection of the vesical reflex, and optic neuritis with blindness succeeding the ordinary amblyopia, may be rather regarded as ingravescence of normal symptoms than as new complications.

Course and event.—Tabes is most insidious in its origin, most irregular, slow, and uncertain in its progress, and most intractable in its course. This is one of the greatest difficulties in tracing its natural history. Patients must be watched, not for a few weeks, but for months and years. They are common in our great hospitals, and by their frequent reappearance in fresh places, and under fresh observers, they give an exaggerated impression of their numbers; but autopsies on cases of tabes are rare.

After lightning-pains, loss of knee-jerk, and myosis have lasted for several years, there may still be no ataxic symptoms. After marked locomotor ataxia is developed, there may be long delay before the advent of the paraplegic, pelvic, or cerebral symptoms which belong to the latest stage. And after apparently steady, and, by comparison, rapid progress, the disease

may suddenly be checked and remain stationary for months or years. Still, the natural tendency is to ingravescence.

Anatomy.—Todd's statement, that in cases of lack of co-ordination without paralysis the lesion is situated in the posterior columns of the cord, has been remarkably confirmed by modern methods of investigation. The lesion is sclerosis (chronic interstitial myelitis, or grey induration with atrophy) of the posterior columns of Burdach. This change is first visible as a grey streak in the deeper part of the white wedge-shaped area presented by these columns (*fasciculi cuneati*) in transverse sections. It is most marked in the upper lumbar and dorsal regions, and diminishes in extent, both above and below. Sometimes it reaches upwards as high as the restiform bodies. Its histological characters are those which have already been given of sclerosis generally. In the more advanced stages most of the nerve-fibres have disappeared, their axis-cylinders being no longer discoverable; but even in the oldest cases a not inconsiderable number of them are found with their structure unaltered, scattered through the dense connective tissue, and can be readily recognised either negatively by carmine staining, or positively by help of osmic acid. The morbid process extends over the whole of the column laterally, including the root-zones and the posterior roots themselves, which become thin, grey, and atrophic. It also reaches the surface, and the pia mater is generally found opaque, thickened, and more or less adherent to the other membranes. The posterior cornua may be also found atrophied in advanced cases; and the sclerotic change is sometimes found to have spread to the posterior median columns (of Goll) or to the lateral columns; these, however, are probably, clinically as well as pathologically, mixed cases.

Romberg described as follows the posterior sclerosis in a typical case which was seen by himself and by Froriep:—"I was not a little surprised to find that the atrophy of the spinal cord (which, compared with the fresh cord of a man of the same age, fifty-two, was only two thirds of its size) was confined to the lower part of the posterior columns and the corresponding nerves. The medullary tissue (*i. e.* white substance) of the former had almost entirely disappeared, so that they were translucent and of a greyish-yellow colour. The posterior roots of the nerves were deprived of the nervous substance (*i. e.* myelin), and presented a watery appearance." He states that the lumbar and lower dorsal regions were alone affected, and that the anterior columns and nerve-roots were free.*

There have been many speculations as to the mode of origin of this lesion. Do the nerve-fibres first undergo degeneration, and is the growth of connective tissue secondary? Or is the original process chronic inflammation of the neuroglia, leading afterwards to atrophy of the nerve-fibres, as cirrhosis of the liver leads to destruction of its secreting cells? May the disease begin as a meningitis, and spread to the cord? or is its starting-point in the sensory nerve-roots? But the most important question is, whether the lesion itself is constant.

Trousseau refers to a case of twelve years' duration, which had been seen by Duchenne and accepted as typical; but when Gubler and Luys and Duchenne himself examined the cord and its posterior roots, they could

* We have already seen that Todd distinguished the symptoms of Locomotor Ataxy from Paraplegia, and on physiological grounds placed the disease in the posterior column of the cord. His hypothesis was confirmed in the cases he examined *post mortem* (1847). Mr Stanley, of St Bartholomew's Hospital, had described the same anatomical condition in a case of tabes (probably for the first time) in the 'Med.-Chir. Trans.' for 1840.

detect no alteration there, whereas many of the nerve-fibres had disappeared from the anterior roots. Dr Alex. Hughes Bennett published in the 'Clinical Transactions,' vol. xviii, a case which presented the classical symptoms of tabes during life; but the posterior columns were found healthy. The lesion was sarcoma of the bulb and recent cerebral disease, but the posterior nerve-roots were involved. He quotes two similar cases of "peripheral tabes" from M. Déjerine. The importance of such negative evidence is enhanced by the fact that the course of the disease is commonly so protracted as to give few chances of tracing it to its end in ordinary hospital practice.

It was at one time believed that sclerosis of the posterior columns may occur as the secondary result of local myelitis without any symptoms of tabes having been present during life. In cases of Pott's disease of the dorsal vertebræ, for example, ascending degeneration is often found in the posterior columns of the cervical part of the cord; but the co-ordination of the movements of the arms and hands is in no way affected. Pierret, however, observed that the secondary ascending sclerosis just described is limited to the internal fasciculi of the posterior columns, the columns of Goll. He finds that in cases of tabes these parts of the cord are affected in the cervical region, whether the upper limbs have escaped or were secondarily involved. But sclerosis in the external fasciculi of the cervical posterior columns (the *bandelettes externes*, or *fasciculi cuneati*, Burdach's columns) occurs only when there have been ataxia of the hands and arms, and other symptoms of tabes. The conclusion is obvious that sclerosis of the tracts of Goll is a secondary ascending degeneration, and has nothing to do with the symptoms of tabes (cf. p. 489).

The sclerosis is not always obvious to the naked eye, nor until the cord has been hardened, so that one can understand how formerly observers declared the cord healthy, when locomotor ataxy had been present during life. The negative results, however, obtained by competent histologists with modern methods cannot be easily set aside, and there seems good reason to believe that tabes symptoms may depend, not only on the classical lesion of the cord, but also in certain exceptional cases on primary atrophy of the posterior roots of the lumbar and sciatic plexus. Whether other cases which have been called "pseudo-tabes" are functional neuroses which only simulate the true disease, or whether they have a peripheral origin, are still open questions.

Pathology of tabes.—Accepting the lesion in the cord, we have still the interesting but difficult question of its physiological connection with the symptoms above described. It has been shown that inability to stand or walk steadily with the eyes shut is no more than an effect of anæsthesia of the lower limbs. Moreover, a similar doctrine is held by some authorities with regard to the failure of co-ordination itself. Basing his views upon the acknowledged fact that guiding sensations contribute to the due execution of movements, Leyden maintains that ataxy is the consequence of interruption in the transmission of such sensations along the cord.

It must be admitted that some plausible arguments can be brought forward in favour of the doctrine in question. One is that the most obvious lesion is seated in a part of the cord which is (or was) believed by physiologists to convey afferent impulses. Another is the frequency with which a more or less considerable degree of anæsthesia is present, even at an early

period. And if there are cases in which cutaneous sensibility appears to be perfect, it may be replied that in many of them the sensibility of the muscles and tendons and joints has not been tested, and that this is no less essential for the even and regular execution of muscular movements. The fact that it is impossible to elicit tendon-reflexes, even at the commencement of the disease, may be pressed into the same argument.

Nevertheless, it would seem that there are facts which make it impossible for us to accept Leyden's views. One is that some cases, at least, of ataxy are unattended with any discoverable anæsthesia or defect of muscular sensibility. Both Friedreich and Erb speak positively on this point. Again, there is no close correspondence in individual patients between the degree of impairment of sensation and that of disorder of co-ordination. Lastly, there may be absolute anæsthesia without the slightest irregularity in the movements of the limbs.

A case in point was recorded by Späth in 1864, and was ten years later followed to an autopsy by Schüppel. The patient had for more than twenty years lost sensation in his arms and hands, and for six years in his feet. He was quite unconscious of the position of his limbs; and when they were moved by another person he knew nothing of it. Yet he could walk steadily, without support, and tolerably fast. When asked to raise his foot to a certain height with his eyes shut, he did it with a perfectly even and regular movement. After death the whole length of the cord was found excavated in the centre; the posterior columns in the cervical region were completely destroyed; the anterior columns were everywhere normal. It is particularly to be noticed that in this instance the muscular sensibility was absent, as well as that of the skin.

But if ataxy is not due to interruption of afferent or sensory impulses, how is it caused by a lesion situated in the posterior columns? Most writers seem to suppose that these columns may possess the special function of conveying impulses from a co-ordinating centre above, in the medulla oblongata or pons, to the motor nuclei in the cord. But it seems clear that co-ordination, instead of being affected by a single centre, must require a very complicated apparatus, probably consisting of several parts situated in different regions of the cerebro-spinal axis. We have seen that the reflex movements which are performed by the spinal cord independently of the brain are never entirely without co-ordination, and that sometimes they are co-ordinated in a very complex way. One can hardly doubt that the machinery which is brought into operation under such circumstances is also made use of when similar movements are excited by the will.

Moreover, we must remember that what is observed in ataxy is by no means an entire absence of co-ordination, such as would occur if the volitional impulses passed straight down to a group of muscles, without relation to any others. On the contrary, it is probable that the failure is very often of the slightest possible kind. Scarcely appreciable irregularities in the force which the muscles severally exert, or even in the intervals of time at which they are increasingly brought into action, would account for the symptoms in some well-marked instances of the disease. No wonder, then, that lesions of the proper motor tracts of the cord do not cause ataxy. We should rather expect it to be due to some affection just touching the outskirts of these tracts so as to injure them only very slightly. A sclerosis commencing in the fasciculi cuneati of the posterior columns seems to be just such a lesion as might produce this effect.

Again, it is certain that even in the earliest stage of the disease other parts besides the posterior columns must be involved. In no other way can one explain many of the prodroma. What is to be said as to the origin of the atrophy of the optic discs? Pathologically the morbid change in them is identical with that which is going on in the cord itself. But why should it occur in cases of tabes, and not in those of diffused myelitis? And, further, why should amaurosis from this cause often precede all spinal symptoms by an interval of several years, so that Charcot believes that the majority of the female patients admitted into the Salpêtrière for simple atrophy of the discs become sooner or later the victims of ataxy? The affections of the ocular muscles are supposed by Erb to be due to slight lesions of the nerve-roots or of their nuclei in the pons and bulb, but he does not attempt to show how it is that only motor structures are attacked, nor why the nerves concerned in moving the eyeballs suffer more than any others. As regards the last point, indeed, we may perhaps see an explanation in the delicate way in which the muscles in question are naturally balanced against one another. This renders the slightest impairment of the power of any one of them at once obvious to both the physician and the patient. The same degree of paresis elsewhere would be altogether incapable of recognition. It is said, however, that actual paralysis of the facial muscles, or of those concerned in mastication, has sometimes been observed in tabid patients.

The arthropathy which is now and then seen among the prodroma of tabes is believed to depend upon an atrophic change in the grey matter of the anterior cornua. Joffroy and Westphal have each made observations which directly confirm this opinion.

On the other hand, the boring and flashing pains are doubtless dependent upon the morbid changes in the fasciculi cuneati, *i. e.* the posterior root-zones, and the nerve-roots immediately adjacent.

There is abundant proof that in the more advanced stages of the disease the morbid process may extend in all directions within the substance of the cord. Changes in the lateral columns (the crossed pyramidal tracts) were long ago noticed by German observers, and may explain the spastic symptoms which sometimes come on in the latter stages of paraplegic ataxia.

Charcot says that the nerve-cells of the anterior cornua may be destroyed so as to cause atrophy in the corresponding muscles. When there has been an absolute loss of power in the lower limbs, almost the whole thickness of the lumbar cord may be wasted, tough, grey, and translucent.

The fact that even the prodroma of locomotor ataxy can be accounted for only on the supposition that changes exist in structures far distant from the posterior columns of the cord, makes it surprising that a definite boundary-line should be traceable between it and other spinal affections. As with Multiple Sclerosis (the disease to be described in the next chapter), it seems that the clinical features of the disease are far more uniform than would have been anticipated by the morbid anatomist. Charcot, however, admits that pains precisely like those that occur in the early stage of tabes may be observed in multiple sclerosis, in general paralysis, in chronic alcoholism, and even in diffuse myelitis or compression-paraplegia.

So difficult is it to connect the symptoms of tabes with its morbid anatomy, so few, after all, are the cases which are watched during life and satisfactorily investigated after death, and so complicated and various are both the clinical symptoms and the local distribution, that some experienced

physicians even now doubt the reality of the connection between locomotor ataxy and posterior sclerosis. (Wilks, 'Brit. Med. Journ.,' 1884, i, p. 886.)

But even if locomotor ataxy is not invariably coincident with sclerosis of Burdach's columns, the combination of disturbed movements, impaired sensations, loss of knee-jerk, defects in the eye and its muscles, and peculiar neuralgic pains, make up a "complex of symptoms" which has a beginning, a middle, and an end, and may therefore from a clinical point of view be called a disease.

Ætiology.—Among tabid patients the preponderance of males over females is very marked, but the proportion is variously stated by different observers, as 8 to 1 (Erb), 3½ to 1 (Cyon), 2½ to 1 (Carré, Schulze). Eulenburg found in 149 cases, 128 men, including 2 youths under twenty and 5 men over fifty, *i. e.* 6 males to 1 female. Among 83 tabid patients in Guy's Hospital there were 75 men and only 8 women.

Tabes very rarely occurs in persons under twenty years old, or begins in those who have passed the age of fifty. The case of an old gentleman of eighty is mentioned by Trousseau; but it is not stated how long he had been a sufferer.*

Some writers assume a family "neuropathic" disposition. One patient of Trousseau's had an uncle and an aunt insane, a brother ataxic, and a brother hemiplegic; another patient was the son of a man who committed suicide, and two of his sons laboured under certain peculiar nervous affections. In many cases, however, no predisposition to "nervous diseases," could be traced; and there can be no real pathological connection between a primary sclerosis of the cord on the one hand, and such diverse pathological conditions as paraplegia from tubercular disease of the vertebræ, hemiplegia from atheroma or Bright's disease, and paralysis from syphilis or drink.

Carré has recorded the direct transmission of tabes itself in a family of which the grandmother, the mother, her seven children, and eight others of her relations were all attacked. But such instances are very rare; and, as we shall presently see, it is possible that they may belong to an independent malady (*v. infra*, p. 539).

With regard to the exciting causes of tabes, there is equal uncertainty. Sometimes it has followed an attack of fever, acute rheumatism, or pneumonia; sometimes it has taken the place of paraplegia after diphtheria, as in cases recorded by Jaccoud and Erb. It occasionally follows injuries, not merely of the spine, but a fracture of the thigh, or a fall on the abdomen. In some cases it has been ascribed to a severe chill, as from falling into water, or sleeping on damp ground; to over-fatigue, as in soldiers after forced marching; to prolonged mental anxiety or distress; or, lastly, to venereal excesses—the supposed cause of tabes dorsalis before its symptoms were defined or its anatomy known. The wars at the beginning of the present century seem to have given rise to numerous cases of ataxy among the French and German soldiery; it is difficult to say exactly how campaigning sets up the affection, possibly by the combined operation of several of the causes above mentioned. The greater liability of men between the ages of thirty and fifty to these various morbid influences is

* Of eighty patients in Guy's Hospital one was under 20, three between 20 and 30, twenty-seven between 30 and 40, thirty-four between 40 and 50, and eighteen over 50 years of age. But in many of them, especially the older patients, the disease had begun several years before their admission into the hospital.

perhaps the reason why male adults are especially apt to suffer from ataxy; or perhaps the liability of male adults to tabes may explain its comparative frequency among soldiers.

Tabes was first definitely connected with *syphilis* by Fournier, who believed that among 102 tabid patients 74 had suffered from that disorder. Erb strongly supported this theory, and his statistics gave the proportion 61 in 99; Vulpian found 15 in 20, Buzzard 49 in 100, Gowers 29 in 50, and some writers made the proportion higher still. Thus Ross, among 20 cases of tabes, could only exclude syphilis as an antecedent in a single one. (See on this point 'Trans. Internat. Med. Congr. 1881,' pp. 32—42.)

Diagnosis.—From the nature of the early symptoms of tabes, it is evident that its diagnosis at that period of the disease is often a matter of extreme difficulty. Premonitory pains would be probably dismissed as "neuralgic" or "rheumatic," but in many cases of this kind a careful investigation would elicit other indications of the disease. There may be a slight squint, a contracted state of the pupils, or an absence of knee-jerk; or the patient may totter a little when told to walk, or to stand with his eyes shut and his feet close together. This last is a most valuable sign when we are in doubt as to the presence of any spinal affection, but much less so as between tabes and other diseases of the cord.

When the complaint is fully developed, its diagnosis is generally easy. Pronounced paralytic symptoms, with wasting of muscles, may sometimes cause one to overlook the previous existence of ataxy. Again, when there is disseminated multiple sclerosis, and the posterior columns are also attacked, it may be impossible to assign the case to one affection rather than to the other. We have seen that tabes not infrequently occurs in association with general paralysis of the insane, so as to puzzle those who happen to be more familiar with one than with the other of these two diseases.

The failure of co-ordination which accompanies disease of the cerebellum does not seem to be likely to be mistaken for the ataxia of tabes. It may generally be distinguished by the absence of other spinal symptoms, and by the presence of headache, giddiness, vomiting, optic neuritis, epileptiform attacks, and the various other signs of encephalic lesion. Moreover, the so-called cerebellar ataxy is not attended with the peculiar mode of walking above described, but with a reeling gait, like that of a drunken man.

Prognosis.—Notwithstanding what has been said above of the relentless progress of the disease, we must remember, first, that this progress is always slow, often exceedingly slow, and frequently interrupted for an indefinite period; secondly, that it does not lead to death unless one of the complications above noticed supervene. Probably most tabid patients die of phthisis, because that is the commonest of diseases, and some of disease of the heart; many of latent pyelitis or consecutive Bright's disease, from vesical complications; others again from the bedsores of paraplegia, from the effects of secondary cerebral or spinal disorders, or from accidental injuries; and others from common acute diseases which come on accidentally, as pneumonia or bronchitis. Great improvement often takes place under judicious general management, and although patients are not cured of the disease, they are relieved of its most distressing symptoms.

As a rule, the duration of tabes varies between six and twelve years,

and it may last twenty or thirty years. Some writers have described an acute form of tabes, and one case was so diagnosed in a woman who died in Guy's Hospital six weeks after admission, and about ten months from the beginning of her illness. But in this instance there was softening of the anterior columns in the lumbar region, as well as sclerosis of the posterior columns.

Erb mentions two cases, in each of which almost complete recovery took place. One patient afterwards held a Government appointment for several years; the other, who had been unable to get about without help, and had suffered from incontinence of urine with cystitis, regained the power of walking for three or four hours at a time, and of holding his water for five or six hours; he married and took the command of an ironclad frigate.

The course of the disease is not always steadily progressive; it is often quiescent for a long period. Generally it is better in the summer than in the winter. In some cases, it scarcely seems to shorten the patient's life.

Treatment.—When tabes is fully developed it rarely is cured by treatment of any kind.

The general management of the patient is of great importance. He must be carefully protected from cold and damp by flannel underclothing. He must avoid all bodily and mental exertion. Dr Radcliffe thinks it advisable that crutches should be used in the early stages, so as to save the lower limbs as much as possible. It has been suggested that maintaining absolute rest in bed might favour the subsidence of the disease, and Dr Weir Mitchell brings positive evidence in favour of this plan. Dr Gowers recommends a sea voyage. Those who can afford it will find great benefit from spending the winter and spring in the South of Europe, or still better in the Azores or the West Indies. *Abstineto tabidus a vino et venere.*

Erb recommends galvanism applied as a continuous current to the spine. He uses a moderate number of cells, for three to six minutes at a time, once daily. Of sixty-six cases thus treated, twenty-five received no benefit, forty-one were more or less improved. He also galvanises the peripheral nerves.

Locomotor ataxy is among the maladies for which persons resort to the various spas. Almost all writers are agreed that hot baths are injurious, many believe that cold baths are useless. Erb says that any temperature above 58·5° F. is undesirable, and that the patient should bathe only once in two or three days, and remain in the water not above fifteen or twenty minutes. Nauheim and Rehme are the places which seem at present to have the highest reputation. Peat baths are also recommended. But the figures given by Erb appear to show that the "cold-water cure" with packing, friction, and douches is quite as useful: of nineteen patients treated in this way, sixteen are said to have been benefited.

Among the symptoms of tabes, the pains most need relief. Sinapisms, blisters, liniments of chloroform or belladonna, veratria ointment, may be of some use; but often one cannot avoid frequent injection of morphia. Of late years *antipyrin* has been used with much success, and the writer is disposed to believe that this is the most useful application of the drug. *Exalgine* (methyl-acetanilide), recently recommended by Dr Fraser of Edinburgh for facial neuralgia, would probably also be of service. ('B. M. J.,' Feb. 15, 1890.)

As a specific alterative, the drug of most value is perhaps the nitrate of silver; it often fails entirely, but sometimes does great good. We generally

order a quarter of a grain three times a day. Belladonna and bromide of potassium seem to be useless, and strychnine to be injurious. Calabar bean, arsenic, and ergot in early cases, may perhaps do good.

When tabes follows syphilis, we should certainly give the patient a fair trial of *antisyphilitic remedies*. No doubt they often fail, and in the later stages of the disease probably do more harm than good, but the only cases of tabes seen by the writer which were cured were treated by mercury.

One was that of a man, about forty-five, who was admitted into Guy's Hospital with marked ataxy, loss of knee-jerk, and other symptoms; indeed, myosis was the only important one absent. He had the signs as well as the history of syphilis, acquired long before. Under a mercurial course he improved so much that he went out, walking well, free from pain, and in fact cured, though the knee-jerk had not returned. He stated while in the ward that he had some years before suffered from similar symptoms (ten years or so after primary syphilis), and had been cured by the same treatment. Another case is that of a patient who has been under observation for five or six years. He had syphilis years ago, and still has occasional tertiary sores on one heel. The worst symptoms of tabes have disappeared under mercury and iodide of potassium, but he still has minutely contracted pupils and loss of knee-jerk. Neither case is conclusive, but this method is well worth trying when we know that syphilis preceded the nervous symptoms.

Mechanical *stretching* of the great sciatic nerves has been used as a remedy for tabes. It was first performed by Langenbuch with apparent success; but when the patient died, the cord was found healthy. On the whole, the good effects seem to be doubtful, and the drawbacks and dangers undoubted. See an interesting paper by Dr Cavafy, with eighteen cases, beside one of his own ('Brit. Med. Journ.,' Dec. 10th, 1881); also Mr. Bowlby's record of cases (loc. cit. p. 360).

Another somewhat analogous plan of treatment has lately been extensively tried. It consists in *suspension* of the patient by the head, so that the weight of the body pulls upon the attachments of the cord to the spinal column by its membranes and nerve-roots. This method was introduced by Dr Motschukowsky, of Odessa, and has been tried on a large scale by Charcot and other physicians on the Continent, in England, and in America. Great care is needed in carrying it out, for in more than one case it has caused death, and in one of these the autopsy showed the absence of posterior sclerosis. The suspension must be gradual, and at first for less than a minute at a time.

Many instances have been recorded in which the lightning pains of tabes have disappeared under this treatment, while faculty of locomotion and virile power have returned. But in many more these effects have been absent or transient; and the spontaneous variations in the symptoms of this disease during its slow and irregular course are so great, that improvement in symptoms, and particularly in subjective symptoms, must be accepted with great caution as proof of the efficacy of this and of any other plan of treatment. A patient on whom the writer carried out suspension said that he was greatly benefited; but the improvement was very doubtful to those who watched the case, and he had been equally gratified by the good effects of a few grains of salt, given as a control experiment while his symptoms were being recorded. In the absence of conclusive evidence of the value of the method,

interest attaches to experiments made on the dead body, which seem to prove that its effect is to straighten the vertebral curves, and to relax rather than stretch the spinal cord.* It may have some effect possibly on meningeal adhesions, possibly on the circulation of the cord; but if so we cannot say beforehand whether either would be beneficial or the reverse. On the whole, it seems at present likely that the method of suspension will be abandoned as of doubtful service, and not unattended with risk.

FRIEDREICH'S DISEASE.†—Friedreich first described in 1863, and more fully in 1876, an *hereditary* (or rather "*family*") form of tabes which presents several peculiar features. He recognised it in only three families; in each of them it attacked in succession several children of the same parents, in all nine patients, of whom seven were girls. It began unusually early, at or near the age of puberty, between the thirteenth and the eighteenth years. Among eleven cases Rütimeyer found seven boys and four girls, the ages being all under puberty (1883). Since then further observations have shown that this form of tabes occasionally occurs in two generations, much more often in several brothers and sisters; that it begins in childhood as early as four, at puberty, or in early adult life as late as twenty-four; and that girls are almost as often affected by it as boys.

The disease is attended with remarkably little disturbance of sensation; the prodromal pains in the limbs and the later girdle-sensation round the body are absent or slight, and there are none of the ocular symptoms of tabes.

A peculiar form of *nystagmus*, however, is sometimes present; it is characterised by occurring only when the patient endeavours to fix his eyes upon an object before him; the movements are always bilateral, and are comparatively slow, being repeated two or three times in a second.

Ataxia begins in the legs, but the upper limbs show a loss of co-ordination at an early period.

In every instance there has been a remarkable disorder of *speech*. The utterance is described as having been at first slightly lisping (Calloud), then as irregularly interrupted and stammering, and finally as almost unintelligible. It is altogether unlike the slow accentuated articulation of a patient suffering from multiple sclerosis. The skin and joints are unaffected. Knee-jerk is absent. The senses and the mental faculties are perfect.

Choreiform movements of the face and limbs have been observed sometimes as the first symptom, so that the disease has been mistaken for chorea.

The duration of these cases has been very long; in one instance it was more than thirty years. A curious circumstance is that five of the patients died of enteric fever.

Clinically it resembles insular sclerosis or functional spasmodic disorders as much as it does ordinary tabes.

Sclerosis of the posterior columns has been found after death, but it has been combined with sclerosis of the direct cerebellar and of the lateral columns. In two cases (Friedreich, and Kahler and Pick) the entire cord was remarkably small.

The author had once under observation a typical example of Friedreich's ataxy. The patient, a medical man, was first noticed to be uncertain in

* See Dr Cagney's paper, 'Trans. Roy. Med.-Chir. Soc.,' Jan., 1890.

† *Synonyms.*—Hereditary ataxy—Juvenile ataxia—Hereditary tabes in children—Hereditary ataxic paraplegia (Gowers)—Early tabes with nystagmus occurring in families.

his gait when about sixteen years old. He was one day walking across a plank in a house that was being built, when he tottered; afterwards he remembers that he had to be careful not to go near the edge of the path where it was raised above the road. But he came to the hospital as a student, passed his examinations, went into practice, and took a wife. At the age of about twenty-five he was obliged to give up his profession on account of increasing inability to walk or ride; he became unable to write, and his speech became laboured and thick. In 1878, when he was thirty, he could just walk from one room to another with assistance; there was even then but little impairment of sensation in his feet; when he moved his lips in speaking a large number of the facial muscles were thrown into action, so that the mouth became drawn outwards into a meaningless smile; the words were tolerably distinct, but some syllables were slurred over, and others pronounced too strongly. The nystagmus was very obvious when he was told to direct his eyes to either side. He was a member of a highly neurotic family, and one sister suffered from some form of paralysis.

We have since had three brothers in Guy's Hospital suffering from this juvenile family type of ataxia with nystagmus. The case of one of them is reported in the forty-fourth volume of our 'Reports' (1887), and is identical with one of the five cases in the same family recorded by Dr Gowers in the fourteenth volume of the Clinical Society's 'Transactions.' One of them died under the late Dr Moxon's care. Sections of the cord were made by Dr Pitt, and are described and figured by him in the article referred to. They showed sclerosis of the posterior columns (both Burdach's and Goll's), with similar change in the crossed pyramidal and cerebellar tracts. The anterior columns, which also have been sometimes found affected, were free, and the grey substance normal. The lesion was most marked in the cervical and dorsal regions, but the cord in its whole length was remarkably small, as if from congenital atrophy.

Dr J. A. Ormerod has published a critical digest of the recorded cases of this remarkable form of disease in 'Brain' for April, 1884; and Dr Bury in the ninth volume of the same journal. Dr Everett Smith (quoted by Gowers) has collected no less than fifty-seven cases in the 'Boston Medical and Surgical Journal' for Oct. 15th, 1885.

INSULAR SCLEROSIS*

Tremor oritur a spiritibus animalibus per apertas valvulas in oppositos musculos influentibus, qui spiritus . . . istas non claudunt; ut ventus semiapertam januam non claudit.—JOH. DOLÆUS, 'Medica dogmatica,' 1691.

History—Anatomy—Symptoms—Ætiology—Diagnosis—Prognosis and treatment. Comparative frequency of diseases of the spinal cord in hospital practice.

IN the great illustrated work of Cruveilhier on pathological anatomy, published between 1835 and 1842, there are figures taken from four different patients representing the spinal cord affected with what he termed "grey degeneration," in the form of patches scattered irregularly through its substance. In two of these cases he also gave the clinical features of the disease. About the same time Carswell figured the same lesion in his 'Atlas of Pathological Anatomy' (1838), and Marshall Hall described the characteristic symptoms in his 'Diseases of the Nervous System' (1841). Afterwards various writers from time to time referred to similar cases, but it was not until about the year 1866 that a series of observations were made at the Salpêtrière, from which it results that the disease in question is characterised clinically by a remarkable and definite group of symptoms, so that it can often be diagnosed with certainty. In these researches Charcot took the principal part. The subject was soon taken up in Germany, but in England it seems to have been neglected until in 1873 the late Dr Moxon published a case in the 'Lancet,' and in 1875 a paper containing eight cases in the 'Guy's Hospital Reports.'

Cruveilhier's drawings show that the bulb and the pons are affected as well as the cord, and the same morbid change has since been found in the hemispheres. Insular sclerosis might therefore with equal justice be placed among cerebral diseases; indeed, several of its symptoms are cerebral. Hence its most appropriate place seems at present to be between diseases of the cord and those of the brain.

Some writers have wondered that so remarkable a disease so long remained unknown, but its slow and hopeless course renders it little likely to go on to its fatal termination within the wards of ordinary hospitals. The author was unable to find a single case which could be assigned to disseminated sclerosis in the pathological records of Guy's Hospital between 1854 and 1873. In the ten years 1880 to 1889 inclusive, we had twelve cases.

Histology.—As a rule, the morbid change is at once obvious when the nervous centres are examined in the *post-mortem* room. Bourneville has, however, recorded one case in which they appeared to be healthy until the microscope was used. The patches of sclerosis are rounded or elliptical or

* *Synonyms.*—Multiple cerebro-spinal sclerosis—Disseminated sclerosis—*Fr.* Sclérose en plaques (Vulpian)—Sclérose en îlots multiples et disseminés (Liouville)—*Germ.* Multiple inselförmige Sclerose (Leube)—Herdweise Sclerose.

irregular in form. In the cord they come to the surface, and are sometimes seen through the pia mater. In the brain they affect the white substance of the hemispheres and scarcely ever penetrate into the cortex, but they are often visible on the surface at the base. In the basal ganglia, the pons, and the cord they show no tendency to spare the grey matter, or to affect one column or cornu more than another. Hence Insular Sclerosis is not a "systemic" disease in its distribution, nor focal, nor transverse, but essentially indiscriminate or scattered.* The grey patches are often scattered in large numbers throughout the white matter of the hemispheres. The cerebellum seldom contains many of them. A similar change may be present in the olfactory bulbs and in the roots of the various cranial and spinal nerves. In the peripheral trunks it has not yet been recognised: Moxon found the brachial plexus in one case normal on each side.

The patches generally feel hard, but recent ones are said to be softer than the rest and of a deeper grey tint, whereas the older ones are yellowish grey and less translucent. They become pinkish when exposed to the air. They may either project somewhat above the cut surface, or lie at the same level, or be slightly depressed. They vary from a microscopic minuteness up to the size of a hazel-nut or more. Histologically they present the characteristic appearances of chronic interstitial or grey degeneration (p. 441). Charcot says that there are fewer corpora amylacea, and that a distinction is afforded by the way in which naked axis-cylinders persist even when the neuroglia has become highly fibrillated, but the last point is contested by Erb. It is to be observed that in thin sections the affected parts are found not to be so sharply defined at their edges as they appear to the naked eye; on the contrary, the sclerosis fades off very gradually into the healthy tissue. Moxon remarked that granule-cells are more numerous at the circumference of the patches (and even in the apparently normal brain-substance beyond) than towards their centres. Deiters' cells and corpora amylacea are present.

Symptoms.—That the clinical features of disseminated sclerosis should vary in different cases is no more than we should expect from the irregular distribution of the patches, which would account for the utmost irregularity in the symptoms. Charcot, indeed, speaks of it as polymorphous in its clinical aspect. He and other French writers describe, in addition to the "cerebro-spinal" form of the disease, separate "spinal" and "cerebral" forms, but it is admitted that such distinctions can seldom be made at the bedside. Moreover, notwithstanding individual variations, the symptoms present a degree of uniformity which is very remarkable. This uniformity Dr Moxon regarded as "a constant average result of the numerous points of disease;" but the explanation leaves much unexplained.

We may refer disturbance of the special senses to sclerosis of their nerves, spasms to implication of the lateral columns, and mental symptoms to cerebral lesions; but neither in this disease nor in spastic paraplegia could we have predicted the symptoms from the anatomical lesions.

The tremors, the nystagmus, and the peculiar speech—the most constant symptoms—are quite unexplained.

* See an excellent plate in Dr Bramwell's work, which shows the distribution of the patches of sclerosis throughout the cord in a well-marked case, and the way in which they stain with carmine. This is an example of the larger-sized islands. In Dr Moxon's paper in the 'Guy's Reports' (1875) is a coloured drawing of the centrum ovale, with insular sclerotic patches, and a microscopic section of the cord.

Early prodromal symptoms are sometimes observed which, according to Dr Buzzard, closely simulate hysteria—loss of power or numbness in one limb, pins and needles, &c.

The first developed symptom is a sort of *tremor*, which until recently was confounded with paralysis agitans. There are, in fact, records of two cases diagnosed as examples of that affection by Skoda and by Hasse respectively, in which patches of sclerosis in the nervous centres were discovered after death. Careful observation, however, has shown that in paralysis agitans the trembling movement is less extensive, more regular, and more rhythmical than in insular sclerosis. In the latter disease, when the hand is raised, the oscillations increase in amplitude as it gets further from the side, until they greatly embarrass and disturb the patient. If he is asked to carry a cup to his lips it becomes more and more violently shaken until at last it is dashed against the teeth, so as perhaps to spill its contents. In slight movements the tremor is not so marked; the handwriting, although shaky, long remains legible.

There is no agitation of the muscles as long as they are at rest and supported. When the patient is lying in bed, one could not tell that anything is the matter; but as soon as he is asked to sit up, the arms, and then the head and the neck, begin to oscillate. When he is resting in a chair the arms are quiet, but the head may still continue to show a slight tremulous movement. If he attempts to get on his feet the whole trunk and all the limbs become violently shaken, so that in severe cases standing is out of the question. Mental emotion or excitement aggravates the tremor considerably. According to Erb there are some cases in which it continues even when the patient is lying still, but this is altogether exceptional.

Charcot has suggested that this remarkable symptom depends upon the persistence of naked axis-cylinders in the parts affected with the sclerosis, and conceives that the transmission of volitional impulses may become interrupted and, as it were, jerking; but such a view appears unlikely. Ordenstein is disposed to think that the tremor depends in some way upon the presence of patches in the pons and still higher in the motor tracts, and Erb says that an analysis of twenty-two recent cases tends to support the notion of the connection of this symptom with abundant islets in the mesencephalon.

A second characteristic symptom of disseminated sclerosis is a peculiar *affection of the speech*. The pronunciation is slow, accented, laboured, as if the utterance of each syllable was a great effort. And yet it is not distinct; Charcot says that the letters L, P, G, are especially apt to be slurred over; and sometimes, though rarely, two or three syllables are run together. German writers describe the articulation as "scanning," each syllable being pronounced separately. In advanced stages speech may become quite unintelligible. Erb further remarks that the voice is pitched in a monotone; or, as in one case that came under his observation, that there may be a rhythmical alternation of notes at a definite musical interval. Leube found with the laryngoscope that the vocal cords, although capable of closing the glottis, were yet apt to relax, and liable to changes of tension ('Deutsches Archiv,' 1871).

Thirdly, *nystagmus* is commonly present. It, however, differs somewhat from the ceaseless oscillation of the eyeballs with which ophthalmic surgeons are familiar. As Dr Moxon pointed out, it is absent so long as the eyes are at rest, showing itself only when they are directed to an object. In

other words, it is identical with the affection which is observed in Friedreich's hereditary ataxy (see p. 539); indeed, both Friedreich and Erb have suggested that the anatomical lesion may be the same, but rather as a matter of speculation than from direct observation.

In addition to these distinctive symptoms, there are others which belong to disseminated sclerosis in common with other diseases.

Subjective sensations of *numbness* and other paræsthesiæ are not uncommon, although they were absent in Dr Moxon's eight cases; anæsthesia, or impairment of tactile sensibility, can seldom be made out.

The gait at an early stage is often *spastic* in character. The cutaneous reflexes are usually normal, but sometimes they are increased. Knee-jerk is exaggerated, and clonus often present. Afterwards there is a marked tendency for the lower limbs to become rigidly extended and adducted; this condition being at first transitory, but afterwards permanent. Moxon mentions that one of his patients was obliged to sit in an elbow-chair, so that when her legs stiffened she could hold on to the arms and save herself from sliding down on to the floor. Such early contractions are painless, but at a later period a painful flexion of the limbs is not infrequent. In ordinary cases there is no reaction of degeneration, and the muscles react to both currents until they finally undergo atrophy.

The characteristic symptoms of locomotor *ataxy* are now and then present in disseminated sclerosis; this is supposed to depend upon the existence of patches in the posterior columns of the cord.

After a long time complete *paraplegia* usually develops itself. The functions of the pelvic organs are at first but little interfered with. Constipation is frequent; and Moxon refers this in part to weakness of the abdominal muscles. The sexual organs retain their powers, and there is no retention of urine until the latter stages of the disease.

Among other *cephalic* symptoms amblyopia often occurs, with narrowing of the field of vision and achromatopsy, and transient *diplopia* is not infrequent at an early stage of the disease. Charcot insists on the fact that the impairment of sight rarely goes on to complete amaurosis, even when the optic nerves have been found sclerosed in their whole thickness; he refers this to the persistence of naked axis-cylinders in the midst of the diseased tissues. The optic discs are generally normal unless there is total blindness; in the latter case they have sometimes been found in a state of white atrophy. Any degree of optic neuritis with disturbed vision would be an important indication of an early stage of this singular disease. Headache, giddiness, and sleeplessness are not uncommonly present.

The spirits are often depressed, and the *memory* and *intelligence* become gradually impaired. The patient bursts into uncontrollable laughter, or sheds tears without any cause. Charcot insists on a peculiar *facies*; the expression is, he says, vague, uncertain; the lips droop and are half opened; the features have an air of dulness or stupidity. Sometimes there is an actual insanity. Charcot mentions two patients who exhibited the *délire des grandeurs* supposed to be characteristic of general paralysis; in others there has been melancholia, with refusal of food.

A curious feature, which is observed in about one fifth of the cases of disseminated sclerosis, is the occurrence of attacks of stupor, followed by transitory hemiplegia. They are attended with flushing of the face, a rapid pulse, and a temperature raised to 104° or even 106°. The coma lasts for a day or two, and then passes off into sleep. Sometimes, however, a

seizure of this kind is directly fatal; one of Moxon's patients died so in about twenty-four hours. Such an attack is said always to lead to an aggravation of the general symptoms of the disease, even when it is recovered from.

Lastly, in some exceptional cases atrophy of muscles is observed, affecting the upper or lower limbs, the face, or the trunk. At the same time their electrical excitability, which ordinarily remains normal, may be lowered; and the natural reaction is doubtless replaced by the "reaction of degeneration." Charcot says that the characteristic symptoms of bulbar paralysis sometimes show themselves, and dysphagia or paroxysmal dyspnœa may then be the immediate cause of death.

It is obvious that many of the above symptoms of insular sclerosis are due to the disease invading the lateral or the posterior columns, the anterior cornua or the nuclei in the bulb and mesencephalon, so as to produce the symptoms which belong to tabes, spastic or atrophic paralysis, and so on. The paraplegic period, again, with bedsores, retention of urine, and other pelvic symptoms, belongs to the last stage of all chronic diseases of the cord.

Ætiology.—Disseminated sclerosis is now ascertained to be equally frequent in either sex, although Charcot found twenty-five females to nine males among the first thirty-four cases which he collected. The age at which it most frequently develops itself is said to be between twenty and twenty-five years, but it has been several times observed at puberty, and instances of its occurrence in childhood have been recorded. It has not hitherto been known to begin in persons over forty or forty-five years old. Now and then it has appeared to be hereditary or has occurred in two children of the same parents.

Like other chronic affections of the cord, it has been ascribed to "chills," to falls and blows on the head, and to moral shock, mental excitement, or, as it is now called, "worry" and "over-strain." Bauwinkel records a case in which a man fell into water and allowed his clothes to dry upon him, three days before the first symptoms of insular sclerosis appeared. Charcot cites instances in which the disease began during convalescence from enteric fever, cholera, or smallpox. All that we can conclude is that these several antecedents do not *protect* a person from insular sclerosis.

Diagnosis.—This is not invariably, or even generally, an easy matter. Charcot says that trembling movements, precisely like those which are present in this disease, may occur not only in mercurial poisoning (when the history will in most cases remove all doubt), but also when the lesion is a "chronic cervical meningitis with cortical sclerosis," and even where it is a "primary or consecutive sclerosis of the lateral columns." The presence or absence of the other characteristic symptoms of these affections must guide the observer to a decision so far as they are concerned.

The diagnosis from paralysis agitans is not the most difficult, for that disease occurs in older patients, the tremors continue when the muscles are at rest, and affect the hands more than the feet, their amplitude is smaller and more uniform, and their rate is generally quicker. Moreover, in paralysis agitans the shake precedes the palsy, whereas slight loss of power in walking is usually the earliest symptom of insular sclerosis, as in a typical case recorded in 1875 by Dr Goodhart, in a woman of thirty-eight, with characteristic lesions after death ('Path. Trans.,' xxvii, p. 17).

Diagnosis of insular sclerosis from tabes, from Friedreich's ataxia, from

spastic paralysis and bulbar palsy, probably means only that we must distinguish clinically in mixed forms of disease the characters of one type which are complicated by some usually associated with another. Corresponding coincidences of posterior, lateral, anterior, or bulbar sclerosis with disseminated cerebro-spinal sclerosis have been found *post mortem*.

Cases presenting during life the characteristic speech, nystagmus, tremors, and increased knee-jerk have after death failed to show insular sclerosis of the brain and cord. Some of these were probably cases of general paralysis of the insane. (See Dr Bristowe's cases in his collected papers on diseases of the nervous system).

Great caution is required to avoid mistaking hysteria for disseminated sclerosis in women; and Moxon says that he has seen several patients about whom he has for a time been in doubt, but who have before long completely recovered. See also Dr Buzzard's instructive address before the Neurological Society on the "Simulation of Hysteria by Organic Disease of the Nervous System" ('Lancet,' Jan. 23, 1890, and 'Brain,' Feb.).

When tremors are absent, the case may appear to be an example of some other spinal affection. Charcot supposes that in these cases the symptom in question was present at an earlier period; but Erb refers to several in which it was wanting throughout the whole course of the disease. Thus he cites Westphal and Killian as having recorded instances in which the only symptoms were dementia and paralysis with contractions of the limbs.

Course, prognosis, and treatment.—This remarkable disease usually begins insidiously, with slight giddiness, affections of sight or hearing, weakness in the legs, and difficulty in walking. As an exception it begins more suddenly, perhaps with an epileptiform fit.

The course of disseminated sclerosis is slowly but irregularly progressive, although remissions in the symptoms may occur spontaneously or under treatment. The average duration of the disease is said to be from five to ten years, the most rapidly fatal termination being in one year from the commencement, and the latest at the end of seventeen years. This, however, partly depends upon the nature of the symptoms. Charcot says that cases in which spinal symptoms alone are present may go on for twenty years or longer. If death does not occur in one of the ways already mentioned, it is generally by the supervention of some intercurrent disease, such as pneumonia, pleurisy, dysentery, œdema of the glottis, or more frequently by ordinary tubercular phthisis.

Few remedies have been found of any service. Charcot thinks that the administration of strychnia or of nitrate of silver has sometimes diminished the tremor and the weakness of the lower limbs, but only for a time. Some observers have seen transitory benefit result from the cold-water treatment, from galvanism, or from the subcutaneous injection of arsenic. Many of the drugs given probably do harm. The only rational treatment is to alleviate individual symptoms, and to make the patient as comfortable as he can be made.

Before passing on to our next subject, Diseases of the Brain, it may be of interest to give the reader some notion of the relative frequency of the Diseases of the Spinal Cord which we have been describing, as seen in the wards of a large London hospital. For the following statistics the writer is indebted to his former house-physicians—Dr Halstead, of Ramsgate, and Dr E. W. Goodall, now Medical Registrar—1880–1889.

Among 301 consecutive cases of diseases of the spinal cord there were 167 of *paraplegia*—105 men to 62 women.

These might be classified as follows, on the plan adopted in the chapter with that heading.

(*a*) Paraplegia due to intrinsic causes: 78 cases, 25 with symptoms of acute *myelitis* (probably in two or three cases hæmorrhage into the meninges or cord), and the remainder chronic cases with more or less marked characters of *spastic paraplegia*. Three of the latter were congenital cases in children.

(*b*) Paraplegia due to extrinsic causes: from compression by *caries* of the spine, 22 cases; from *cancer* of the spine, 3. Paraplegia due to *tumours* of the cord or meninges: 4 cases—tubercle of the cord, glioma of brain and cord, and two meningeal tumours.

(*c*) Eighteen cases of paraplegia were diagnosed as due to *syphilis*; 12 were *alcoholic*, probably not all of them due to peripheral neuritis, and 30 were classed as *hysterical* or *functional*. One of the latter, in a man under the writer's care, was a case of divers' paraplegia.

In each variety, except hysterical paraplegia, there were more male than female cases.

There were 39 cases of *atrophic paralysis*, 25 male and 14 female. Of these, 3 were due to chronic cervical meningitis, and were thus distinct from the rest; only 15 were cases of infantile paralysis, but more were treated in the out-patients' room; there were 2 of adult spinal atrophic paralysis, 11 of progressive muscular atrophy, 1 of bulbar palsy and 8 of pseudo-hypertrophic paralysis in children.

Lastly, there were 83 cases of tabes, including 2 of Friedreich's disease, of which 75 occurred in men; and 12 of Insular sclerosis—5 men and 7 women.

There were no cases recorded of Landry's paralysis, nor of hemi-paraplegia. Cases of lead palsy were numerous, but were not classified among other forms of paralysis, and many of them were treated as out-patients.

DISEASES OF THE BRAIN

CEREBRAL DISORDERS DUE TO LOCAL LESIONS OF THE ARTERIES

'Αποπληξίη ὅλου τοῦ σκηνεὼς [*i. e.* σώματος] καὶ τῆς αἰσθήσιός τε καὶ γνώμης καὶ κινήσιός ἐστι παράλυσις. Ἦν δὲ κατάρχῃ κεφαλή, ἐπὶ μὲν τοῖσι δεξιοῖσι τὰ λάια παραλύεται δεξιὰ δὲ ἐπ' ἀριστεροῖσι.—ARETÆUS, A.D. 70.

"Paralyticos plerumque altero latere captos spectamus. Hæc hemiplegia vocatur. Fertur, et sane plurimorum jam medicorum observationibus confirmatur, latus adversum ab eo in quo cerebri vitium est, sic resolvi."—GREGORY'S *Conspectus*, 1832.

Anatomical effects of Obstruction of the cerebral arteries by embolism, thrombosis, syphilis, red and white softening—Effects of Hæmorrhage—Renal disease, atheroma, miliary aneurysms, as causes of cerebral hæmorrhage.
HEMIPLEGIA—*Its characters and extent—The local lesion—Course and sequelæ.*
APHASIA—*Amnesia—Agraphia—Their characters, seat, and physiology.*
APOPLEXY—*Symptoms, onset, and course—Determination of locality—Its diagnosis from injury, narcotic poison, alcoholic intoxication, pyæmia, uræmia, epilepsy—Diagnosis of the anatomical causes of apoplexy.*
Treatment of apoplexy and hemiplegia.

THE brain differs from the spinal cord in two particulars, which have an important influence on its pathology. Its lateral halves, instead of being closely united, are separated from one another, and expanded each into an independent mass. Its arteries, beside the minute twigs which run in from the pia mater to supply the cortex, are large trunks with well-defined areas of distribution. Consequently an important group of cerebral affections depend directly upon disease of the arteries of the brain, and their rupture or occlusion; and in these affections the symptoms are in a marked degree unilateral; any paralysis, or spasm, or loss of sensation in the body or limbs, is confined to one side or the other.

The result is either *Hemiplegia*, or loss of power in one arm and the corresponding leg; or the lesion is so severe as to interfere with both sides of the brain—a condition which has long been known as *Apoplexy*. Thus the affections in question contrast broadly in their features with those of the cord, which, as we have seen, are usually attended with a paraplegic form of paralysis, and are commonly slow in endangering life.

The boundary line is not fixed at the foramen magnum, for the medulla oblongata—which the physiologist recognises as the continuation upwards of the medulla spinalis—is subject to at least one of the diseases of the cord, that already described as bulbar paralysis. The pons may be the seat of lesions which are due to changes in its arteries, and cause a fatal form of apoplexy. In fact, both the bulb and the pons are liable at once to the special affec-

tions of the cord, and also to lesions of their blood-vessels, exactly like those of the higher cerebral centres.

To avoid the necessity of going over the same ground more than once, we will first give an account of the several diseases of the cerebral blood-vessels, of their respective causes, and of their anatomical effects; then we will proceed to discuss the symptoms to which they may give rise; and finally we will endeavour to show how we can diagnose these lesions from one another, and from affections of a different nature.

The vascular lesions in question fall into two groups: (1) those in which there is an arrest of the flow of blood through one or more of the cerebral arteries; and (2) those in which an artery is ruptured, allowing the blood to escape into the brain or beneath its membranes. The former group includes chiefly *embolism*, *thrombosis*, and softening from *syphilitic disease* of the cerebral arteries; the latter the various forms of *cerebral hæmorrhage*.

Obstruction of the cerebral arteries.—This may occur in various ways, which require to be separately considered.

(1) The obstruction may arise *outside the cranial cavity*. An example of this unusual event was Sir William Gull's case, in which arteritis deformans of the arch of the aorta led to complete obliteration of the innominate and left carotid arteries, so that the left subclavian artery alone was left to carry on the circulation in the brain. The patient, a woman aged forty-one, died paralysed and insensible; and certain parts of the cerebral centres were found to be in a state of softening. Some years ago a woman affected with carotid aneurysm was admitted into Guy's Hospital, who had nearly a year before been attacked with hemiplegia and loss of speech. Mr Durham performed the operation of ligaturing the common carotid artery, whereupon she regained to some extent the power of speaking. However, she died soon afterwards, and it was found that the aneurysmal sac extended upwards by the side of the internal carotid artery, and so pressed upon it that until the tension was lowered by ligature of the vessel below, the blood which reached the internal carotid from collateral sources had been unable to pass on to the brain. Much more commonly it is *after* ligature or compression of the carotid artery that hemiplegia sets in. Five or six cases of this kind have occurred at Guy's Hospital within recent years. In most of them the paralysis was the direct result of the operation; but in one instance it did not occur until three days afterwards, when the patient became suddenly hemiplegic while the dressings were being changed. It is possible that in those cases in which cerebral symptoms manifest themselves under such circumstances the communicating arteries which make up the circle of Willis may be abnormally small.

(2) *Embolism.*—In the immense majority of cases the cause of arrested circulation in the cerebral arteries lies within the skull; and it is generally an embolus—a clot derived from some distant source, and washed into the vessel by the blood-stream. Most frequently the starting-point of the morbid process is in vegetations on either the mitral or the aortic valves, the result of simple or ulcerative endocarditis. At an autopsy made in 1877 a rough friable mass of calcareous deposit was exposed upon the surface of the mitral valve, and a fragment of the same material was found wedged in one of the Sylvian arteries.

Sometimes stenosis of the mitral orifice is present, without roughness of its surface; the clot is in such cases almost always derived from

the dilated left auricle, where thrombi form in the recesses between the fleshy columns of its appendix; occasionally the formation of *ante-mortem* coagula in the chambers left of the heart may be independent of any valvular affection. In yet more rare instances the primary lesion is in the aorta itself. We have had at Guy's Hospital two such cases: in one there was a patch of softening thrombus which adhered to a diseased part of the wall of the great artery; in the other ulceration existed.

In the records of inspections during twenty-three years, there were forty-seven cases in which there was reason to believe that embolism of one of the cerebral arteries had occurred. It is true that in a very large number of them (twenty-one) no clot was discovered in any of the vessels at the autopsy, the interpretation of the cerebral symptoms as due to this cause being based upon their occurrence in persons who had disease of the cardiac valves, and upon the presence of such changes in the brain as are known to result from embolism. All writers admit that anatomical proof of plugging of the arteries is wanting in some fatal cases, especially when life has been prolonged for a considerable period. They suppose that the clot has in the meantime undergone absorption. That this may be the case is likely enough, but another possible explanation is that the spot at which the embolism was impacted may have been one of the bends of the carotid artery within the petrous bone or in the cavernous sinus, or where the vertebral artery winds round the arch of the atlas—parts which generally escape examination.

The vessel into which the clot passes is almost always one of the Sylvian arteries. In twenty-three cases embolism of these vessels was found at the autopsy, as against three cases in which its seat was the vertebral artery. The fact that embola entering the carotid are almost always carried into the Sylvian branch is doubtless owing to its course being more in a line with the trunk than that of the two other branches. Writers have stated that the left Sylvian artery is much more apt to be plugged than the right one, and an explanation for this supposed fact has been found in the difference of the angles at which the innominate artery and the left carotid artery respectively leave the aorta. But this statement is not confirmed by our experience at Guy's Hospital, for among twenty-one cases of embolism limited to the Sylvian artery of one side there were eleven in which the left one was affected, and ten in which the clot entered the right artery, a difference too slight to be noticed. The error (if error it be) has arisen from the statistical collection of miscellaneous cases from Journals and Transactions; the interest attaching to aphasia—a frequent result of embolism on the left side—must have often led to the publication of cases in which that symptom was present, while many of those in which it did not occur have been left unrecorded.

Our records do not bear out another statement often made—that cerebral embolism is most frequent in very young adults; for 12 cases occurred between the ages of thirty-one and forty years, as compared with 10 between twenty-one and thirty, 9 between forty-one and fifty, 8 between fifty-one and sixty, 6 between eleven and twenty, and 1 above the age of sixty. A slight preponderance of cases in male over those in female patients was probably accidental.

(3) *Thrombosis* of the cerebral arteries—the formation of clots *in situ*, independently of any extraneous source—is far less frequent than embolism. It is generally secondary to disease of the arterial wall, either atheroma, or

thickening with calcification. Occasionally it may perhaps be a direct result of feebleness of the heart's action. Nothnagel believes that the vertebral arteries are more liable to be affected by thrombosis than the carotids; and this accords with our experience at Guy's Hospital, where, against three or four cases of thrombosis of the basilar artery or of the posterior cerebrals, there was only one of a similar affection limited to the middle or anterior cerebrals. In one remarkable instance, both the carotid arteries, the middle cerebrals, the anterior cerebrals, and (it is said) the posterior cerebellar arteries were all obstructed by adherent clots. The patient was a man of thirty-five; and the other cases occurred in persons aged thirty, thirty-five, forty-eight, and forty-nine respectively. It would therefore appear doubtful whether thrombosis usually occurs at a more advanced period of life than embolism.

(4) *Syphilitic disease* of the cerebral arteries is one of the most important effects of the *lues venerea*. The first monograph devoted to it was written by Heubner, of Leipzig, and appeared in 1874. He assigns to a Danish writer, Steinburg, the credit of having first attributed to an affection of the blood-vessels many of the cerebral symptoms which arise as a result of syphilitic infection; and to Wilks that of having asserted that the change in the arterial coats is one of a special kind, different from the ordinary atheroma with which it had been confounded.

Heubner finds that the process begins between the endothelium and the fenestrated membrane. Here a number of cells accumulate, which form a mass that encroaches on the calibre of the vessel. In one instance the fenestrated membrane was in part destroyed, the growth penetrating into the muscular coat. He speaks of this as exceptional; but we have had two cases at Guy's Hospital in which what seemed evidently syphilitic ulceration of the vertebral artery passed right through the walls of the vessel into the substance of the pons Varolii. Heubner points out that with the arterial affection there are often associated gummata in the loose tissue of the pia mater at the base of the brain. The cells in the intima of the artery undergo development; the inner run into spindle-cells arranged transversely, the outer into interlacing stellate cells. The new tissue becomes vascular, and this perhaps saves it from undergoing retrograde changes. At any rate, it has hitherto been constantly found greyish white and semi-translucent, so that Heubner confirms the statement, which was originally made by Dr Allbutt, that it does not show the tendency to caseate which marks syphilitic affections of other parts. This may partly depend upon the fact that syphilitic disease of the cerebral arteries has hitherto been observed only in the large vessels at the base of the brain; one can hardly doubt that it must also occur in the smaller branches, where its effects would be less serious, so that it would be less likely to be seen in an early stage. As the channel of the artery becomes narrowed, the blood often coagulates in its interior, and thus the circulation through it is arrested. Although the process is known as *endarteritis obliterans*, Heubner doubts whether the growth by itself ever leads to complete obliteration independently of thrombosis. Ultimately a process of cicatrisation may take place, the cells developing into connective tissue, and the vessel undergoing conversion into a fibrous cord.

When a single vessel has been affected with syphilitic disease it has much more frequently been the carotid artery, or one of its branches, than the basilar, the proportion given by Heubner being as twelve to one. In

one of his cases the circulation at the base was interrupted at four distinct points; the left vertebral artery was obliterated, and so were also the basilar, the left middle cerebral, and the origin of the right anterior cerebral arteries. In most instances several arteries become diseased simultaneously, but syphilitic endarteritis is not diffused so widely as atheroma, and it tends to narrow and occlude the affected arteries rather than to dilate and render them tortuous. Moreover, it affects smaller arteries.

It is generally at an advanced stage of syphilis that the cerebral arteries become affected. Among the cases collected by Heubner the oldest patient was fifty-one years old, the youngest twenty-two; and the numbers were pretty evenly distributed over the three decennial periods between twenty and fifty years of age.

All Dr Gowers' fifty cases occurred between the ages of twenty-one and forty-five, and there were three in men to one in women. The Sylvian branch of the internal carotid artery was affected in nineteen out of twenty cases.

Syphilitic disease of the arteries of the brain, like every other remote effect of syphilis, occurs in persons who are ignorant of having the disease, and who are free from eruptions, nodes, and its other obvious signs. At Guy's Hospital, since the first case recorded by Wilks in 1863, we have had numerous well-marked instances of the affection, besides others of a more doubtful character; but except in a few with a history of syphilis, the proof of their origin depended upon the presence of gummata in the liver or testes, or of lardaceous disease.

Effects of obstruction of cerebral arteries.—The histological state of the brain, when there has been arrest of the circulation in one or more of the arteries, nearly corresponds with what might have been expected from the effects of interference with the arterial blood supply to other tissues. In general it may be said that the affected parts are *softened;* indeed, the various affections now under discussion include the majority of those cases which by the pathologists of the last generation were classified as "softening of the brain."

The anatomical relations of the vessel which happens to be obstructed of course determine the regional distribution and extent of the morbid change; and upon this, in turn, depends the nature of the symptoms. With regard to the Sylvian artery, we shall find that the whole clinical aspect of a case differs according as this vessel is plugged at its origin, or a little further on in its course.

Thus it is of great importance to the physician to be acquainted with certain investigations as to the exact mode of distribution of the cerebral arteries, which have been worked out by two independent observers, Duret in France, and Heubner in Germany. As they point out, the small arteries that alone enter the tissue of the brain form two separate systems, which may be distinguished as that of the cortex and that of the central ganglia (the corpus striatum and thalamus) respectively. These systems are altogether independent, and no anastomoses take place between them; the zone at which they meet within the cerebral substance is situated about an inch and a half beneath the convolutions. The "central" arteries arise directly from the trunks forming the circle of Willis; they are entirely unprovided with anastomoses among themselves. The "cortical" arteries spring from a network in the pia mater, in which tolerably free communications exist between the tertiary branches of the same trunk, and even (in some individuals) between the branches of different trunks.

Let us now apply these facts to explain the effects of obstruction of the

Sylvian artery at different points. That artery, close to its origin, gives off a number of small "central" twigs, which supply the whole corpus striatum (except the inner end of the caudate nucleus), and also the anterior part of the thalamus. It then divides into four terminal branches, of which one is distributed to the third frontal convolution, while the other three pass to the second frontal, the two "central" ascending frontal and ascending parietal, the three convolutions of the parietal lobe, and the three temporal convolutions.

When the middle cerebral artery is obliterated beyond the point at which its "central" offshoots arise, the superficial parts of the brain are the only ones to suffer. And since the subdivisions of the vessel in the pia mater anastomose with those of the anterior and posterior arteries, it is not certain that in the supposed case any softening will result. There may be merely a temporary interference with the circulation in the area to which the Sylvian branches are distributed; or a limited part in the midst of this area may be permanently deprived of its blood supply; or, lastly, almost the whole of the convolutions enumerated above may undergo destruction. Thus Charcot has recorded an instance in which an enormous superficial patch of softening involved the ascending frontal and the ascending parietal convolutions, as well as those of the insula, the corpus striatum and thalamus remaining intact. The differences in the result in different individuals depend partly upon variations in the extent to which the vessels communicate with one another, partly upon whether the plugging takes place suddenly or gradually. It is, of course, quite possible for a single one of the four terminal branches, such as that to the third frontal convolution, to undergo obstruction apart from the rest.

On the other hand, when the seat of the lesion is at the spot where the Sylvian artery arises from the internal carotid, the basal ganglia suffer; and in their case softening is almost inevitable, because their arteries have no anastomoses: it is only when the closure of the vessel takes place very slowly, as the result of chronic disease of its coats, that collateral channels sometimes seem to develop themselves, so that the blood supply is maintained. When the main channel of the Sylvian artery has been obliterated, extensive morbid changes in the corpus striatum and thalamus are likely to occur, while the convolutions may entirely escape.

Local effects of obstruction.—The appearances presented by the affected parts of the brain vary considerably under different conditions. When the patient dies rapidly the cerebral substance may look perfectly healthy. This was found to be the case in an autopsy on a man who was attacked with hemiplegia twenty-six hours after ligature of the internal carotid artery, and who lived only fifty-seven hours afterwards. Where closure of an artery takes place gradually, the regions of the brain that are deprived of their normal blood supply often fail to suffer as much as might be expected. Heubner remarks that in syphilitic affections of the cerebral arteries the nutrition of the cortex is seldom seriously interfered with, unless two adjacent trunks out of the six that arise from the circle of Willis are completely obstructed. In the case already referred to, in which the circulation at the base of the brain was interrupted at four distinct points, there was no softening at all, although one Sylvian artery was obliterated.

When arrest of the flow of blood through a cerebral artery takes place gradually—as in thrombosis and in syphilitic disease—the morbid changes in the brain, if any, take the form of *softening*. The affected parts are

sometimes diffluent, being replaced by a milky liquid, which occupies an ill-defined cavity, containing some loose shreds of connective tissue, and perhaps roofed in by the pia mater or by the ependyma of the lateral ventricle. Every intermediate degree of consistence may occur up to a point at which one can hardly perceive any difference from the healthy substance around, until one allows a gentle stream of water to play over the diseased surface, when it soon becomes ragged and assumes a worm-eaten appearance.

The colour of softened parts of the brain is very variable; it may be white, or yellow, or brownish. Writers have been accustomed to describe separate varieties of the affection according to colour. But (except "red softening," which will be described hereafter) such distinctions have no real existence, and have led to great confusion. As a matter of fact, the condition now under discussion is that which most English pathologists call "white softening," but by Rindfleisch it is named "yellow softening;" and each of these two epithets is susceptible of an entirely different application; for the German writer uses the term "white softening" for a morbid appearance in the central parts of the brain in cases of hydrocephalus, and in England we have been accustomed to describe as "yellow softening" the œdematous state of the cerebral substance that is so often found in the neighbourhood of tumours.

The differences in colour in different cases of softening of the brain depend on the presence of more or less extravasated blood, or on its entire absence. The blood undergoes disintegration with the tissue-elements among which it lies, and under the microscope it is easily recognised in the form of yellow or red granules, or of hæmatoidin crystals. The microscope also reveals in softened brain-substance "compound granule-masses," or "corpuscles of Gluge," often in large numbers. These mulberry-like aggregations of minute granules of oil have already been mentioned as occurring in softening of the spinal cord, and indeed they are found in many degenerating tissues. They are probably degenerated exudation-cells.*

In cases of *embolism*, softening is, as a rule, the only change presented by the parts of the brain deprived of their blood supply. Sometimes, however, there is reflux hæmorrhage from the veins, as occurs in the lungs and spleen. After ligature of the carotid artery the corresponding cerebral hemisphere is now and then found in a condition of vascular turgescence, precisely like the early stage of an infarctus. Thus in an autopsy made by Dr Wilks, all the vessels on the affected side of the brain—including the veins as far as the longitudinal sinus—were distended and filled with coagula, and the substance of the organ, which was pulpy, was of a dark red colour. So, again, in those cases of embolism that have occurred at Guy's Hospital, a condition of "red softening" is described as having been present, and in two other cases tough yellow masses were found which exactly resembled the wedge-shaped patches that are so common in the spleen and the kidney.

"Red softening" is a change of doubtful significance. It may be softening as above described in which a multitude of minute extravasations of blood have taken place; or it may be an early stage of "white" or "yellow softening"—the reason for its not being seen in cases of thrombosis

* Huguénin admits no less than seven origins of these corpuscles—the nuclei of the neuroglia, the cells which make up the walls of the capillaries, those of the "adventitia" of the arteries, the nuclei of the smooth muscular fibres of the vessels, those of the perivascular lymph-spaces, the spindle-cells of the cortex, and very probably its ganglion-cells.

or syphilitic endarteritis being that in these cases death does not take place soon enough; or it may be a true local *inflammation*, to be described in a future chapter.

Again, the coats of the artery itself often become softened, and yield immediately below the seat of obstruction, so that an *aneurysm* is formed. Repeated examples of this have been observed at Guy's Hospital within the last few years; in one of them Dr Goodhart found an aneurysm on the Sylvian artery on each side. It was not indeed proved in every instance that plugging had occurred, but all the patients had disease of the cardiac valves, and in other cases the anatomical evidence of embolism sometimes fails us. Death was in most cases due to rupture of the aneurysm, blood being extravasated in considerable quantity into the pia mater, the corpus striatum, or the lateral ventricle. But, in addition, cerebral hæmorrhage has been the cause of death of seven other persons who were affected with ulcerative endocarditis or had vegetations on their mitral or aortic valves; in all probability an aneurysm, itself the result of embolism, was present in these cases likewise, although it escaped notice at the autopsy.

Sir William Gull long ago taught that it was chiefly in young subjects that intra-cranial aneurysm should be suspected as a cause of cerebral hæmorrhage; and Dr Church, in tabulating a series of cases of this kind, confined his attention to individuals under twenty years of age. But of nine cases which occurred at Guy's Hospital, while seven were between fourteen and twenty-six, one patient was thirty-four and another fifty years old. We have already seen that embolism is by no means so strictly limited to an early period of life as has been generally supposed.

Cerebral hæmorrhage.—Although effusion of blood into the brain may be the result of embolism, of purpura, or of mechanical injury, yet in the great majority of cases it is due to primary disease of the cerebral arteries and their consequent rupture.

Since the days of Morgagni it has been known that hæmorrhage is far more apt to occur in or near one of the corpora striata than in any other part of the brain. But certain anatomical facts have been made out by Gendrin and other observers which have not only led to a more exact determination of its seat, but have also gone far towards explaining why this spot should be so generally the seat of rupture of an artery. Outside each lenticular nucleus there is a mass of white substance called the "external capsule." In recent brains the two structures seem to be continuous; but after a brain has been hardened in spirit it is found that they can be separated from one another with great ease, and apparently, although not really, without any nervous fibres being torn through. Thus the connection between them must be somewhat imperfect. Now, the central branches of the Sylvian artery, which supply the corpus striatum, run upwards for some distance outside the lenticular nucleus before penetrating into its interior. These are more liable to laceration than those of the hemispheres generally, because they are less supported. Moreover, they are much larger than the nutrient arteries of the cortex, they have no anastomosis with one another, and they arise directly from a large trunk, which may almost be said to come in a straight line from the heart. Indeed, as Watson long ago noticed, when injections are forced into the cerebral arteries of the dead body it is in the corpora striata that the vessels are most apt to give way, and the material injected to be extravasated. Charcot calls one particularly large lenticulo-

striate branch "the artery of cerebral hæmorrhage." It is true that the rupture of any artery in the brain is always preceded by the occurrence of morbid changes in its walls; but while this diseased state is common to most of the cerebral vessels, the special liability to rupture of those in the external capsule may be explained by the considerations just offered.

The blood begins by making for itself a space between the lenticulus and the external capsule. At first it forms a thin layer, but as it increases in quantity it gradually becomes a rounded mass. It now flattens out the claustrum and the island of Reil, which lie below and to its outer side; it pushes inwards the corpus striatum, and also the thalamus if it extends far enough backwards. When it accumulates slowly it may press upon the parts around, so as to form a smooth-walled cavity for its reception. But more frequently it tears up irregularly the white matter of the hemisphere and the outer part of the lenticulus. In some rare cases it reaches the superficial convolutions; far more often it ruptures into the lateral ventricle. Coagulation quickly occurs, and at an autopsy we may find a mass of clot weighing four ounces or more. Within the lateral ventricles there is sometimes a red liquid, consisting of blood mixed with the cerebro-spinal fluid; sometimes a clot occupies one ventricle, while the contents of the other are only bloodstained, with perhaps a little clot in the cornua. Dr Broadbent has noticed that the middle ventricular cornu, close to the seat of the hæmorrhage, is commonly empty, having been compressed by the extravasated blood before rupture into the cavity of the ventricle took place. Not infrequently the third ventricle, the aqueduct, and the fourth ventricle are all filled with moulded coagula exactly fitting their cavities; the blood may even escape along the subarachnoid space so as to reach the exterior of the bulb and of the pons, and the parts at the base as far as the opposite Sylvian fissure. The fissure on the side of the hæmorrhage is too closely compressed to receive any of the blood. Indeed, the convolutions of the vertex of that hemisphere are often greatly flattened, so that as soon as the skull-cap is taken off one sees at once which side is affected.

Seat of hæmorrhage.—Of 95 consecutive fatal cases of cerebral hæmorrhage that have occurred at Guy's Hospital—

In 70 the seat of the lesion was in or near the *basal ganglia*. In only 4 of these cases is it stated that the blood was confined to the substance of the hemisphere, not having found its way either to the surface or into the ventricles. In 57 it is expressly recorded that laceration into the lateral ventricle had taken place; and in 22 of these the fourth ventricle also contained either a clot or bloodstained fluid. In 6 cases the superficial convolutions were reached. In 6 instances the part into which the blood was effused was behind the thalamus, somewhat beyond the limits of the region which is the usual seat of cerebral hæmorrhage. Twice the caudate nucleus was alone affected. The right side of the brain was attacked in 36 of the 70 cases, the left side in 34.

In 12 of the remaining 25 cases the seat of the hæmorrhage was in the *pons Varolii;* in 1 it was in the right half of the *cerebellum*. Among the cases of "apoplexy of the pons" there were 5 in which the blood had escaped into the fourth ventricle; and 2 in which it had oozed out through the convex surface of the pons, so as to reach the base of the brain. In no less than 8 of the cases in which there was hæmorrhage in the neighbourhood of the corpus striatum, blood was also effused into the pons; in one instance three independent hæmorrhages were found in this part.

Lastly, there were 12 cases in which the extravasation was into the *membranes* on the surface of the brain. "Meningeal apoplexy" is often described as a distinct affection; and this is justified by the fact that it is sometimes connected with blood poisoning, purpura, anæmia, or pneumonia, and is sometimes caused by blows or falls on the head. But in the twelve cases here cited it was associated with exactly the same changes in the heart and kidneys which we shall see to be commonly found with ordinary cerebral hæmorrhage, and there is every reason to believe that the difference in seat was accidental. The quantity of blood was often large; the nerves at the base of the brain were buried in a thick clot, and the extravasation extended along the Sylvian fissures and in the meshes of the pia mater over the surface of the hemispheres, and also in some instances along the subarachnoid space to the spine, and into the fourth ventricle.

Among 912 cases of non-traumatic intra-cranial hæmorrhage, collected from various sources, the writer found that nearly 65 per cent. were in the *cerebrum*, and more than five sixths of these in the corpus striatum, thalamus, or adjacent parts; that more than 20 per cent. were *meningeal*, nearly 10 per cent. in the *pons*, and about 5 per cent in the *cerebellum*—a proportion not widely different from that observed in the above ninety-five cases collected for the first edition of this book.

Histological effects of hæmorrhage.—When life has been prolonged for a few days after the occurrence of cerebral hæmorrhage, the brain tissue round the clot commonly exhibits reactive changes. At first it is reddened, or visibly ecchymosed; then it becomes œdematous and of a yellow colour, exactly as occurs around a tumour of the brain. At a still later period it may be found in a state of softening. Whether suppuration ever takes place round a clot, as was previously supposed, is very doubtful. Sir Thomas Watson relates a case for which he adopted this explanation; but it is not unlikely that the clot, which was found lying in an abscess, was really of later date than the pus.

But often cerebral hæmorrhage does not destroy life, so that there are other morbid appearances, besides those that are seen in cases which prove directly fatal. In the records of *post-mortem* examinations at Guy's Hospital the author found twenty-one cases in which the remains of effusions of blood of old date were discovered in one of the central ganglia or in their immediate neighbourhood. And ten of the seventy-one cases of recent cerebral hæmorrhage presented additional patches of earlier origin. Sometimes all that was left was a soft tawny discoloured spot, containing abundant granule masses and crystals of hæmatoidin; or a tough, flat, fibrous mass, an "apoplectic cicatrix." Sometimes there was a well-defined membranous cyst, with shreds of connective tissue and a clear or turbid fluid in its interior. In one instance the cavity was of the size of a walnut; in another it was as large as a bantam's egg. Such appearances have been plainly recognised thirteen or fourteen years after the attack, when the patient has died of some different disease. None of our cases enabled us to say within how short a time it is possible for a clot in the brain to undergo absorption and for a cyst to take its place. It may be worth while to notice, that in no single instance in which the remains of an apoplectic clot have been found in the brain after recovery, has there been any indication that the blood had made its way into the lateral ventricle. But according to the statements of Rokitansky and Charcot it would seem that in some very rare cases even effusion into the ventricle is not inevitably fatal.

Ætiology.—With regard to the causes of cerebral hæmorrhage considerable differences of opinion still prevail. In this country the most obvious pathological change found in the bodies of those who have died of effusion of blood into the brain is undoubtedly *chronic renal disease.* Among 116 cases* which came under observation consecutively in the dead-house of Guy's Hospital some morbid condition of the kidneys is stated to have been present in eighty-six; while in only fifteen out of the whole number is it reported that these organs were healthy. But, strange to say, Continental pathologists seem not to have found the kidneys diseased in a similar proportion of cases. Charcot and Bouchard say that interstitial or parenchymatous nephritis was present in only three among forty-nine cases of theirs in which it was looked for, there being, however, thirteen other cases in which the kidneys were "simply atrophied." Nothnagel, in 'Ziemssen's Cyclopædia,' appears to be equally ignorant of the close connection between renal affections and cerebral hæmorrhage, for he quotes, almost without comment, the statement of Frerichs that among 241 cases of Bright's disease there were only six in which effusion of blood into the brain occurred. This, indeed, is not so incompatible with the English observations as might at first sight appear. For none of our cases have been examples of the tubular (epithelial, parenchymatous, or catarrhal) form of Bright's disease; and comparatively few of them seem to have presented those conditions of mixed epithelial and interstitial changes which are so commonly the cause of dropsy and other symptoms in persons at the middle period of life. In the great majority the renal affection has been of the "granular" kind, the organs having shrivelled up into mere relics of their former structure, but retaining their red colour unaltered. In a few instances one kidney had undergone destruction from calculous affection of its pelvis, or the two organs had each become converted into a large tumour made up of a congeries of cysts. It is possible that in such cases the occurrence of cerebral hæmorrhage was merely an accidental coincidence.

All trustworthy observers are agreed that morbid changes in the arterial walls precede their rupture, but when the cerebral arteries are already diseased, there can be little doubt that the hypertrophied left ventricle and high tension of chronic Bright's disease make hæmorrhage more likely, just as we shall see that it not infrequently follows a sudden and temporary increase of blood-pressure.

Sometimes the arteries which arise from the circle of Willis are found to be extremely *atheromatous*, or their walls are extensively calcified.

In the case of a man aged forty, who died in Guy's Hospital in 1869, and who had a hypertrophied heart and granular kidneys, there was a small *aneurysm* on the left middle cerebral artery which had given way and poured blood into the Sylvian fissure, while there was also a large clot outside the corpus striatum, with laceration into the lateral ventricle, apparently of independent origin.

In 1866 Bouchard discovered, in a case of cerebral hæmorrhage from the wards of M. Charcot, two small aneurysms upon one of the vessels; and since that time these two French observers have published seventy-seven cases of the same kind, in every one of which they demonstrated the presence of what they term *miliary aneurysms.* These are described as minute

* All those instances were excluded in which there was reason to believe that embolism was the starting-point of the lesion, and also a few cases of meningeal hæmorrhage, which accompanied purpura, or were due to an injury.

globular or fusiform swellings, from one fifth of a millimètre to one millimètre ($\frac{1}{125}$ to $\frac{1}{25}$ of an inch) in diameter, so as to be plainly visible to the naked eye. They are sometimes very soft, sometimes firm and elastic, sometimes hard like grains of sand. In colour they are purple, or reddish brown, or greyish, according to the thickness of their walls, and the state of their contents, which may be either fluid blood or more or less decolourised clot. They are generally multiple, but sometimes only two or three can be discovered in the whole of the brain after careful search; sometimes as many as a hundred have been counted. They are most conspicuous upon the surface of the convolutions and in the sulci between them; their favourite seat is the deep layer of the grey matter or the line of junction between it and the white. But they may also be found in the thalami and corpora striata, in the pons, and elsewhere. MM. Charcot and Bouchard admit that, when an extensive effusion of blood into the brain has occurred, it is difficult to find the miliary aneurysm the rupture of which they suppose to have led to the hæmorrhage. The best way is to place the affected hemisphere in a basin, and to change the water frequently by tilting, without skaking it. After a time the clots become loosened and float away, leaving a number of little bloody masses connected by vascular filaments with the brain-tissue beneath. Most of these, when magnified, show only the open ends of vessels, torn across secondarily by the extravasated blood, and covered with little caps of fibrin that require to be carefully distinguished from actual aneurysms; but at length some are found which contain spherical ampullæ, continuous with small arteries and split on one side.

It is evident that the discovery of MM. Charcot and Bouchard affords only a partial explanation of the pathology of cerebral hæmorrhage, since miliary aneurysms are not found with especial frequency in that region of the brain which we have seen to be so generally the seat of effusions of blood. Indeed, the French observers themselves point out that the lesion which they describe is, after all, but one of the effects of a diffused morbid change in the cerebral arteries—a chronic "sclerous periarteritis,"—consisting in thickening of the arterial sheath and of the adventitia, with overgrowth of nuclei, while the muscular coat undergoes atrophy. At first they supposed that this condition was peculiar to the vessels of the brain; but another observer at the Salpêtrière, M. Liouville, has since demonstrated the occurrence of a similar affection in the arteries of other parts, and even speaks of having found miliary aneurysms beneath the mucous membrane of the œsophagus and upon the surface of the heart. We are brought by these investigations on sclerous periarteritis to almost precisely the same point to which we are led by the observations of Sir William Gull and Dr Sutton on "arterio-capillary fibrosis." Indeed, it seems clear that the French and the English pathologists have been studying the same morbid process, and their general agreement confirms the accuracy of their conclusions. It is strange, however, that MM. Charcot and Bouchard failed to notice the affection of the kidneys.

There can be little doubt that this diffused change in the arteries of the brain is often the cause of impairment of the nutrition of the brain, attended with such obvious softening of its substance, as must increase the risk of arterial rupture. Charcot and Bouchard quote Rochoux as having first taught the doctrine of *ramollissement hémorrhagipare*, which was further developed by Todd; but they maintain that the only "softening" that bears any rela-

tion to cerebral hæmorrhage is the secondary affection which we have seen to be of frequent occurrence in the tissue round a clot. In the records of *post-mortem* examinations at Guy's Hospital, however, there were no fewer than twenty cases in which localised patches of white softening were found in different parts of the brain—sometimes in the corpora striata or thalami, sometimes in the pons, sometimes in the hemispheres. Many of the patients had suffered from gout; a large majority had granular kidneys and hypertrophied heart; and there is not a single instance in which the kidneys are said to have been healthy. In the ages at which they prove fatal, and in the circumstance that the large majority of the patients were males, these cases corresponded exactly with those of cerebral hæmorrhage; and probably it was a mere accident that death occurred before any of the arteries in the softened patches gave way.

Sex and age.—Cerebral hæmorrhage does not occur with equal frequency in the two sexes or at different periods of life. Men are much more liable to it than women. Of 113 consecutive fatal cases at Guy's Hospital, 82 occurred in men, and only 31 in women.

Among 110 of these cases, in the reports of which the ages of the patients are stated, there were 6 between twenty-one and thirty, 18 between thirty-one and forty, 37 between forty-one and fifty, 29 between fifty-one and sixty, 17 between sixty-one and seventy, and 3 above the age of seventy. These figures do not altogether correspond with the statements of writers in general, who make the frequency of apoplexy increase up to the age of seventy or even eighty years, not only relatively to the numbers of persons living at different ages, but also absolutely.* It may be that men at a middle period of life are more likely than those who are older to be brought to the hospital when attacked with apoplexy, because their relatives are not so apt to suppose that the case is hopeless.

Whether cerebral hæmorrhage is particularly common in men with short thick necks and florid faces is doubtful; but there can be no question that gout is indirectly a cause, by setting up granular disease of the kidneys.†

Cerebral hæmorrhage sometimes occurs first while the patient is making some violent effort, or subjecting his vascular system to an excess of pressure, so that one can hardly help regarding this as the exciting cause of the attack. Thus two of our patients had been running to catch a train; another was a woman who had recently suffered from sea-sickness in crossing from France. All writers mention sudden emotions, violent efforts, cold baths, straining at stool, prolonged laughing, coughing, or sneezing, indulging in stimulants, as capable of bringing on the rupture of an artery in the brain, provided that the vessel is in the diseased condition which seems to be a necessary antecedent of hæmorrhage. Among labouring men many are attacked while at work; but one must not forget that this takes up a great part of their daily lives, and cerebral hæmorrhage occurs in a large number of persons during sleep, when the pressure in the cerebral vessels is supposed to be particularly low. Intoxication seems to be a frequent immediate occasion of cerebral hæmorrhage. Sir Thomas Watson refers to two cases in which men were attacked by hemiplegia while in the act of sexual intercourse;

* Gintrac's 658 cases (excluding meningeal apoplexy), as quoted by Bastian, give for the corresponding ages: under 20, 6 per cent.; 21—30, 7 per cent.; 31—40, 11 per cent.; 41—50, 15 p. c.; 51—60, 20 p. c.; 61—70, 23 p. c.; and above 70, 19 p. c.

† Quæ (animæ) subito revelluntur e crasso corpore multum corporeæ molis secum ferunt: tales autem mittit apoplexia.—*Erasmi Colloquia: Charon.*

and a most severe case of apoplexy followed by hemiplegia occurred under these circumstances in a man fifty years of age, a patient of the writer, who remained hemiplegic for more than two years, and then died of a second attack.

The symptoms of obstruction and of rupture of the cerebral arteries differ in some points; but the three most important—*Hemiplegia*, *Aphasia*, and *Apoplexy*—are common to them all, and will now be separately considered. They are each of great pathological and clinical interest.

HEMIPLEGIA.—The division of the encephalon into two separate hemispheres, each with its own system of blood-vessels, makes unilateral lesions frequent, in striking contrast to the extreme rarity of hemiparaplegia.

Motor symptoms.—The limbs which are affected are always those on the side opposite to the lesion in the brain. This depends upon the fact that the motor columns cross over in the anterior pyramids; those which emerge from under the right half of the pons Varolii passing down into the left half of the spinal cord, and *vice versâ*. Thus any unilateral affection of the nervous centres, situated above the decussation of the pyramids, if it causes paralysis at all, affects the opposite side.*

It is remarkable that this fact was well known to the Greek physicians, who correctly explained it as due to the nerves crossing like the letter X, but was forgotten or doubted as late as the present century. Compare the quotations which are printed at the head of this chapter from Aretæus in the first century, and from Dr James Gregory, professor of medicine at Edinburgh, in the nineteenth.

Although hemiplegia means a stroke paralysing half the body, the extent to which it affects the muscles varies greatly. As a rule, in what may be called "common cerebral hemiplegia"—the *hémiplégie cérébrale vulgaire* of Charcot—the paralysis extends to the opposite arm and leg, the lower part of the face, and the tongue. In the most severe forms the muscles of the trunk are obviously weakened, in slighter attacks the tongue and face almost entirely escape, and in some the leg also is so little affected that the case might be mistaken for one of brachial monoplegia. Moreover, the hand is always more affected than the arm, and the arm than the shoulder, the foot than the hand, and the leg than the thigh. In the facial muscles, commonly all that is noticeable is a little loss of expression

* There are some cases on record which seem to be exceptions to this rule, and these were once marshalled forth by Dr Brown-Séquard, who believes that they establish the possibility of the occurrence of hemiplegia on the same side as the lesion on which it depends. But, whatever the absolute number of cases of this kind that can be collected from medical journals and other sources, it is altogether insignificant in comparison with that of cases on which the rule of decussation is based.

In some of the supposed exceptions it is most likely that the reporters have written "right" for "left," or "left" for "right." Everyone who has done much case-taking will admit how easy it is to fall into this error. Our records at Guy's Hospital contain at least two cases in which it is stated that the disease was on the same side as the paralysis; and one of these is in the hand-writing of Dr Wilks, who certainly must have been struck by such a fact, if he had observed it; but he leaves it entirely without comment, and there can be no doubt that it was a slip of the pen.

In other cases it is probable that there really were two lesions: one, the more conspicuous, on the same side as the hemiplegia; the other on the opposite side—perhaps a minute spot of softening—which really caused the paralysis, but was overlooked.

Another explanation, viz. that in some persons there is no crossing of the anterior pyramids, is unsatisfactory: first, because there is no evidence that the crossing is absent in the cases in question; and secondly, because in man and animals the crossing is of variable completeness, and when deficient is probably made up for by crossing lower down.

about the mouth, which may appear to be drawn over to the opposite side.* This was well shown in the case of Henry W—, admitted into Guy's Hospital on February 20th, 1860, under the care of Dr Gull, for right hemiplegia, which was almost complete. The state of his face was very interesting. When told to shut his eyes he could close them both. But whereas he could wink with the left eye alone, he could not do it with the right alone; and when he was asked to compress the eyelids firmly the left orbicularis palpebrarum alone obeyed the will, the right one merely bringing the upper and lower lids into contact, without any wrinkling of the skin. This, and the further observations which the author made on the same patient, led him to the conclusion that "those movements which are performed in harmony by the two sides of the face or body remain unimpaired in hemiplegia."

In 1866, in an article in the 'Medical and Chirurgical Review,' Dr Broadbent first laid down this law, and it has received general acceptance.†

The exemption of movements which are habitually bilateral (but not always necessarily performed by corresponding muscles of the two sides) is seen in the case of the muscles of the eyeballs, of the masticating muscles, and of those of the neck, chest, and abdomen, all of which retain their power.

Not infrequently the face escapes entirely. Sometimes the leg can be moved, while the arm is completely paralysed. Almost invariably, when recovery takes place, the patient regains power in the leg earlier than in the arm; he may perhaps be able to walk with a crutch at a time when he can scarcely move the fingers at all, and if any part remains permanently paralysed it is the upper limb. These facts may be explained by the movements of the arm being more independent than those of the leg, and furnish a further illustration of Broadbent's rule that the limb of which the movements are more highly specialised suffers most and longest.

Again, the highly differentiated movements of fingers are more affected than the coarser movements of the shoulder. The *tongue*, however, is often only partially paralysed; when it is protruded from the mouth its tip is more or less distinctly turned towards the side on which the limbs are paralysed, and the patient often articulates indistinctly and mumbles in his speech; so much that he may in some cases be unintelligible.

It was formerly considered difficult to understand why the hypoglossal nerve and the portio dura—arising as they do above the decussation of the anterior pyramids—should nevertheless resemble the nerves of the limbs in showing impairment of function on the side opposite the lesion in the brain. Physiologists are now agreed that this depends upon the fact that the fibres which pass to the roots of those nerves undergo an independent decussation within the substance of the medulla oblongata or pons.

Dr Broadbent's rule must not be taken to mean that there is never any loss of power whatever in muscles which are used in association with those of the opposite side. It has been proved by several independent observers that the chest sometimes does not expand fully on the paralysed side, at least when a deep breath is being taken. Dr Walshe ('Lancet,' 1849) determined this by actual measurement. Dr Broadbent himself points out that when a hemiplegic patient is made to raise himself from the recumbent

* Todd ascribed this to impaired power of the buccinator muscle (which he believed to be supplied by the fifth nerve), and maintained that the facial nerve escaped altogether.

† In 1860 or 1861 I read before the Physical Society of Guy's Hospital a paper in which I developed the same theory; of course, as I did not print it, the priority rests with Dr Broadbent.—C. H. F.

posture by his abdominal muscles alone, the rectus on the paralysed side can be felt to act somewhat less powerfully, and a little later than on the healthy side. So, also, if he is told to close the jaws firmly, the masseter and temporal muscles do not contract quite simultaneously or with equal force on the two sides.

It is remarkable that when there is profound paralysis of the face or arm, movements may still be called forth by emotional or involuntary stimuli, which the patient is quite unable to execute by his own efforts. Thus a hemiplegic may raise his shoulders in yawning, or contract his zygomatic muscles in laughter, when he cannot perform the same movements in the least by a voluntary exertion. We may associate these facts with the curious observation that a patient who is speechless from apoplexy sometimes makes a short exclamation when it is forced from him by some sudden emotion, and with the escape of the muscles of respiration and of organic life in hemiplegia. But it is hard to say where we are right in seeking an anatomical explanation, and where we ought to be content to regard them as examples of a physiological or evolutionary differentiation of function.

As Dr Jackson many years ago pointed out, patients suffering with aphasia, from disease situated above the corpus striatum on one side, are sometimes quite unable to protrude the tongue from between the teeth. We have several times observed this at Guy's Hospital, and at the present time there is an instance of it in Philip Ward (Jan., 1890). It seems clearly to be an instance of double paralysis from a unilateral lesion; but we may perhaps attribute it to an inhibitory influence transmitted downwards upon the associated nuclei of the ninth nerves in the pons, rather than believe that both sides are "represented" in each cerebral hemisphere. This is the hypothesis suggested by Dr Jackson to account for the supposed occurrence of paralysis of all the limbs as the result of an affection of one hemisphere ('Brit. Med. Journ.,' 1874).

In some cases of hemiplegia the eyes, instead of being directed straight forward, are turned to one side, and this side is always opposite to that of the paralysis, so that, for the purpose of artificial memory, one may say that the patient "is looking towards the lesion in his brain." This *conjugate deviation* of the eyes is by some writers regarded as due to a paresis of certain ocular muscles—the external rectus on the side of the paralysed arm and leg, the internal rectus on the other side. Others have supposed it to depend upon spasm of the antagonist muscles, and thus to be analogous to the state of "early rigidity" which will presently be described. But the face also is sometimes drawn over, away from the hemiplegic limbs, and in such cases Prévost found that the sterno-mastoid and the trapezius muscles are not necessarily in a state of contraction. Moreover, Dr Jackson has pointed out that when "early rigidity" comes on in the arm and the leg the "conjugate deviation" becomes reversed, the eyes being now directed towards the side of the paralysis; a case in point occurred at Guy's Hospital in 1867 in the practice of Dr Rees. Thus it seems clear that this remarkable symptom is not of a spasmodic nature, and Vulpian and Prévost are probably right in supposing that it represents, in a rudimentary form, the rotatory movement (*mouvement de manège*) which is so commonly developed in animals after unilateral injuries to the head. Indeed, a woman under the care of Charcot for left hemiplegia with conjugate deviation of the eyes was several times found lying across her bed with her head to the right and her feet to the left; and another of the patients whose cases are

related by Prévost showed a tendency to turn round like a top when placed upright. This writer has proved that the occurrence of conjugate deviation of the eyes is not limited to affections of any one region of the brain: among fifty-eight cases which he collected there were four of meningeal hæmorrhage, and four others in which the superficial convolutions were alone diseased. It is generally transitory, lasting only a few hours or a day or two; but sometimes it persists for several months.

We have seen that in common cerebral hemiplegia there is no complete paralysis of any of the cranial nerves, but in a less common group of cases palsy of the third or of the seventh nerve is associated with hemiplegia, and then the seat of the disease can be determined with more than usual exactness.

The *facial nerve* may be affected in two different ways. On the one hand, the lesion, if it is situated in the upper part of one lateral half of the pons, may destroy the facial nucleus, and so cause a total loss of power in the face on the same side on which the arm and the leg are paralysed; and since the nucleus of the sixth nerve is very close, paralysis of the external rectus may perhaps be present in addition. On the other hand, an affection of the lower part of the pons on one side may cut the fibres of the facial nerve as they are passing across the motor tract, after they have decussated on their way outwards from their nucleus. The paralysis of the face is then on the opposite side to that of the limbs. It is possible that an extensive unilateral lesion of the pons might cause paralysis of both sides of the face by destroying the nucleus of one facial nerve and the fibres of the other.

Again, association with hemiplegia of paralysis of the ocular muscles supplied by the *third nerve* shows that the seat of disease is the crus cerebri. The muscles in question are those affected on the side opposite to the paralysed arm and leg. An instance of this has been placed on record by Dr Hermann Weber ('Med.-Chir. Trans.,' vol. xxviii). A man, aged fifty-two, was attacked with paralysis of the right side of the body and of the muscles supplied by the left third nerve; he died after two months, and an oblong clot of blood was found in the left crus. Following Gubler, recent French writers have given a special name, that of "*Hémiplégie alterne*," to cases in which either the facial or the third nerve is affected on the side opposite to the limbs. "Crossed hemiplegia" is the English equivalent.

Sensation.—Statements that *anæsthesia* is absent in hemiplegia appear to have been based upon observations made on cases of long standing. There is every reason to believe that anæsthesia is generally present in recent cases, although its recognition may be difficult, or even impossible, in patients whose consciousness is much obscured. It is, however, far more transitory than motor paralysis, often passing off in a few days, and sometimes still earlier. In this respect lesions of the brain resemble those of the spinal cord and of the peripheral nerves (cf. pp. 403, 430). In slight cases of hemiplegia sensation is apparently unaffected, and even in the most profound it is never completely abolished. The hands and feet suffer more than the proximal part of the affected limbs, and the face least. But in exceptional cases, as Sir William Gull stated in his Gulstonian lectures (1849), anæsthesia is more marked than motor paralysis. In one case the writer noticed this in each of a series of hemiplegic attacks, some affecting the right and others the left side.

Subjective sensations of formication and numbness are frequently associated with partial anæsthesia, and sometimes there is a condition of hyperalgesia, or increased susceptibility to painful impressions. Sometimes, too,

spontaneous pains are experienced in the joints on the affected side, and particularly in the shoulder-joint.

Trophic changes.—Hemiplegic limbs are often reddened and warmer than those of the opposite side—a difference which is attributed to interference with the functions of the vaso-motor nerves. The inequality of temperature commonly amounts to something less than one degree Centigrade; after some months it ceases to exist. In some cases there is excessive sweating; sometimes slight œdema is discoverable. In a case at Guy's Hospital, dropsy (of renal origin) was limited to the arm and leg that were paralysed; and the late Dr Laycock, of Edinburgh, published examples of this curious fact. Charcot lays stress on the frequency of rapid sloughing of the skin over the gluteal muscles—an acute bedsore, as he terms it. One can hardly suppose that, in cases of this kind, there is much difference in the amount of pressure upon the two sides of the buttocks; and he regards the affection as a more or less direct effect of an interference with the nutrition of the tissues, caused by irritation of a particular region of the brain. Such bedsores are certainly very rare in cases of hemiplegia seen in this country; and equally so other trophic affections of the skin such as are noted in peripheral paralysis (p. 404).

As a rule contractions in the paralysed limbs by reflex action are less readily excited than on the healthy side; sometimes, but very seldom, more readily. In the latter case, some amount of tonic spasm is always present; the elbow is generally flexed and resists extension, and the fingers are bent in upon the palm of the hand. This condition was described by Todd under the name of "early rigidity," and was attributed by him to laceration of the surrounding healthy brain-substance by effused blood. This explanation has been adopted by most subsequent writers, although it has not been shown that there is more injury to the nervous tissues than in cases in which the muscles remain flaccid. So long as the muscles are in a state of early rigidity they do not waste; and subsequently they only undergo atrophy from disuse, without losing their faradic contractility, or showing any other signs of the reaction of degeneration. Rigidity only occasionally comes on early—more often it supervenes as a "late contraction" of the limbs, among the sequelæ of hemiplegia (p. 568).

Seat of the lesion.—It was once questioned whether paralysis of the arm and leg can be caused by a lesion limited to the *white substance* of the hemisphere or the *convolutions*, to the exclusion of the basal ganglia.

Mr Hutchinson has insisted on the frequent occurrence of hemiplegia when meningitis appears to be limited to one half of the brain, as so often happens after surgical injuries; but as a rule even extensive destruction of the cortex of the cerebrum is followed by no definite signs of paralysis, if it spares what is now known as the "motor area." The experiments of Fritsch and Hitzig, and those of Ferrier and Yeo, Schäfer and Horsley (to which we shall more fully refer in the next chapter), have shown that irritation of convolutions of this area causes movements in the opposite limbs; and also that lesion of these convolutions can produce a permanent paralysis. Cortical lesions, to use Dr Jackson's nomenclature, are not only "discharging," but also "destroying" lesions. Diseases of the ascending frontal and parietal convolutions, and of the adjacent parts of the frontal and of the superior parietal convolutions, may, if sufficiently extensive, give rise to permanent hemiplegia, although the corpus striatum remains perfectly healthy. Softening of this motor region of the brain is an occasional result

of obstruction of the Sylvian artery, beyond the origin of its "central" branches, whether by thrombosis or embolism (p. 553). The paralysis seems to be indistinguishable from that caused by a lesion of the basal ganglia; and it is followed by the consecutive degeneration of the spinal cord which will be described a little further on. On the other hand, similar affections of the sphenoidal, anterior frontal, and occipital regions cause no permanent hemiplegia.

We may therefore conclude that lesions of the motor gyri, and of the fibres which run thence through the corona radiata, may cause hemiplegia, just as lesions may which destroy the external capsule, the crura cerebri, or any other section of the motor tract.

As regards the lesions of the *basal ganglia*, Charcot maintains that, whenever a lesion is limited to one of the grey nuclei of the corpus striatum (the lenticulus or the cauda), the hemiplegia is always transitory, and very often incomplete; whereas if the internal capsule is involved it is commonly complete and persistent; but he impairs the force of this remark by adding that neither the nucleus caudatus nor the lenticulus is ever *wholly* destroyed without other parts being affected at the same time. He maintains that the limitation of paralysis to a single limb is never due to the fact that the morbid process is confined to a special seat within one of the ganglia: such limitation points to a lesion of the cortex. Dr Jackson agrees with this statement, and to explain it, supposes that "the whole corpus striatum is represented in miniature by every single part of it." Ferrier found that irritation of this part of the brain by faradic currents causes a general contraction of the muscles on the opposite side of the body, without its being possible to differentiate individual movements. But it is possible that, at least in the internal capsule, the fibres which belong to the upper limb may be distinguished from those that pass to the lower limb. We shall hereafter see that the cortical centre for the leg-muscles lies nearer the median plane than that for the arm-muscles; and, if there is a distinction of fibres in the internal capsule, those for the leg are probably situated further inwards and forwards than those for the arm. Since in cases of cerebral hæmorrhage the inner and fore part of the capsule often escapes laceration when the outer and hinder part is torn through, may not this be the reason why the upper limb so often remains powerless after the patient has regained the use of his lower limb, and why the arm sometimes becomes paralysed alone, while the leg escapes altogether?

Charcot believes that a broad line of distinction must be drawn between affections limited to the anterior two thirds of the internal capsule and those which involve its posterior third. In the former case there is no loss of sensation, in the latter there is hemianæsthesia, affecting the same side as the paralysis. He even believes, in opposition to some other authorities, that a lesion limited to the posterior third of the internal capsule may cause loss of sensation without any loss of power. The anæsthesia affects not only the limbs of one side, but also the corresponding half of the body and of the head. It includes insensibility to pain and to changes of temperature, as well as to tactile impressions; it extends to the deeper parts, and also to mucous surfaces; the patient feels nothing when his muscles are made to contract by the application of a current of electricity; and it affects the special senses of hearing and smell and taste.

Vision in hemiplegia.—In these and certain other cases hemiplegia impairs the sight; but there is difference of opinion as to the kind of imperfection

which results. According to Dr Hughlings Jackson this is lateral homologous hemiopia,* corresponding halves of each retina being affected in such a way that the patient is unable to see towards the paralysed side. In 1875 he had seen some thirteen cases of hemiplegia attended with the symptom in question. In one instance, in which the paralysis was on the left side, a *post-mortem* examination was made by Dr Gowers, who found a single lesion of the right thalamus, the posterior half of which was softened and of a greyish-yellow tint ('Lancet,' May, 1875). The hemiopia had been of the most marked description. The man had sometimes seen only half of a word, reading "land" for "midland," and remarking to his son that "Liver" was a queer name, when it really was "Oliver." Notwithstanding the statements of Mandelstamm, Michel, and others, it is probable that only the inner parts of the optic nerves decussate at the chiasma; and if so, we have a satisfactory explanation of this "homologous lateral hemiopia."

Charcot, however, denies that this affection of sight forms part of hemianæsthesia. On the contrary, he maintains that there is a "crossed amblyopia"—an impairment of vision in both halves of one eye—the one on the side which is deprived of sensation. He says that the acuteness of vision is often diminished by one half or even in a still higher degree. The visual field is generally narrowed on all sides, but for some colours the range is far more limited than for others. The first to be lost is violet; then, in succession, green, red, orange; the colours for which vision is retained longest are yellow and blue, but even they may at last be indistinguishable, so that every object appears of a brown colour, as if painted in sepia. Charcot quotes a case of Dr Bastian's, in which a lesion limited to one of the anterior corpora quadrigemina caused crossed amblyopia and not lateral hemiopia. If this view is correct, the only possible explanation seems to be that which he gives, namely, that there is a second decussation of the optic tracts for those fibres which failed to cross in the chiasma.

After-effects.—Hemiplegia is often recovered from, the patient gradually regaining the use of his limbs, until at length no difference can be made out between the two sides. As already remarked, the rule is for the leg to recover before the arm; the patient may be able to walk quite well with a crutch at a time when the hand is still motionless. Trousseau, however, relates two instances in each of which power was restored in the arm earlier than in the leg, and he maintains that when this is the case the prognosis is particularly unfavourable.

In many cases hemiplegia is permanent. The patient may for the rest of his life be unable to move any part of his arm, or he may regain power in it to a greater or less extent.

In almost every case the affected muscles pass, sooner or later, into a state of contraction, which is known as *late rigidity*. Bouchard found it absent in only one out of thirty-two cases that he examined. It generally consists in flexion of the joints; the elbow is bent at nearly a right angle, the wrist is pronated and flexed on the forearm, and the fingers are drawn in upon the palm of the hand, so that the nails sometimes penetrate the skin and produce painful ulcerations. Much more rarely a position of extension is assumed; the elbow may then be straight, the wrist may be thrown back, and the fingers may assume the griffin's-claw attitude which was de-

* These terms (half-sight or half-blindness) are now usually confined to lateral division of the field—whether homologous (or homonymous) or "crossed." The terms *hemiopia, hemianopia, hemianopsia*, are used indifferently to express the same condition (cf. p. 608).

scribed as occurring in progressive muscular atrophy (p. 497). The leg is less apt than the arm to become contracted, even when it remains paralysed; if at all, the hip- and the knee-joints are flexed, and the heel drawn up. This form of rigidity sets in so gradually that the exact period at which it begins cannot be determined. Bouchard speaks of a case in which it was present in a marked degree two months after the apoplectic attack which had caused the hemiplegia. Sometimes one can overcome the spasm by a little traction upon the affected parts; sometimes it resists the application of even a painful amount of force. The muscles are generally wasted, and feel like tight cords beneath the skin; but this atrophy is much later, and never so complete as that of anterior poliomyelitis.

Formerly it was supposed that late rigidity was caused either by the slow contraction of an apoplectic cyst, or by an inflammatory process in the surrounding brain-substance. But in 1866 Bouchard suggested, in the 'Archives Générales,' that it was probably an indication of the supervention of certain changes in the lower parts of the cerebro-spinal axis, which had been pointed out by Türck in 1853, and had been previously noticed by Cruveilhier as occurring after unilateral lesions of the brain. These changes are to some extent visible to the naked eye; the crus cerebri is smaller than on the healthy side, and presents a grey streak towards its inner edge; the corresponding half of the pons may be flattened; the anterior pyramid is of a greyish colour and wasted, so that the decussation of the pyramid is more conspicuous than usual. Below this point there is no marked alteration on the surface, but transverse sections of the spinal cord may show, in the lateral column of the side *opposite* to the lesion in the brain, a triangular grey patch, which gradually diminishes in size, but is traceable down to the lumbar enlargement. Or it may be only after hardening and staining that the change is discoverable by the microscope. It is found to consist, not only in a degeneration of the nerve-fibres which pass down the cord from the brain, but also in the formation of a delicate new connective tissue. In other words, there is descending sclerosis of a definite tract of the lateral column. Many nerve-fibres which probably arise from the spinal nuclei escape the degenerative process; and Bouchard's theory is that these unaltered nerve-fibres become irritated and cause the contracted state of the muscles. In support of it he adduces the analogous rigidity of the lower limbs which occurs when the upper part of the spinal cord is compressed.

Charcot believes that, once set up, the sclerosis may spread as an independent affection to the anterior grey cornua, causing the muscles to become again flaccid and to undergo a rapid degenerative atrophy; or to the posterior cornua, inducing partial anæsthesia; or, lastly, to the opposite lateral column, giving rise to a contraction of the other lower limb, as is supposed to have occurred in a case of Dr Bastian's.

More recently, however, Hitzig has proposed a different explanation of "late rigidity." He points out ('Arch. f. Psych.,' 1872) that prolonged rest is followed by a relaxation of the contracted muscles; that, for instance, after a night's sleep, the paralysed limbs are commonly flaccid and supple, and remain so until the patient begins to exert the opposite limbs in getting out of bed, or in some other way. Again, he has observed that voluntary efforts with the unaffected arm often cause an increase in the rigidity of the affected arm. In one of his cases, as soon as the patient was made to lift a heavy weight with his left hand, the thumb and forefinger of the paralysed right hand became quite stiff, although they had before been free from

spasm. He supposes that the contractions of hemiplegic limbs represent an excess of those co-ordinated movements in distant parts which naturally accompany every action of the body. He assumes that the spinal centres are in a state of irritation, and that, as a consequence, the movements in question become far more marked than under normal conditions. In further illustration of his meaning he instances the well-known fact that patients who are altogether unable to lift a hemiplegic arm by any voluntary effort sometimes see it move when they yawn or stretch themselves.

Whatever its explanation, "late rigidity" bears a close relation to certain forms of mobile spasm which may also present themselves in hemiplegic limbs, but more rarely, and only when the loss of voluntary power is incomplete. They vary greatly in character in different cases. Sometimes a more or less violent *tremor* is observed, either while the limb is at rest, or only when it is made to execute some voluntary effort. Charcot, for example, mentions an instance in which the act of carrying a glass to the mouth was attended with rhythmical movements, so that the liquid in it would be thrown in all directions. In other cases *spasms* are seen, which are more or less choreiform in character, so that Dr Weir Mitchell in 1874, and Charcot since, have spoken of a "post-hemiplegic chorea."

Lastly, some cases of chronic hemiplegia are attended with slow involuntary movements, principally affecting the thumb and fingers, and exactly like those described by Dr Hammond in 1871 under the term *athetosis*. The name (ἄθετος, without a fixed position) is intended to signify that the parts concerned in the spasm cannot be kept still. They are constantly moving, without any voluntary effort on the part of the patient. The fingers are alternately flexed and extended, with varying degrees of adduction or abduction, so as to give the hand a very odd appearance; and the toes may present similar changes of position. Cases of this kind have been described by Dr Allbutt and Dr Gairdner; and Dr Gowers has fully discussed "post-hemiplegic disorders of movement" in the 'Med.-Chir. Transactions' for 1876. Those which the writer has seen have been in young patients. Athetosis is often observed without antecedent hemiplegia.

APHASIA.*—Beside hemiplegia another symptom of cerebral obstruction or hæmorrhage is Aphasia, or loss of speech—the defect being of a special kind, and presenting characters which merit careful study.†

It is altogether distinct from a mere impairment of articulation, such as occurs in bulbar paralysis (p. 508), and often forms part of common cerebral hemiplegia (p. 562). These are due to imperfection in the movements of the tongue and palate, and the speech is more or less thick and difficult to be understood. But in aphasia, if the patient can utter a word at all, he commonly pronounces it quite clearly and dis-

* It has been called *alalia* and *aphemia;* but of these names the first is of doubtful authority, and the second would mean in Greek not loss of speech but loss of reputation. ἀφασία μ' ἔχει is a phrase used by Euripides, and in the form ἀμφασίη ἐπέων by Homer. To express want of power to write the term *agraphia* has been coined, and for want of ability to recognise written characters, *alexia*.

† "The inability to speak is sometimes owing not to the paralytic state of the organs of speech only, but to the utter loss of the knowledge of language and letters: which some have quickly regained and others have recovered by slow degrees, getting the use of the smaller words first, and being frequently unable to find the word they want and using another for it of a quite different meaning; as if it were a language which they had once known but by long disuse had almost forgotten. After an apoplectic state for several days, one person was forced to take some pains in order to learn again to write, having lost the ideas of all the letters except the initials of his two names."—*Heberden's Commentaries*.

tinctly. He may be altogether mute; or he may occasionally, when excited, ejaculate some oath, but it is impossible to get him to repeat it deliberately; or he may possess the power of saying two or three words or short sentences; these he uses sometimes correctly, but often in contradiction to his real meaning, and in answer to every question that is put to him. Thus Trousseau relates the case of a man named Paquet, who for four months said nothing but *tou-si-si*, *cousisi*, and who kept uttering these three syllables on all occasions, whether he was in a passion, or wished to express gratitude, or wanted to ask for or refuse something. Only, if he became very excited, he would sometimes say *sacon*, *sacon*—probably an abbreviation of the oath *sacré nom de Dieu*. Dr Broadbent had a patient who could scarcely say anything but *Oh, shameful! shameful!—Oh! pity, pity!* Of two celebrated cases recorded by Broca, one was that of a man who, possessing four words, *oui*, *non*, *trois*, and *toujours*, said *tan*, *tan*, to every question for twenty-one years. If there is a more extensive vocabulary the patient is very apt to use one word for another. Sometimes there is a likeness in sound between the two, as when *purging* takes the place of *perjury*, *pamphlet* of *camphor*, *dispersion* of *dispensary*; sometimes none, as when a patient of Dr William Ogle said *boat* instead of *tub*, or *two-shilling-piece* for *spectacles*.

A person who has known two or more languages may entirely lose the power of speaking in one of them, while he retains it in the others. Trousseau gives an instance of this in a Russian, who before his illness spoke French like a Parisian; and Bastian says that he has seen two similar instances, one being in a German who had long been resident in England.

Dr Ogle ('St George's Hosp. Rep.,' vol. ii) draws special attention to the fact that grammatical form is always observed; substantives are used in the place of substantives, verbs for verbs, numerals for numerals. M. Broca's patient employed *trois* to express any number, but corrected what he said by holding out the proper number of fingers at the same time. Dr Broadbent's patient possessed only one name for a locality, namely, Burlington, where she had lived as a child; and she used this for any place whatever.

Sometimes a patient who can utter only one or two words by himself is able to repeat a good many other words if he is prompted. Thus Trousseau relates the case of a man named Marcon, who could only say *Ma foi* and *Cré nom d'un cœur*, but who, when asked, "Are you from the Haute Loire?" replied *Haute Loire;* and then to the query, "What's your name?" echoed again *Haute Loire.* "Your profession?" *Haute Loire.* "But your name is Marcon?" *Yes, sir.* "What department do you come from?" *Marcon;* and so forth. We shall hereafter see, however, that such a case as this is not *mere* aphasia; nor should the name be applied to such a case as that recorded by Dr Bateman of a woman at the Salpêtrière, who, although she said nothing of her own accord, repeated everything that was said to her, and mimicked every gesture.

In 1889 we had for several months a patient in Mary Ward who had "common cerebral hemiplegia" of the right side with aphasia, and, excepting a word very occasionally ejaculated at random and immediately forgotten again, her only language was the repetition of the syllables *ten-ten*, or sometimes with a rather more open vowel sound, which must have come very near that of Broca's patient. She seemed intelligent, and was cheerful, taking interest in the slow and gradual recovery of her paralysed muscles; and it was curious to see how perfectly by help of her one word, uttered with perpetual variety of accent and intonation, she was able to convey acquiescence

and negation, pleasure and pain, entreaty, remonstrance, and gratitude. Much pains were taken to teach her fresh words; but though she learnt one for a time, she quickly lost it again; and when after several months she left the hospital, able to walk with support, and having some power over her arm, she went out repeating *ten-ten*, with a new meaning of farewell.

Agraphia.—In some, but not in all, cases of aphasia the patient loses the power of expressing himself in other ways as well as in speech. The person who is aphasic is sometimes able to make all his thoughts known with his pen. Trousseau relates the case of a carrier belonging to the Paris Halles, who came into the consulting room making signs that he could not speak, and handed in a note in which the history of his illness was detailed. He had written it himself, and it was perfectly well worded. A few days previously he had suddenly lost his senses, and remained unconscious for nearly an hour. When he came to he had no paralysis, but he could not articulate a single word. In the course of five or six weeks he completely recovered; but what was remarkable was that during the whole course of the disease he could manage all his affairs by substituting writing for speech. Such cases are very exceptional. As we shall presently see, paralysis of the right hand is very often present in those who have aphasia; and of course it is then difficult for the patient to write. One way of ascertaining whether the power of combining letters to form words is preserved is to give such a person the loose wooden letters that children play with, asking him to spell his name or to put together a sentence. He may be quite incapable of doing so; indeed, he may not even be able to pick out the letters that make up the one or two words that he is perhaps able to articulate (*alexia*). Trousseau's patient, for instance, whose one word was *oui*, could not point to the letters *o*, *u*, *i*, in the title-page of a large quarto volume.

The impairment of power to write is sometimes marked, when speech is but little affected. Dr Jackson records the case of a partially aphasic patient who could talk pretty well, although she made mistakes in speaking, and called her children by wrong names; but when told to sign her name she wrote *Sunnil Sielaa Satreni*, in which there was no resemblance to the real name either in sound or spelling; and when told to write her address she put down *Sunesr met ts mer tina—lain.* We shall presently see that if there is much impairment of the intelligence the value of agraphia as a symptom is much diminished; but both Trousseau's patient and Jackson's appeared to have their full mental faculties.

Again, it has been shown that in some cases of aphasia the memory of *other modes of expression* is lost, beside spoken and written language. Trousseau says that a patient may be unable to imitate the face of a person who is crying, although when he feels grief the expression of his countenance shows it clearly enough. In the case of Paquet, who said nothing but *Cousisi*, Trousseau first held out his hands and moved his fingers, as though playing the clarionet, and asked this man to do the same. Paquet immediately executed the same movements with perfect precision; and when asked whether he knew that the attitude was that of a clarionet player he would assent by nodding his head. Yet when told, a few minutes afterwards, to place himself in the same attitude, he seemed to think, and often was unable to do it.

An aphasic patient, whose case was carefully studied by Dr Scoresby Jackson ('Edin. Med. Jour.,' 1867) was totally unable to play the piano.

He put himself in the proper position, and placed his fingers on the keys, but he could not play a single bar, not even of a piece of music with which he had been familiar before his illness; yet he could hum the same tune pretty well. He played draughts with skill; and Paquet also could play backgammon and dominoes perfectly, and would cheat when he found himself losing.

Amnesia.—It is important to notice that the very words which an aphasic patient is unable to utter are perfectly understood by him when spoken by another person. This, indeed, is but an illustration of a broad distinction which is traceable throughout the faculty of speech. A child learns the meaning of words addressed to it by others long before it can itself speak. A person partially acquainted with a foreign tongue recognises many words, if spoken distinctly by anyone else, which he would have been unable to call from his memory if he wanted them in conversation. Nay, we all of us understand the meaning of many English words when we hear them which we never use ourselves. Adopting terms used by Dr Moxon, we may say that there is a great difference between "incoming" and "outgoing" language; or, following Dr Broadbent, that words are to be considered in two distinct aspects: first as "intellectual symbols," and then as "motor processes." In ordinary aphasia and agraphia it is the *outgoing language* and *motor processes* that are interfered with; in amnesia and alexia it is the *incoming language* and *perception* of intellectual symbols.

The same distinction is denoted by Wernicke's terms: *motor* (ordinary) *aphasia*, including agraphia, and *sensory aphasia*, including alexia; and by Charcot's "verbal and graphic amnesia" which are represented in English by "word-deafness" and "word-blindness" respectively.

It may be that perception of the meaning of the words spoken by others, and uttering words for one's self, are functions of different parts of the brain. Or, again, it may be that both "incoming" and "outgoing" language have their seat in the same locality, but that the latter requires for its execution some subordinate nervous centres which have nothing to do with the former. The second of these hypotheses was defended with great ability by Dr Broadbent in the 'Med.-Chir. Transactions' for 1872.

The local lesion.—It has now been established that aphasia—including agraphia—is almost invariably dependent upon a lesion of the *left side* of the brain. This was assumed by Broca as the result of a case which he published in the 'Bulletin de la Société Anatomique' for 1861.*

This association, though previously completely overlooked, is beyond question, and can at any time be determined by clinical observation. If a number of hemiplegic patients be taken, it will always be found that many of those in whom the paralysis is on the right side are speechless, whereas probably every one among them whose left arm and leg are paralysed will be able to articulate as distinctly as is compatible with the condition of the tongue and lips. Moreover, in much rarer cases, aphasia will be found to exist without any paralysis, and the lesion is almost invariably in the left side of the brain. Lastly, to complete the argument, the exceptions themselves illustrate the rule, for in left-handed persons (whose right hemisphere takes the precedence in movements which the left does in the rest

* It was stated nearly thirty years after the event that Dr Marc Dax, of Sommières, read a paper on the subject before a Medical Congress at Montpellier in 1836, but the memoir has never been seen since. Its title was "Lésion de la moitié gauche de l'encéphale coincidant avec l'oubli des signes de la pensée." Certainly no one, either in Paris or in London, was acquainted with the views of M. Dax when Broca published his paper.

of mankind*) loss of speech goes with paralysis of the left limbs, and not with that of the right limbs; or, in other words, it depends upon disease of the right hemisphere instead of the left. Examples of this association have been recorded by Dr Jackson ('Med. Times and Gaz.,' 1866), Dr John Ogle ('Lancet,' 1868), and Dr Wadham ('St George's Hospital Reports,' 1869), and two well-marked cases have occurred in Guy's Hospital.

Further, it was proved by Broca that aphasia is caused by lesions of a particular part of the surface of the brain on the left side. Bouillaud was long ago led by clinical observation to connect the faculty of language with the *anterior lobes* of the brain, and, indeed, Gall had previously suggested the same localisation.†

But it was not until a second case of M. Broca's was published, in November, 1861, that anything like precision of localisation was arrived at in regard to loss of speech. The case in question is that of a man named Lelong, aged 84, who after an attack of apoplexy lost the power of uttering words, with the exception of four, although he knew all that was said to him, and could make himself understood by gestures. He died at the end of a year of a fracture of the femur. At the autopsy the posterior third of the second and of the third left frontal convolutions were found to have been destroyed by softening, their place being occupied by a secondary hæmorrhagic cyst.

Ever since, the *third left frontal* has been known as Broca's convolution, and it is now admitted that aphasia is always dependent upon a lesion involving that region, if we limit it to the posterior part of the third left frontal gyrus, and extend it to the back of the second frontal and adjacent parts of the ascending frontal and *gyri operti*.

In the first place, no disease of the corpus striatum, or even of the white substance outside it, is capable of causing loss of speech. The general accuracy of this assertion is proved by the fact that cerebral hæmorrhage, in the position in which it usually occurs, leaves the speech unimpaired. In 1876 Dr Broadbent was able to say ('Brit. Med. Journ.,' i, p. 436) that he knew of no case on record in which lesion of the corpus striatum alone had given rise to this symptom. At first sight such a fact might seem inexplicable, since it is difficult to see how any effect can be produced by destruction of a convolution which may not equally follow the division of those conducting fibres which connect it with the basal ganglia, or even with the spinal centres. No writer appears to have faced this difficulty except Dr Broadbent himself. He believes that there are two distinct routes by which impulses pass from Broca's convolution to the nerve-nuclei in the pons, medulla oblongata, and medulla spinalis. One is the straight path through the left corpus striatum, the other is by commissural fibres which go to the third frontal convolution on the opposite side, and thence down through the right corpus striatum. So long as either of these routes remains open, speech is possible; it being of course assumed that the nuclei on opposite sides are so closely associated together by cross connections as

* The relation of the left side of the brain to movement and speech was discussed by the writer in the 'Guy's Hosp. Reports' for 1870, and more fully and satisfactorily by Dr Wm. Ogle in a paper on 'Dextral Pre-eminence' ('Med.-Chir. Trans.,' 1871.)

† Bouillaud, as M. Broca handsomely admitted, had also recognised the special relation of the left side of the brain to spoken language. "N'est-ce pas que nous écrivons, nous dessinons, etc., de la main droite? . . . eh bien, serait-il absolument impossible, que pour certains actes aux quels sont affectés les hémisphères cérébraux, la parole par exemple, nous fussions pour ainsi dire gauchers?" (Discours à l'Acad. imp. de Médecine, 1865.)

to be set in action simultaneously. It may be noted, as one inference from Dr Broadbent's hypothesis, that if hemiplegia and aphasia occur together, the lesion must be one which either involves simultaneously Broca's convolutions and the corpus striatum, or else affects an extensive area of the left hemisphere in the neighbourhood of the fissure of Rolando. Whichever is the case, the cause of the disease must almost always be obstruction of the Sylvian artery, whether by embolism, thrombosis, or syphilis.

If in a given case plugging of the Sylvian artery should be overlooked—and until lately it was not usual for pathologists to devote special attention to the condition of the cerebral vessels—it might easily be supposed that the only disease was in the corpus striatum, and that this had caused the aphasia. In July, 1877, the author made an autopsy in which it would have been quite possible to commit this mistake; the patient was aphasic, and the only obvious lesion of the brain was in the corpus striatum; but the Sylvian artery contained an embolism at its very commencement.

In the second place, aphasia cannot be caused by a lesion of any part of the surface, even of the left hemisphere, with the exception of Broca's region. Perhaps this position is not so completely established, because cases are very rare in which a morbid change is limited to a single spot on the convexity of the brain with sufficient accuracy to throw any light upon the question. A woman at Guy's Hospital suffering from cancer of the breast had transitory attacks of aphasia: it was conjectured that she had a secondary nodule in the third frontal convolution of the left hemisphere of her brain; and this diagnosis was afterwards verified by an autopsy. As Dr Broadbent observed in 1872, it is remarkable how large a proportion of apparently exceptional cases break down under careful scrutiny.

It has been asserted—but more on theoretical grounds than by convincing anatomical evidence—that the centre for sensory aphasia, or word-deafness, is in the superior temporo-sphenoidal convolution, and that for agraphia, or word-blindness in the angular gyrus.

Theory of aphasia.—Reverting now to the physiological causation of aphasia, we have to ask how it can be that loss of speech is always dependent on a lesion of the surface of the left hemisphere—except in left-handed persons, in whom, as we have seen, the disease is in the corresponding part of the right hemisphere. Dr Moxon first threw light upon this curious fact, and suggested an explanation which has virtually been accepted by subsequent writers. His view ('Med.-Chir. Rev.,' 1866) is that the two halves of the brain are originally symmetrical, and resemble one another in their functional capacities, but that in the course of education one side only becomes stored with those ideas of associated movements which are required for the performance of bilateral actions, such as are concerned in speech; or, as he elsewhere puts it, one side of the tongue *guides* the other; just as the right hand guides the left one when they are made to execute similar motions, it being notorious that this requires infinitely less attention than the execution of opposed motions by the two hands at once. Dr Moxon does not more fully explain why the process of education should take place on one side of the brain rather than the other; and this has been regarded as a difficulty by some later writers; but, as we have seen, the associated movements which make up the act of writing, and in which only one hand is concerned, are so closely connected with those that constitute speech, that agraphia very commonly accompanies aphasia. Now, it is true that all men do not learn to write; but there is perhaps no savage who does

not, from his earliest infancy, become accustomed to employ one hand rather than the other in the gestures by which he supplements speech; and it was a suggestion made by Broca himself that this preference of the left hemisphere for so many other purposes led to the education of convolutions of that hemisphere for the "motor processes" concerned in speech.

Theory of amnesia.—We have still to ask what are the relations of aphasia to "incoming" language—that part of the faculty of speech which consists in the recognition of words spoken by others. Many modern writers, following Dr Sanders and Dr William Ogle, admit a separate variety of aphasia, which they term *amnesic* or *amnemonic*. The characteristic symptom of it is, they say, that the patient is able to utter words, provided that he has first heard them spoken by another person. In fact, one may be able by prompting to make him speak pretty freely, although he may be incapable of saying anything in reply to a question which does not suggest its own answer. Now, it is, in the first place, to be observed that the value of this test is limited to those cases in which it yields a positive result. If the man continues altogether speechless, after one has repeated again and again the word or the sentence which he is asked to utter, one gains nothing by the experiment. That the memory for "incoming" language is not altogether lost may, indeed, sometimes be obvious from the gestures of intelligence which such a person makes if the right word is suggested to him, and from his strongly marked dissent when other words are substituted for it. Moreover, in those cases in which an attack of aphasia rapidly passes off, the patient may be able to give a complete account of all that occurred during his illness, and to state that his power of thinking was altogether unaffected. Prof. Lordat, for example, who once suffered in this way, said that he was able to combine abstract ideas, and to distinguish them accurately. Being accustomed to teach, he thought over the subject-matter of a lecture, and found that he could dispose in his mind the chief points without difficulty, and introduce any changes that he pleased in their order. He thought of the Doxology, but he was not able to recollect a single word of it. We must leave it to the metaphysicians to discuss how far it is possible for the mental processes to be carried on without the revival of words as symbols in the consciousness; but it is clear that in M. Lordat's case the fault lay in the machinery of expression alone—always supposing that he was able to recall past states of consciousness with complete accuracy.

Again, when aphasia is incomplete, so that the patient can utter one or two words, and uses them in answer to every question that may be put to him, he is often perfectly conscious of the mistakes that he commits. Every physician has seen such cases, and will remember the shake of the head, the puzzled look, the smile—half amusement, half vexation—the repeated attempts to find the right word, and the beam of satisfaction if by chance it at last comes off the tongue.

These facts seem to prove beyond dispute that the memory of language may be unimpaired, even when there is complete aphasia.

Mental condition in aphasia.—Let us now look at another side of the question. Many patients affected with partial aphasia go on uttering the same word for months or years, without seeming to know that anything is amiss with them. Trousseau studied very carefully the case of an artist who, according to his own account, suffered from nothing but failure of speech, being able to understand perfectly all that he read, and being in full possession of his intellect. It turned out that he made the grossest

mistakes in reading aloud, that he wrote one word for another without being aware of it, and that when asked to sketch a human figure he drew like a child who had never been taught. The same writer, referring to another case, lays stress on the fact that the patient, who read the newspaper, and expressed by signs that he understood it perfectly, was nevertheless in the habit of reading the sheet over and over again in the same day. And a girl under his care had for a whole year one book in her hands, and almost always read the same page. He suggests that a very good test of the understanding of an aphasic patient is to take up a book, and to read a few lines, telling the patient to follow with his eyes, and to turn over at the proper moment. The artist, whose case has already been referred to, could never do this correctly. From these observations Trousseau concludes, in spite of the facts mentioned in the previous paragraph, that in aphasia there is not merely loss of speech, but also impairment of intelligence. The lesions which cause aphasia are in most cases such as interfere with the supply of blood to a large part of one hemisphere, and therefore they often produce mental symptoms independent of loss of speech.

That other symptoms have no connection with the aphasia is proved by the fact that there is often *anosmia* in the corresponding nostril. In the 'Med.-Chir. Transactions' for 1870, Dr William Ogle refers to seven instances of this: he supposes that the so-called external root of the olfactory bulb becomes implicated when it is passing to the floor of the fissure of Sylvius.

The question is—not what defect of understanding *may* be found in aphasic patients, but what amount of intelligence they ever retain. Instances of pure aphasia, unattended with paralysis of the limbs, are especially worthy of study in reference to this question. In such cases, while the loss of speech is often absolute, the intellect may be unimpaired.*

But they are the exceptions; for one sees patients with right hemiplegia who go on for years, unable to utter a single word, or to communicate in any way with their friends. It would be sad to suppose that such persons are really in possession of all their mental faculties, and that they are living, as it were, imprisoned within an iron mask. Happily this is as unlikely as it would be melancholy, for these patients show often by their gestures that they are not miserable, and often they are obviously impaired in intellect. Moreover, those who suffer from left hemiplegia, dependent upon any extensive lesion of the right hemisphere, manifest an equally marked deficiency of intelligence, although they are not deprived of speech.

These considerations lead to the conclusion that, although loss of speech is often associated with inability to understand "incoming" language or recognise mistakes made in "outgoing" language, yet the two things are essentially independent of one another, and that aphasia associated with

* The following is a striking instance:—"About three in the morning, as near as I can guess, I woke and sat up; when I felt a confusion and indistinctness in my head, which lasted, I suppose, about half a minute. I was alarmed, and prayed God that, however He might afflict my body, He would spare my understanding. This prayer, that I might try the integrity of my faculties, I made in Latin verse. The lines were not very good, but I knew them not to be very good. I made them easily and concluded myself to be unimpaired in my faculties. Soon after I perceived that I had suffered a paralytic stroke, and that my speech was taken from me. I had no pain. . . . . I then wrote to Dr Taylor to come to me and bring Dr Heberden. . . . . In penning this note I had some difficulty; my hand, I know not how or why, made wrong letters" (Dr Johnson to Mrs Thrale, June 17th, 1783, *anno ætatis* 74). He recovered quickly in a few days from this attack of aphasia with agraphia, but without motor paralysis; and the cerebral hæmorrhage (for such no doubt it was) did not return.

amnesia indicates the existence of a lesion extending beyond the limits of Broca's region.

Dr Broadbent arrived at nearly the same conclusion (cf. 'Med.-Chir. Trans.,' 1872, p. 174). He maintains the truth of Broca's theory, so far as concerns the seat of the lesions which are attended with aphasia when it occurs independently of any failure of intelligence. This theory, however, is very far from implying that the whole of the faculty of language is localised in any one part of the hemispheres; it only asserts that a certain spot in the left hemisphere contains machinery, without the use of which a person cannot utter words, nor convey his thoughts to the pen in writing.

Recovery from aphasia.—It is remarkable that, although a patient, after a paralytic stroke, regains his power of speech, if it has been lost by "the shock," soon after consciousness is restored, and although he regains the articulation which has been damaged by partial paralysis of the tongue and lips before the power in his leg, and usually long before that in his arm—yet when a patient with right hemiplegia is also aphasic (in the strict sense of the word) his recovery of speech comes, as a rule, latest of all, or at least when there only remains slight awkwardness in the fingers or an almost imperceptible halt in the gait.

There is reason to believe that when Broca's region has been destroyed, the power of speech may still be recovered, not by regeneration of the lost tissue, but by the corresponding part of the right hemisphere taking on the action of the left; just as a draughtsman after losing his right hand would, in learning to draw with his left, be educating not only his muscles and nerves, but also the motor centres of his right hemisphere.

The writer had several years ago under his care in Stephen Ward a young man who was admitted with complete aphasia and right hemiplegia, probably the result of syphilitic stenosis of his left Sylvian artery. While slowly regaining power of movement and improving in health, the sister of the ward tried to teach him to speak, and found the only way was to begin from the beginning by naming objects (and printed words—so as to teach him to read again also), and making him imitate the sounds over and over again. The result was a good degree of success. In the same way the monophrasic woman mentioned before, who said *ten-ten*, was taught by two successive clinical clerks to print letters with her left hand, and to recognise printed letters. Here the success was but slight, perhaps owing to the greater age of the scholar or the smaller patience of the teachers.

Aphasia in lunatics.—There is still one point of view from which it is necessary to consider the pathology of aphasia; namely, as to whether this symptom, when occurring in persons of unsound mind, is necessarily dependent upon a lesion in Broca's region. Dr Bastian mentions the case of a lunatic who did not utter a single word for several years, except during two brief intervals, once when he had an attack of pleurisy that lasted some days, and once when he was suffering from toothache. The late Dr Forbes Winslow relates a similar instance; a person who had been insane for fifty-two years did not speak during thirty years, but recovered speech and answered questions perfectly well during the last fifteen years of his life. In such cases there is surely no reason to suppose that there was any local lesion in the brain.

The same observation may be made in reference to some of the cases which Trousseau gives as examples of aphasia; for instance, that of the lady who was wont to welcome her visitors by exclaiming *pig*, *animal*, *fool*, without

understanding the meaning of the expressions she used. It is also applicable to an example of supposed agraphia, in a lunatic at Broadmoor, which has been recorded by Dr Bastian; here the inability to write was of an amnesic type, and may be regarded as one of the symptoms of insanity ('The Brain as an Organ of Mind,' 3rd ed., p. 660).

APOPLEXY.*—This term originally signified a "stroke" (see note), by which the patient falls "like an ox struck down by the butcher;" and we shall here use it in this its proper sense. But the word apoplexy was transferred by metonymy to effusion of blood upon the brain, which was found to be the most common cause of such attacks when autopsies began to be made; and this use of the word was afterwards extended to hæmorrhages in other parts, so that pulmonary apoplexy, retinal apoplexy, and apoplexy of the suprarenal capsules are spoken of. There is, however, no advantage in employing apoplexy as a mere synonym for cerebral hæmorrhage; and in clinical medicine it is desirable to have a name for that form of coma which is due to disturbance of the cerebral circulation from some local cause acting within the cranial cavity—usually but not always rupture of an artery—as distinguished from failure of the heart's action on the one hand, and, on the other, from narcotic or alcoholic poisoning, uræmia, epilepsy, or external injury. For this purpose the word apoplexy appears to be suitable, and it has been so applied by a long chain of authorities, including some of the most distinguished names in medical literature.

Symptoms.—A patient in an apoplectic fit lies "deprived of sense and motion." He cannot be roused, but there may be varying degrees of insensibility. Sometimes the well-known voice of a wife or son may elicit an unintelligible muttering or growling sound in reply, or the application of a spoon to the lips may cause them to be closed in automatic refusal of food; or one hand may be used to rub or scratch the side of the face or body. More often there are no such indications of even partial consciousness; the limbs remain in whatever position they happen to fall; the respiratory movements and the beating of the heart alone indicate life.

The pupils are generally torpid, and often insensible to light; they are sometimes equal and of normal size, sometimes both dilated or both minutely contracted, sometimes unequal. The conjunctivæ can often be touched without exciting reflex movement in the eyelids. The cutaneous reflexes are diminished or abolished, rarely increased. Pinching or pricking the skin seldom leads to any manifestation of consciousness. Sometimes, however, the hand or the foot is drawn away on one side and not on the other, or it may be found that when the arms are lifted and allowed to drop upon the bed, one is a dead weight; or the arm and the leg on one side may be rigid, on the opposite side relaxed. Such indications of hemiplegia are important, as is also the conjugate deviation of the eyes described above (p. 563).

The appearance of the countenance varies greatly in different cases; sometimes it is pale, sometimes congested and purple, with lividity of the lips and tongue; the features are often turgid and swollen, and the forehead and cheeks bathed in perspiration, which saturates the linen and stands in large drops upon the face.

The *temperature*, as measured by the thermometer, has been carefully

* *Gr.* Ἀποπληγία, ἀποπληξία, a stroke, a classical Greek word from ἀποπλήσσω, to strike, to disable (Soph., 'Antigone,' v, 1189, where it is applied to syncope). Used by Hippocrates of a "stroke" of the palsy, but applied to one limb, as σκέλος ἀποπληκτικόν, a paralysed leg.—*Lat.* Sideratio.—*Fr.* Coup de sang.—*Germ.* Schlagfluss.

investigated by Bourneville. He finds that there is at first a slight fall, amounting to about 2° Fahrenheit. In rapidly fatal cases this continues until death; but if life is prolonged the temperature rises, and for several days it may remain at about 100°. When death occurs at an interval of more than ten hours from the commencement of the attack it may be preceded by a rapid elevation of temperature. This was noticed by Dr Hughlings Jackson, and, as he observes, is a very unfavourable sign. In one fatal case at Guy's Hospital the thermometer registered 107°. When recovery is to take place, the temperature, if it has risen, commonly returns to the normal point two or three days after the commencement of the attack.

The *pulse* may be either increased or diminished in frequency; the prognosis is bad if it is greatly above or greatly below the average—say below 60 or above 120. Unfortunately, the converse is not always true; for example, Dr Jackson mentions a case in which the pulse was 72 within five hours of the death of the patient. As a rule the pulse becomes more rapid as the case goes on towards a fatal termination; but the worst sign of all is irregularity of the pulsations of the heart, a succession of rapid beats being followed by a series of beats at long intervals. Formerly great stress was laid upon the "full" and "labouring" character of the radial pulse in apoplexy, and it was supposed to be a proof of the necessity for venæsection. We now refer this character either to want of elasticity from atheroma of the aorta, or to the high arterial tension of chronic Bright's disease.

The *breathing* is sometimes infrequent; there may be as few as three or four respirations in the minute. Towards the last it often happens that the patient ceases to breathe for perhaps a minute, and a purple flush diffuses itself over the countenance, but afterwards a deep breath is again drawn, the face resumes its natural colour, and the respiration goes on as before. This may be repeated several times, until at length a final pause occurs; the heart may then go on beating for a considerable time, but at length its pulsations cease and the patient is dead. In some cases the respiration assumes the characters observed by Cheyne and Stokes, and known by their names.

In many cases apoplexy destroys life by a more gradual increase of obstruction to the breathing, which seems to depend upon a concurrence of several distinct causes. One of these is the supervention of inflammatory œdema, beginning in the bases of the lungs and spreading upwards through the back parts of those organs. Another is the accumulation of a thin secretion in the air-passages, which becomes beaten up into a froth by the inhaled air, and may completely fill the main bronchi and even the trachea. It is probable that the pouring out of this fluid is often partly caused by the entrance of fluid nourishment; for when a patient is comatose, milk and beef-tea and brandy are very apt to run down into the larynx without giving rise to any warning cough or sensation of choking; but no doubt another important cause is the impediment to the act of respiration which is due to paralysis of the tongue and fauces. Everyone who has made many examinations on the bodies of those who have been suffocated knows how large a quantity of frothy fluid is found in the air-passages, even when death has been rapid; but an apoplectic patient may lie for days in a condition in which there is much interference with the breathing. This is shown not only by the lividity of his countenance, but also by the fact that the entrance and exit of air are accompanied by a noise which is commonly called *stertor*. The causes that lead to the occurrence of this sound were clearly pointed out by Dr Bowles, of Folkestone, in the 'Med.-Chir. Trans.' for 1860. He

admits that when the mouth is partially open the soft palate sometimes drops upon the tongue, and vibrates as the air rushes in beneath it; but he ascribes far more importance to a change in the position of the tongue itself; he shows that when the mouth falls open, the point of attachment of the lingual muscles to the symphysis is carried backwards, and he thinks that the tongue then comes into contact with the posterior wall of the pharynx. As might be expected, this is especially apt to occur while the patient lies supine. Dr Bowles finds that turning him over on his side, with the mouth inclined so that the saliva and mucus can drain away, often causes the entire disappearance of stertor, and may be followed by a decided improvement in some of the other symptoms.

When the breathing is noisy the cheek is often puffed out at each expiration in consequence of paralysis of the buccinator muscle; this symptom suggests an unfavourable prognosis.

Duration.—The period that an apoplectic seizure lasts is very variable. In some exceedingly rare instances death takes place a few minutes after the commencement of the cerebral symptoms. Thus Dr Jackson mentions the case of a woman who was sitting at the tea-table, when she stopped in the middle of a laugh, cried out, "Oh, my head!" fell back in her chair, and died within not more than *five minutes*. Abercrombie relates a similar occurrence in a woman who was one evening attending a crowded meeting, and who seemed to be in perfect health. Towards the conclusion she uttered a loud scream, and fell down insensible. She was immediately carried out, remained pale and unconscious, and within *five minutes* she was dead. In both these cases meningeal hæmorrhage was found.

In 1864 there was brought to Guy's Hospital the body of a woman who had died *almost instantaneously* as she was returning home from the theatre with her children; in that instance also a large quantity of blood had been poured out over the sides and base of the brain. The author once found at an autopsy a large clot in the left hemisphere, bursting into the lateral ventricle. The patient, a man, aged forty-one, was in a surgical ward for some laryngeal affection, and had been sent down to the dispensary to fetch the medicines; on his way he had a fit, became comatose, and died within *ten minutes*.

One would have expected that hæmorrhage into the pons would often cause instantaneous death. But Dr Jackson remarks that he has never seen such a case, although he has known a woman lie deeply comatose for some hours, in whom this part of the brain was hollowed out into a mere shell. In the most rapidly fatal case of a clot in the pons recorded at Guy's Hospital death occurred in *forty minutes*; two patients lived each for two hours, one nine hours and a half, one thirteen hours, one sixteen hours, and one *two days*.*

Meningeal hæmorrhage commonly proves fatal within forty-eight hours. But in the ordinary form of apoplexy, in which blood is effused into the neighbourhood of the corpus striatum, life is often maintained for a much longer period. In twelve cases at Guy's Hospital death did not occur until between the second and the seventh days; and in six cases at the end of ten, twelve, thirteen, sixteen, nineteen, and again nineteen days respectively. It is worthy of remark that two among these six cases were examples of a very rare occurrence, the formation of a clot within the substance of the brain, which was large enough to destroy life, but which yet failed to reach either

* Of seventy-eight cases of hæmorrhage into the pons collected by Bode and quoted by Ross, forty-six proved fatal within twenty-four hours.

the lateral ventricle or the surface; and in a third case the seat of the hæmorrhage was quite exceptional, being the interior of one occipital lobe.

In twenty-four consecutive fatal cases of cerebral hæmorrhage under the writer's care, death took place under twelve hours in seven, under twenty-four in eleven; on the second or third day in three; on the fourth, fifth, or sixth in eight; and on the ninth in two.

An apoplectic attack does not necessarily prove fatal. In many cases consciousness is regained. This always takes place more or less gradually. After a few hours one is perhaps able to rouse the patient so that he will give his name, although when left to himself he still takes not the slightest notice of anything that goes on in the room. For several days he generally remains drowsy and apathetic, or his ideas may be confused and perplexed; or he may even be delirious, talking incoherently, and throwing himself out of bed. In such cases, however, the prognosis is not good, in spite of the fact that the coma has passed off. Nothnagel says it is exceptional for recovery to occur if insensibility persists for as long as forty-eight hours. In cases which terminate favourably, as the patient regains consciousness the symptoms of local damage to the brain become manifest, of which the most important is hemiplegia, with or without aphasia.

Onset.—The older writers laid great stress upon certain symptoms which they believed to be frequent precursors of apoplexy, and which they therefore designated "warnings" or "molimina." But under these names they included a great variety of complaints, without any definite limits as to time: —epistaxis, for instance, happening several months before the cerebral attack; or an ecchymosis of the conjunctivæ, perhaps the result of a violent effort in coughing or sneezing; or even the frequent recurrence of giddiness or headache in a person advanced in years. These last symptoms, indeed, are not unlikely to be due to a morbid condition of the blood-vessels of the brain, which may presently lead to their rupture; and the local patches of softening, which we have seen to be more direct effects of such vascular changes, afford a ready explanation of other nervous disorders—such as thickness of speech, diplopia, partial ptosis, sensations of numbness and formication in the hand, or in the fingers, partial loss of power in the arm, or dragging of the foot,—any one of which, if occurring in an old man or woman, is pretty sure to be taken as threatening the supervention of an apoplectic seizure, even though it may after a few weeks disappear, leaving the patient apparently as well as ever. One cannot altogether reject this view of the matter; but it is to be remembered that each of the symptoms in question may arise from other causes, and that even if one could be sure that they were due to a diseased state of the cerebral arteries it would not necessarily follow that apoplexy was about to occur.

On the other hand, the very same symptoms may be actually results of cerebral hæmorrhage, a vessel in the brain having already given way; and, unless the extravasation remains small in amount, the patient is very likely to become comatose a few hours later. In such cases it is obviously incorrect to speak of "warnings."

Abercrombie, many years ago, pointed out that cerebral hæmorrhage comparatively seldom leads to sudden loss of sense and motion, or (in other words) to the classical form of apoplectic seizure. A precise clinical history of the cases in which extravasation of blood is most apt to be found is given in his celebrated work. The first symptom, he says, is a pain in the head, which may be so violent as to make the patient scream. The face

at the same time becomes pale, the body cold, and the pulse very weak; there is sickness, or even vomiting; and he may fall to the ground faint and exhausted. Often a slight convulsion occurs. After a little while he may be able to walk home; he is quite sensible, but oppressed; he remains cold and feeble, with cadaverous pallor of the countenance. By degrees he recovers his warmth, his face regains its natural appearance, and his pulse improves in strength. Then he becomes flushed, he answers questions slowly, and gradually he sinks into coma, from which he rarely recovers.

All subsequent writers have recognised the truth of this picture. After fracture of the skull, when the middle meningeal artery is torn through, there follows a very similar series of events; and the name of "ingravescent apoplexy" has been given to cases which run such a course. But, until recently, most observers have regarded this as only one form of the disease, and have supposed that cerebral hæmorrhage often begins with sudden coma. Now, it is true that the patient is frequently insensible when first seen. Perhaps he is picked up in the street, or found lying on the floor, or in a water-closet; or the seizure may have come on during sleep, so as to give no opportunity of tracing its earlier symptoms. But Trousseau says that although his attention was for fifteen years directed to the question, he did not in that time meet with a single instance in which, when an attack of cerebral hæmorrhage occurred in the presence of witnesses, it did not begin more or less gradually. An exception must be made for those rare cases of hæmorrhage which destroys life in a few minutes (p. 580). Another exception is allowed by Trousseau himself for certain cases which begin with an epileptiform seizure, and this is endorsed by Dr Hughlings Jackson, who has laid special stress upon the frequency of this mode of commencement. But "ingravescent apoplexy" is no particular modification of the disease; it is the rule in cases of large cerebral hæmorrhage.

Moreover, it must be admitted that the symptoms before coma sets in are more variable than would appear from Abercrombie's description. Pain in the head is often absent. Not infrequently the only thing which is noticed by the patient is a numb feeling, or a sense of weight in one of the limbs. Thus Trousseau relates the case of a woman who while returning from market noticed that she dragged her right leg, and that her right arm felt heavy, so that she changed into her left hand a folded newspaper which she was carrying, lest it should fall into the mud. She walked upstairs into her room, took off her clothes, and got into bed; after which she became hemiplegic and comatose, and remained in a state of stupor for three days. Another patient of Trousseau had noticed while at dinner that one of his hands felt heavy; he was not giddy, but faltered a little in his speech. He tried to rise from his chair, but one of his legs being paralysed he fell down. His children lifted him up, and with their assistance he walked into the next room. Trousseau arrived in three quarters of an hour, and found him perfectly conscious; but his left arm and leg were almost powerless. Profound coma set in in a few hours, and he died the next morning.

Another modification of the early symptoms produced by cerebral hæmorrhage is marked by a transitory loss of consciousness, from which the patient quickly recovers, so that it is separated by an interval from the final coma. To this Trousseau gives the significant name of "cerebral surprise." A satisfactory explanation is not easy. We must suppose the injury to the brain at the moment when the vessel gives way causes a shock that is diffused over the nervous centres. Trousseau cites, in illustration, the expe-

riment of trephining the skull of a dog or rabbit, and introducing a leaden ball into its interior; symptoms of stupor, he says, are immediately manifested, which quickly pass off, leaving a degree of hemiplegia proportionate to the compression. But Nothnagel says that in hundreds of observations upon animals, in which he injected chromic acid into the brain, or produced artificial extravasation of blood, he never saw such an effect produced. He refers to the analogous effect of suddenly cutting through the spinal cord, in instantaneously suspending for a time the reflex excitability of the centres below the line of section (cf. p. 432).

Locality of the lesion.—Speaking generally, the most probable seat of cerebral hæmorrhage is the most common one—the region of the corpus striatum. If a second or third attack has proved fatal there is very likely ventricular hæmorrhage. If death has come on unusually rapidly there is probably either moderate effusion in the pons, or a very large one on the surface, or into the ventricles.

The fact that lesions affecting the cerebral convolutions so often give rise to convulsive seizures has led some observers to suppose that such symptoms are especially apt to occur in cases of *meningeal hæmorrhage;* but our cases do not bear this out. Nor is rigidity of the limbs noted as having been commonly present—a fact which Dr Goodhart pointed out in the 'Guy's Hospital Reports' for 1876; but it does seem that a definite hemiplegia is comparatively seldom observed in this form of apoplexy; the blood makes its way too easily along the subarachnoid space at the base of the brain to compress one hemisphere more than the other. In some cases the coma is preceded by delirium.

A curious question is, whether it is possible to make out at the bedside the existence of extravasation into the *lateral ventricle* when the original lesion is in one of the basal ganglia. The recurrence of coma, after recovery from a first seizure, has been attributed to this cause by Nothnagel and some other writers; but we have seen that it is common in all cases of apoplexy. Others have supposed that the presence of ventricular effusion is indicated by paralysis of all four limbs succeeding to hemiplegia, or by early rigidity of the arm and leg on the side opposite to the lesion. The latter opinion was maintained by Durand-Fardel. But probably the exceptional cases in which profound coma and death are caused by a clot limited to the substance of one hemisphere do not differ from those of extravasation into the ventricle. In 1874 the author made an autopsy in the case of a lad, aged seventeen, who had died fifteen hours after having been found lying on the ground, unconscious. All the cavities of the brain, including the third and fourth ventricles, were full of clot, which was in equal abundance on the two sides. No cause for the hæmorrhage could be found; but there had been repeated epileptiform fits. In 1876 an old woman died in the Clinical Ward of Guy's Hospital of a seizure which was unattended with paralysis of the limbs. The right lateral ventricle was full of clot, which had come from the superficial part of the caudate nucleus, the internal capsule being quite uninjured. Thus the absence of hemiplegia is not peculiar to cases of meningeal hæmorrhage.

In apoplexy of *the pons* the pupils are often contracted to pins' points, and the respirations are exceedingly infrequent—perhaps not more than six or four in the minute; but in one case dilatation of the pupils was present, and the breathing is sometimes hurried and irregular. Nothnagel seems to think that convulsions are more apt to occur than when blood is effused

into other parts of the brain; but this seems to be doubtful. Other symptoms enumerated are rigidity of the neck, tonic contractions of the limbs, vomiting, hyperpyrexia, and in rapidly fatal cases paralysis of the limbs on both sides. Crossed hemiplegia can seldom be made out satisfactorily while the patient is insensible. Hæmorrhage into the pons sometimes leaves behind it a permanent difficulty of articulation.

Diagnosis.—In the diagnosis of apoplexy one has to bear in mind many morbid conditions with which it may be confounded; in scarcely any other disease is it more important or more difficult to form a right judgment. The liability to error arises in two different ways: sometimes from the absence of any history as to the origin of the attack, or from the history being vague or untrustworthy; sometimes from the doubtful nature of the symptoms.

Thus a man may be discovered comatose and stertorous in bed or upon the pavement of the street, and there may be no one to say what has happened to him; or it may be impossible to depend upon the statements of those in whose company he is found; or there may be suspicion of foul play. In all such cases the following possibilities must be taken into careful consideration before deciding that the case is one of apoplexy: (1) that his brain has been injured by external violence, as by a blow or a fall, with or without fracture of the skull; (2) that he may have taken poison; (3) that he may be intoxicated; (4) that the coma is due to pyæmia, to uræmia, to diabetes, or to the effects of an epileptic fit.

1. As regards *injury to the brain*, difficulties arise in several ways. There may be no bruise upon the face or head, and no displacement of any part of the calvaria, so that it is only at the autopsy that we discover that the skull is fractured, or that the prominent parts of the brain on one side are bruised in such a way as to show that external violence had been the cause of death. Even then, however, one is by no means justified in concluding that the case was originally one of accident or of injury inflicted by others. The man may have had a fit or been drunk, and in falling he may have fractured his skull. Thus in 1859 a man was admitted into Guy's hospital who was driving in a cart, when he fell and was picked up insensible. He died after four days, and the surface of the brain was found to be extensively bruised; but as he was known to be subject to epilepsy, it appeared clear that one of these attacks had been the cause of his falling. We must bear in mind that meningeal hæmorrhage often occurs as the result of disease; it is, therefore, only when the brain-substance itself is ecchymosed that we are justified in attributing to external violence cases in which blood is found effused on the surface of the brain. On the other hand, we have had several instances in which a severe injury of the skull with laceration of the cerebral convolutions has been associated with hæmorrhage into the central parts, exactly like that which occurs in apoplexy. Some of these have undoubtedly been examples of spontaneous effusion of blood into the brain, the superficial injuries having been caused by the patient's falling upon a kerbstone or a hard floor, or the like, but it is certain that a blow or fall upon the head may in rare cases cause rupture of an artery in the interior of the organ. Thus in 1866 a man was admitted into a surgical ward an hour before his death who had been steering a barge on the Surrey Canal, when a steamer came too close and struck the rudder; the tiller knocked him over, and he was picked up insensible. There was no injury to the cranium, nor to the surface of the

brain. The right lateral ventricle was found full of effused blood, the septum was broken down, and there was a moulded clot in the fourth ventricle. In 1868 a patient in the ward for ophthalmic cases, being unable to see, missed his footing and fell, striking his left temple. A large quantity of blood was effused upon the cerebral convolutions, but the right corpus striatum was also extensively ecchymosed, and had in its interior a mass of blood of the size of a hazel-nut. In 1870 a man came to the hospital with a cut on his forehead, saying he had slipped upon a flight of stone steps at the Victoria Theatre. He ultimately became hemiplegic and comatose, and died, and the right lateral ventricle was found to contain a large clot, the blood having oozed from a rent in the thalamus. In 1855 a patient was admitted who had been found lying by the side of a crane, the handle of which seemed to have struck him on the head, there being a wound of the scalp. At the autopsy the left corpus striatum and thalamus were found to be destroyed by a large mass of blood lying between them.

Sometimes it is impossible to decide between injury and disease. In 1862 a man was admitted who had fallen several feet upon his head from a platform on which he had been working. His skull was found fractured, but the lateral ventricles were full of blood, and the right corpus striatum was broken up by a large clot which projected through an opening in its surface. His fellow-workmen thought he had overbalanced himself, as he had shown no sign of having a fit; but Dr Wilks, who made the autopsy, left the question open in his report.

It will be noticed how closely the appearances found in these five cases resembled those of spontaneous cerebral hæmorrhage. In the reports of three of them it is distinctly stated that the kidneys were granular, or wasted, or cystic. So that, although the rupture of the artery was the direct result of a blow or fall in each instance, it seems that they may fairly be regarded as examples of ordinary apoplexy, the main cause having probably been indirect, namely, the existence of chronic disease in the coats of the vessel. Most likely, if no accident had happened, these patients would have died of spontaneous hæmorrhage into the brain a few weeks or months later. One cannot be surprised that it is impossible to diagnose such cases in the absence of a history during life, when the right interpretation is so difficult after an autopsy.

2. Narcotic *poisoning* may be mistaken for apoplexy, or *vice versâ*. We have seen that in some instances in which blood is effused into the pons the pupils are contracted and the respiration is regular and very infrequent, just as in persons who have taken large doses of opium. The author was summoned one night to see a lady some six or seven miles away from London, and found her comatose, with the symptoms just mentioned. There was a suspicion that she had poisoned herself, for the day was the anniversary of the death of a son; and a few hours before her illness began she had said to a sister, who had come to visit her, "Well, have you come to see me die?" But she had been sitting up in bed talking to this sister, and apparently as well as usual, when she suddenly fell back comatose. This fact appeared to exclude the possibility of her having taken opium; and the conclusion seemed clear that there was hæmorrhage into the pons. But the next morning she was quite well again, and never confessed that she knew the cause of her illness. The case shows that a patient deeply comatose from opium *may* recover without being walked about or swallowing strong coffee

or stimulants. In one case of suicide from prussic acid, recorded by Dr Stevenson ('Guy's Hosp. Rep.,' 1869), death did not occur for more than an hour and a quarter after the discovery of the fact that the poison had been swallowed, and the symptoms could not have been distinguished from those of apoplexy; but all difficulty was removed by the fact that there was on the table a half-empty, although corked, bottle containing some of the poison. A marked odour of prussic acid was diffused through the room, but the patient's breath seemed not to smell of it.

3. It is often difficult, and sometimes impossible, to distinguish apoplexy from *alcoholic intoxication*. A man who really is dead drunk may be supposed to have cerebral disease; but the only result likely to follow this mistake is that a patient who had been expected to die, or at least to become hemiplegic, may in a few hours completely recover. In 1868 a boy, aged fourteen, was admitted into Guy's Hospital, who with another boy had stolen two bottles of brandy, and, according to his companion's statement, had drunk a reputed quart (twenty-six and two thirds fluid ounces) without any admixture with water. This occurred at about 2.15 p.m.; by four o'clock he was insensible, and he was at once taken to the hospital. He was completely insensible, but without stertor; his pupils were at first of natural size, but they afterwards became contracted. An œsophageal tube was passed, and the contents of the stomach were pumped out, with a most powerful odour of brandy. He was then put to bed, and an enema of coffee was administered. He remained unconscious for twelve hours; then he asked for a glass of water, and before long was well. Dr Stevenson, commenting upon this case in the 'Guy's Hosp. Reports' for 1869, says that no doubt the boy owed his recovery to the prompt use of the stomach-pump.

It very rarely happens that anyone swallows a fatal dose of alcohol except for a wager or out of bravado, when the medical man who is called to the case is sure to be told of what has occurred, and to mistake intoxication for apoplexy is to commit an error which is scarcely ever likely to be injurious to the patient, even though one should omit to use the stomach-pump. On the other hand, to suppose that a man who has apoplexy is merely intoxicated is a most serious matter. This is the mistake which is often made by the police, and into which medical men themselves have too often fallen, from failing to appreciate the real difficulties in the diagnosis between the two conditions. Perhaps the patient is found in a public-house, or is known to have been drinking heavily, or smells strongly of spirits. The only safe course for us is to ignore these facts altogether. The odour of brandy, does not even prove that any stimulants have been taken before the attack commenced; it may have been administered afterwards. The only safe rule, which should never be departed from, is that a man who is insensible, and who cannot be roused, is never to be sent away to the police station, nor be left by himself to sleep off the supposed drunkenness. He should always be put to bed and carefully watched. The presence of convulsions, hemiplegia, or inequality of the pupils may make one certain that the case is due to something beyond alcoholic intoxication; and the same conclusion may be drawn, although with less confidence, from the occurrence of rigidity of the limbs, or of stertor; but the *absence* of any or all these symptoms is no disproof of cerebral disease.* The most

* It would seem that by testing the urine one can obtain positive evidence that a poisonous dose of alcohol has been swallowed. According to Anstie, this inference may be drawn if to fifteen drops of his chromic acid solution (made by dissolving one part of

important cases, however, are not those in which coma, or even partial unconsciousness, is present, but rather those in which the patient is noisy and excited, grimacing and throwing his limbs about; one is perhaps sure that such a man is drunk, but it may be impossible to say whether or not he also has hæmorrhage going on within his skull. Rupture of a cerebral artery seems often to be the direct result of indulgence in drink, and to detect its early symptoms must obviously, in such circumstances, be exceedingly difficult. Medical men who have had the largest experience are those who most freely acknowledge the impossibility of speaking confidently, particularly when the patient is advanced in years, and may have chronic disease of his heart, or of his kidneys and cerebral arteries.

4. Even when we have decided that a patient who is comatose is not suffering from an injury to the head by external violence, nor from narcotic poisoning, nor from the effects of alcohol, there are still some other questions to be considered, though of subordinate importance, before we can safely conclude that he has apoplexy as above defined (p. 578). One possibility is that the case may be one of *pyæmia*, or some other specific febrile disorder. The author saw two cases in each of which a profound and rapidly fatal coma was proved by the autopsy to be due to this cause. One was that of a man, aged twenty-six, who was brought to the hospital insensible, and died in a quarter of an hour. Very little could be learnt about him; but it was stated that he had recently arrived on board ship in the port of London, and that he had been giving evidence in a court of law on the day of his death. The brain appeared healthy; but there was suppurative inflammation of the peritoneum, and also of one knee-joint. The other case occurred in a girl eleven years old. She was admitted into the hospital one morning at half-past six, and, immediately after being put to bed, she gave one gasp and died. Her mother said that she had been quite well until the previous day, when she complained of pain in the left side on returning home from school in the afternoon. She went to bed, and fell asleep; but at 4 a.m. (two hours and a half before her death) she was found to be unable to speak, and to be rolling over and throwing her arms and legs about. When brought to the hospital she was unconscious, and was breathing heavily with froth upon her lips; the right pupil was dilated, the left contracted. The temperature was 101·2°, the pulse 116, the respirations 32 in the minute. All that could be discovered at the autopsy was that the surface of the right lung was ecchymosed, and that there was a partial hepatisation of the lower lobe of the left lung, with much lymph effused on the pulmonary pleura over a circumscribed area. The probable interpretation of these cases is that there was some early change in the brain so intense as to kill before it could be recognised by the naked eye. We shall see that tubercular meningitis may prove fatal at a stage when the microscope is required to demonstrate its presence.

5. Another possibility is that the coma may be due to *uræmia*. The older pathologists were familiar with cases in which, finding no blood effused upon the brain, they were driven to suppose that the cerebral symptoms which had proved fatal were due to the presence of fluid in the ventricles and beneath the arachnoid; and they described the affection under the name of "serous apoplexy." At the present day, however, this name, and the idea on which it was based, have alike fallen into oblivion.

bichromate of potass in 300 parts by weight of strong sulphuric acid) one drop of urine is added and turns immediately of a bright emerald-green colour. In the case of the boy who was under my care in 1868, it took two drops to produce this effect.—C. H. F.

Several years ago Dr Wilks suggested that the majority of the cases in question were examples of Bright's disease; and his opinion has been endorsed by all later writers. But in reading the detailed clinical reports given by Abercrombie it is far from clear that the cause of death in his cases was uræmia, rather than some one of the less obvious cerebral lesions which at that time would have escaped recognition; and there seem to have been few cases at Guy's Hospital in which the symptoms were like those of an apoplectic attack, and in which the only disease that could be discovered at the autopsy was in the kidneys. On the other hand, it has often happened that albumen has been detected in the urine of a patient who had been brought into the ward in a state of insensibility, and uræmia diagnosed, until a *post-mortem* examination showed that the coma was due to cerebral hæmorrhage. The common occurrence of albuminuria in such cases is partly due to the frequency with which the kidneys are found to be granular; but albuminuria is often met with in apoplectic patients whose kidneys are afterwards proved to be perfectly healthy; so that it is probably to be regarded as a result of venous congestion.

Among the few instances in which an apoplectiform seizure has been found at Guy's Hospital to have been caused by renal disease, without any cerebral lesion being discovered, the following may be cited.

In 1867 a man, aged forty-eight, was brought to the hospital, conscious, but unable to speak; his friends said that he had often suffered from headaches; the day before his admission he complained that his mind was wrong; his manner was strange, but he answered when spoken to. Afterwards he became insensible, passing his excretions under him. At one time he regained his senses sufficiently to say "yes" and "no;" and he could swallow; but he soon relapsed into coma, and died on the following day. His pupils were rather contracted, and his breathing was laborious. The brain was found pale and anæmic; the ventricles were nearly empty. The heart was hypertrophied; the kidneys were extremely wasted, as a result of dilatation of the calyces secondary to stricture of the urethra.

In 1868 a man, aged forty-four, who had been intemperate and gouty, and who had been suffering from a severe headache for a fortnight, was brought to the hospital in a drowsy state, from which he could just be roused to speak. He had had a fit, and had bitten his tongue. His body and limbs were in a state of constant jactitation. His pupils were rather contracted. His urine was retained, but his fæces were passed into the bed. Before his death he had two more fits. The only changes found in the brain were that it was tough and wasted, with large ventricles, and with an excess of fluid in them; but the kidneys weighed only two ounces and a half, and were exceedingly granular. At the bedside it would probably have been impossible to say, in reference to either of these cases, that an artery had not given way within the brain.

It has sometimes happened that patients already under treatment in the hospital for Bright's disease have died quickly with cerebral symptoms; but under such circumstances hæmorrhage is comparatively infrequent. In the immense majority of cases the stupor caused by uræmia alternates with convulsions, and passes off again and again without leaving hemiplegia—so as to prove that no structural damage has been done to the brain.

6. Coma from *diabetes* or from *cholæmia* may simulate apoplexy, but the presence of glycosuria or of jaundice will decide the nature of such cases.

7. Of cerebral diseases, the one which is most likely to be mistaken for

apoplexy, is a form of *epilepsy* which was described by Andral and other French writers as a separate malady under the ponderous name of "apoplectiform cerebral congestion," until Trousseau pointed out its real nature and distinguishing characters. In all probability Abercrombie would have included it under what he termed "simple apoplexy," in which after death no morbid appearance could be discovered in the brain. Sir William Gull in his lectures used to teach that "simple apoplexy" was nothing but epilepsy; but a careful perusal of Abercrombie's cases leaves one doubtful whether all of them can be fairly interpreted in this way, and whether, if precisely similar ones were to occur now, the more accurate pathological methods which we possess would not enable us to place them in different categories. The question, however, is not of any practical importance.

Among the cases related by Trousseau are the following:—In 1845 a gentleman, aged forty-two, was found in his bed insensible; his face was turgid and livid, there was stertor, and all power of motion and sensation was lost. How long he had been in this condition his wife could not tell—she had been awakened by a strange snoring noise. Trousseau had the patient placed in a half-sitting posture, threw cold water in his face, and applied ligatures round the upper part of the thighs to retain the blood in the legs. Scarcely one hour elapsed before he regained his senses and the use of his limbs, and on the following day great lassitude was the only remaining symptom. Some time afterwards the same physician was fetched in great haste to a neighbour, aged seventy, who was said to have been attacked with apoplexy on the Boulevards. He had been unconscious for a quarter of an hour, but was recovering his senses when Trousseau arrived. He did not at first recognise him, and looked vacantly round, throwing his arms and legs about without knowing what he was doing. Within a few hours, he gradually and completely recovered, although no active treatment was used.

In both instances the real nature of the disease was afterwards established by the recurrence of the seizures at more or less frequent intervals, attended with all the symptoms of epilepsy. Indeed, it must be observed that in each case the attack began when no skilled observer was present, so that there is no proof that convulsions did not occur. And Trousseau himself goes on to say that in almost every instance of the same kind in which he was consulted, and in which the commencement of the seizures had been seen, "nervous twitches" or convulsions had been present. Thus, after all, the identification of "apoplectiform cerebral congestion" with epilepsy involves little more than the recognition of two facts—that the spasms which usher in an epileptic seizure may be but slightly marked, so as to escape the notice of a non-professional observer; and that when one is called to a patient who is comatose one must make sure that the disease is *not* epilepsy before committing oneself to a diagnosis of apoplexy. In determining this latter point, we must first ascertain whether the patient has ever before suffered from seizures which could have been epileptic.

The author was one evening called out in great haste to see an old man, and found him lying on the sofa in his sitting-room, comatose and stertorous, with puffing cheeks and a purple countenance. His pupils were dilated, but the general aspect suggested positive apoplexy, had not his housekeeper, who had found him insensible, known that he had once or twice before had epileptic attacks. Before long he completely recovered. One of Trousseau's cases is that of a solicitor, aged thirty-five, who was sent to him from

the country, with the history that in the course of the previous six months he had had three apoplectic fits. They had lasted, however, an hour at the most, and they had left no paralysis behind them. Trousseau accordingly declared the disease to be epilepsy, and his diagnosis was before long found to be correct. But of course there must in every instance be a first attack, and then the criterion fails, and if this seizure should prove fatal the real nature of the disease must remain a matter of inference. In 1865 a man, aged sixty-seven, a tanner in Bermondsey, was brought into the hospital comatose and died in a few hours. He had followed his occupation until the day of his death, when he was found insensible, having fallen to the ground. He had convulsive movements and appeared to be paralysed on the right side. It could not be ascertained that he had ever before had a similar attack, but he was said to have suffered much with his head. No recent morbid change could be found in the brain, but there was chronic wasting, the convolutions being shrunken, with much fluid in the sulci, and the ventricles being enlarged and their surface granular; the skull also was dense and had no diploë, and the membranes were thickened and opaque.

A very similar case occurred in 1876. A woman, aged sixty-two, was brought into the Clinical Ward, having fallen down in the street in a fit. The right pupil was larger than the left. There was right hemiplegia, with complete anæsthesia in the right arm and in the right side of the face. After a time she partially regained her consciousness, but had several convulsive attacks. The coma then again became deeper, and she died at the end of five days. No morbid change could be found in the brain except wasting with dilatation of the ventricles; and there was only slight wasting of the kidneys.

There appears to have been no marked engorgement of the cerebral vessels in these instances; but from the way in which death is brought about it is evident that in similar cases such a condition is very likely to be found. May not some of the cases which Hammond and other writers describe as fatal "congestion of the brain" be really epilepsy?

There is a further criterion by which we may often distinguish an attack of epilepsy from one of apoplexy, and on which Trousseau has laid special stress—the mode of onset of the coma. We have seen that it is rare for a man attacked by cerebral hæmorrhage to fall down suddenly, deprived of sense and motion, and to remain persistently comatose. There are, indeed, cases in which apoplexy begins with a fit which is perfectly epileptiform in character; of this the writer saw a marked and repeated example, due undoubtedly to cerebral hæmorrhage; but such cases are exceptional. The practical rule, therefore, is that if we are informed that the commencement of a seizure was gradual, we may safely conclude that the case is not one of epilepsy; but if it began suddenly, we may often feel doubt as to its nature. In such a case our prognosis must be most cautiously guarded; a few hours later the patient may be apparently as well as ever, or he may be dying.

8. Several of the *organic diseases* of the brain may occasionally give rise to a rapidly fatal attack, resembling an apoplectic seizure in its symptoms. This is the case, for instance, with cerebral *abscesses* and *tumours*, and occasionally with tubercular *meningitis*. Sometimes, indeed, cerebral hæmorrhage does really occur as a complication of the softer forms of tumour, the blood-vessels within the growth giving way, and pouring out a large quantity of blood. It is not improbable that in many of the cases in which hæmorrhage has been found in the cerebellum, or in some other

unusual seat—the patient being perhaps young and the arteries healthy—the original disease has been a soft tumour of small size, which has been torn up by the extravasated blood and overlooked at the autopsy.

Diagnosis between the anatomical causes of apoplexy.—We now come to the differential diagnosis of the several lesions, which are each capable of causing apoplexy, aphasia, or hemiplegia; namely, hæmorrhage, embolism, and thrombosis; softening from syphilitic disease of the arteries may be separately considered.

The diagnosis of cerebral hæmorrhage from embolism and the allied lesions differs, according as the patient, immediately after a stroke, becomes unconscious (*apoplexy*) or retains his senses (*hemiplegia*).

(*a*) In cases of *apoplexy* we must be guided by such clinical cases as the following.

Cerebral embolism.—In 1868 a man, aged forty, was brought into the Clinical Ward with paralysis of the left side, having suddenly fallen down insensible while wheeling a barrow. He presently regained his senses and conversed about his symptoms; then he again became comatose, and remained so until he died a few hours afterwards. In 1869 a man was taken in for paralysis and partial anæsthesia of the left side; he was reported to have suffered from continuous headache for two or three weeks. A few days before his admission he had suddenly become giddy and lost the use of his left arm and leg; afterwards he was unconscious for two or three hours. In the same year a woman, who was in the hospital suffering from dropsy, had a fit which was followed by aphasia and right hemiplegia, and she died within two days. In 1874 a woman, who was in the ward for uterine cases under Dr Braxton Hicks, became comatose with left hemiplegia, and remained insensible until she died on the following day.

In none of these four instances was there any hæmorrhage into the brain, nor any change beyond softening of the part which should have received its supply of blood through the obstructed vessel. Yet, if no autopsies had been made, these very cases might well have been quoted as illustrating the chief varieties in the mode of onset of an apoplectic seizure due to the rupture of a cerebral artery. Nothnagel, indeed, states explicitly that there is no absolute difference between the symptoms of embolism and those of hæmorrhage; and there can be no question as to the inadequacy of certain points of distinction suggested by earlier writers—that in embolism the face is pale rather than red; and that cases of hæmorrhage are accompanied by excessive pulsation of the carotids, stertorous breathing, and inequality of the pupils.

Nothnagel quotes Dr Eliza Walker as having shown in her inaugural dissertation at Zürich in 1872 that embolism is very often ushered in by an epileptiform attack; this was the case in twenty-four out of ninety-seven cases which were collected by her. Nothnagel adds that embolism can never have any prodroma; but the statement is contradicted by one of the cases already cited, in which the attack was preceded by headache for two or three weeks.

Among the cases of spontaneous *thrombosis of a cerebral artery* at Guy's Hospital there have been the following:—A man, aged forty-eight, was attacked with pain in the head a month before his death; he fell back insensible, with right hemiplegia; afterwards he could walk, but he ultimately became again semi-conscious and rambling in his talk. Another

man, aged thirty-five, was attacked with unconsciousness and paralysis of the left side six days before his death; he then had convulsive fits, in one of which he died. Apoplexy due to this lesion cannot be distinguished from that caused by cerebral hæmorrhage.

As regards *syphilitic* disease of the cerebral arteries, the cases related by Heubner in his monograph suffice to show how closely its symptoms may resemble those of rupture of an artery in the brain. In eight cases out of twenty-two collected by him the symptoms were ushered in by an attack of apoplexy. In some rapidly fatal cases the insensibility lasted until death. In others there was a transitory loss of consciousness, accompanied by hemiplegia, and followed after an interval by the supervention of coma from which the patient never again awoke; in other words, the clinical features of an "ingravescent" nature were closely imitated. Case 33, for instance, is that of a man who had suffered from headache and sleeplessness, and who one day after having appeared to be as well as usual in the morning, was found later on to be comatose and paralysed on the right side: he could at first be roused a little, so as to make an attempt to open his eyes; but afterwards the insensibility became more profound, the face flushed, the pupils immovable, and he died the next evening. Case 47 is that of a journeyman furrier, admitted into the Leipzig Hospital in a state of unconsciousness. He had been attacked on the previous day with paralysis of the arm; this came on gradually, so that he watched its progress upwards from the thumb to the shoulder, being then in perfect possession of his senses, and able to point out with his left hand how high the numbness extended at any particular moment. There are, however, comparatively few syphilitic cases in which the symptoms and course resemble those of cerebral hæmorrhage.

Thus we find that an attack of apoplexy is by no means necessarily due to the extravasation of blood in the brain. Omitting spontaneous thrombosis on account of its rarity, we have embolism and syphilitic arthritis as possible causes of precisely similar symptoms.

The diagnosis is based entirely upon collateral circumstances. *Age* is an element of the highest importance. In persons more than fifty years old one is seldom wrong in attributing an apoplectic seizure to hæmorrhage; and the younger the patient the greater the probability that one of the other two causes is in operation. To decide between them we must look for indications of cardiac disease on the one hand, and, on the other, for eruptions on the skin or fauces, enlargements of the testicles or lymphatic glands and nodes. It must not be forgotten that cerebral hæmorrhage may be an indirect result of embolism, by the formation of an aneurysm in the obstructed artery (p. 555). In most cases of this kind the primary disease is ulcerative endocarditis, and the embola possess septic characters; so that enlarged and tender spleen, or albumen or blood in the urine, may suggest the correct diagnosis.

Notwithstanding the caution expressed above (p. 558), the *presence of albumen* when combined with a low specific gravity of the urine, a hard pulse and signs of an hypertrophied left ventricle are of great diagnostic value, for these symptoms of chronic interstitial nephritis much increase the probability of a ruptured artery. If we find evidence of *retinal hæmorrhage* by the ophthalmoscope we feel almost certain of our diagnosis. A *gradual onset* of coma, with severe previous symptoms of pain, giddiness, &c., for a day or two, points to occlusion of an artery (probably syphilitic in a young

adult), and consequent softening. A sudden and profound attack is probably due to hæmorrhage.

A shock, like syncope, with revival and then torpor, gradually deepening into coma coming on within an hour, points almost certainly to hæmorrhage, checked by failure of the heart, and returning as the circulation improves.

Pain in the head, with hemiplegia and subsequent coma, suggests cerebral embolism, and in a young subject with a cardiac murmur makes it practically certain.

(*b*) In cases of *hemiplegia* the diagnosis between its several causes depends first upon the question whether coma was present when the attack of paralysis occurred. If so, the considerations stated in the last paragraph are applicable; and in proportion to the duration and the severity of apoplectic symptoms is the probability that there was rupture of an artery. On the other hand, if the seizure was unattended with any, even transient, loss of intelligence, the presumption is strongly in favour of its being the result of a mere arrest of circulation in some part of the motor tract. This may depend upon embolism of a cerebral artery, or upon syphilitic endarteritis. Arteritis deformans often leads to the formation of patches of white softening in the substance of the brain. One would have expected that a point of considerable diagnostic importance would have been the insidious and gradual commencement of hemiplegia due to arteritis. But experience scarcely bears this out; the attack is generally sudden. It is probable that the nervous elements continue to perform their functions up to a certain point, notwithstanding impairment of their structure; and that several of them at length suddenly and simultaneously give way, just as a rope will sustain a weight until the moment before the last strand breaks.

The discrimination of the various lesions of the brain that may give rise to hemiplegia depends on a careful study of the other nervous symptoms. We have already seen the diagnostic value of the association of *aphasia* with hemiplegia, as showing that the Sylvian artery is obstructed, whether by embolism, thrombosis, or syphilitic thickening.

It is probable that persistent *hemianæsthesia*, when associated with hemiplegia, points to hæmorrhage rather than to arterial obstruction. For such a combination of symptoms shows that the posterior third of the internal capsule is involved; and this lies towards the periphery of the area of distribution of the Sylvian artery (p. 553), so that it might probably be fed from another source if arrest of the blood supply were the cause of the hemiplegia. However this may be, most of the cases hitherto recorded, in which loss of sensation has been permanent, have occurred in old people, so that rupture of an artery was probably the cause of the symptoms. In the case referred to above (p. 564) the patient was seventy-one at the time of the first seizure. Among fifteen fatal cases collected by Veyssière in 1874 there is only one in which the patient was young, that of a girl, aged twenty-two years; in several instances, indeed, the lesion is described as having been a patch of *ramollissement;* but no details are given to throw light upon the cause of the softening.

On the other hand, *syphilis* is apt to affect several of the large cerebral arteries, simultaneously or in succession. It is therefore not surprising that hemiplegia dependent upon this cause is sometimes accompanied by a series of *incongruous* or *irregular* symptoms, which cannot be referred to a lesion

limited to any one spot in the brain. Thus there is sometimes complete paralysis of one or more of the cranial nerves, or the patient may regain the use of the arm and leg which were first affected; and subsequently he may be attacked with loss of power on the opposite side. This, indeed, is not in itself characteristic of the syphilitic affection; for old people with atheromatous vessels are very liable to have patches of softening develop themselves in the two hemispheres in succession, or to suffer from the effects of small hæmorrhages into both corpora striata in turn. But symptoms due to syphilis are more apt to be irregular in their locality and course than those which depend upon other causes; the patient perhaps becomes able to stand, or even to walk, and then after a few days relapses; and such changes may occur again and again.

Heubner lays stress on a peculiar *somnolent* condition, as indicating the presence of syphilis: the patient, he says, is half awake, half asleep; he lies with his eyes shut, taking no notice of anything, and refusing to answer questions; but when one tries to examine him, he resists and turns round in his bed. That such symptoms are comparatively rare in cases of embolism or of cerebral hæmorrhage is true; but they are common enough when the disease is a *tumour* or some other local lesion, and one must always remember that these affections may also give rise to hemiplegia, if they are so seated as to involve the motor tract.

There is, in fact, no one of all the organic changes to which the brain is liable that may not sometimes induce loss of power in the arm and leg on one side; beside *epilepsy*, *hysteria*, *chorea*, and some other neuroses which will be described in their place.

Conversely, it occasionally happens that affections of the cerebral arteries may run their course to a fatal issue without hemiplegia appearing. Such cases may be attended with headache, loss of memory, drowsiness, delirium, vomiting, thickness of speech, dysphagia, involuntary evacuations, and other symptoms, but none of them are characteristic.

Prognosis.—The gravity of a case of Apoplexy may be judged of first by the depth of the coma; the ingravescent cases are the worst, while gradual recovery of consciousness is of good omen. In cases comatose from the first, death frequently follows on the third day, but it may occur after a week or even longer; and, on the other hand, recovery may follow prolonged unconsciousness if the pulse and breathing are not seriously affected, and if the patient is not fed. Rise of temperature is a bad sign. So are early rigidity, convulsions of the paralysed limbs, flapping of the cheek in breathing, insensitive conjunctivæ, and increasing cyanosis. The longer coma persists the less hope there is. When consciousness is once restored, life is generally preserved for that attack at least.

Treatment.—(*a*) That the treatment of Apoplexy is unsatisfactory is universally admitted. There is no disease in which it is more difficult to estimate the effect of therapeutical measures of whatever kind.*

Rest is always essential; the patient should, if possible, be left in the room in which the seizure occurred; a mattress placed on the floor does perfectly well for a time. His head and shoulders must be raised, and he should be turned over on his side, so as to prevent the tongue from falling

* So Hippocrates: "To cure apoplexy when severe is impossible, and not easy when it is slight" ('Aphor.,' ii, 41).

backwards against the pharynx. In cases in which it is clear that death is impending one ought to abstain from all active treatment. No good can be done by the application of a blister to the neck, or of mustard plasters to the calves. A patient whose coma is so deep as to threaten his life cannot be roused by such means; and if he is capable of feeling, the irritation must surely be injurious rather than beneficial.

When the case appears not to be altogether hopeless, five or ten grains of calomel or two drops of croton oil should be placed on the tongue, particularly if there is reason to believe that there is an accumulation in the bowels; or an enema of turpentine may be given. One must be on the watch for the necessity of passing the catheter if the insensibility should last more than a few hours. Whenever there is serious difficulty of swallowing, the administration of food or drink by the mouth should be altogether forbidden, on account of the danger of its running into the air-passages and setting up pneumonia. No harm results from keeping an apoplectic patient for a day or two without nourishment; but if it is thought desirable, enemata of beef-tea or milk may be employed. The lips and mouth may be kept moist with a feather.

Bloodletting, whether by venæsection, cupping, or leeches, is now seldom practised. Sir Thomas Watson, indeed, speaks of patients so insensible as not to feel the puncture made by the lancet, who have yet emerged from their coma while the blood was flowing. But it may be fairly doubted whether in such cases the disease was not that "simple apoplexy" which is probably a form of epilepsy, and in which a spontaneous recovery of consciousness often occurs with unexpected rapidity. If the pulse be large and labouring, the face flushed, the carotid arteries full and throbbing, it may be thought right to bleed. But the majority of patients suffering from cerebral hæmorrhage are advanced in years and impaired in health, so that one hesitates to employ a practice which can be of service in lowering the pressure within the vessels of the brain only by its general effect on the circulation. In a younger person the coma may be due to embolism or to syphilitic arterial disease, when the maintenance of a vigorous cerebral circulation is essential to recovery.

There is, however, one particular set of cases in which it seems probable that judicious management may sometimes prevent a fatal issue; namely, those in which the symptoms of cerebral hæmorrhage are slowly ingravescent. When one is called to such a patient in the early stage (that of collapse), one should keep him absolutely recumbent, with head and shoulders raised, and he should be allowed neither to speak nor to move. The limbs may be warmed by friction with hot flannels, but the administration of brandy, and even of ammonia, must be rigidly abstained from. As reaction comes on, the question of bloodletting must be most seriously weighed. It is true that a rapid death almost always occurs, and that the autopsy generally confirms the opinion that the case was hopeless from the first. But it is possible that free venæsection, just at the time when the vigour of the circulation is being re-established, may, by lowering the pressure in the cerebral vessels, prevent further effusion of blood.

Hæmostatics, such as ergot, acetate of lead, or gallic acid, are not often prescribed; but Dr Bristowe assents to their employment, and one is certainly encouraged to try them by their apparent success in cases of hæmoptysis. Cooling lotions, or a bag of ice, may be applied to the head, if they do not disturb the patient.

When coma is due to Embolism, the only treatment is to keep the patient in a state of the most perfect quietude, so as to reduce to a minimum the activity of the nervous centres, deprived of their blood supply.

In cases of obstruction from Syphilitic arteritis, inunction of mercury should be commenced with the first cerebral symptoms.

(*b*) A person attacked with Hemiplegia without unconsciousness should for some days be kept in bed, and as free from all cares as possible. If advanced in years, or suffering under heart disease, he should be supplied with soup, beef-tea, milk, and perhaps a little wine.

A syphilitic patient should at first have rather a scanty diet while the proper treatment is being carried out: say a twelfth of a grain of bichloride of mercury, with ten or twenty grains of iodide of potassium.*

But in too many cases of Hemiplegia—whatever its cause may have been—the limbs remain more or less completely paralysed, in spite of the most judicious treatment. Recourse is then had to electricity, which undoubtedly is sometimes beneficial. The continuous application of a weak galvanic current to the head, as suggested by Remak, is said by Nothnagel to be occasionally followed by a decided increase of power and diminution of rigidity in the affected muscles. Not more than from four to twelve cells should be brought into operation; the poles should be gradually applied and withdrawn, and should never be suddenly reversed; they should be kept in position for not longer than three minutes at a time; whether they are both placed behind the ears, or one upon one mastoid process and the other upon the forehead, seems to be a matter of indifferenee. Other methods are to galvanise or to faradise the muscles. Dr Reynolds lays down the rule that little or no good can be thus effected if the muscles contract to the normal extent. But if the contractility is diminished, electricity will often in the course of a few weeks restore it; at the same time the muscles will become less wasted, and the previously cold limb will regain its normal temperature. By faradising the extensor muscles of the fingers, one may diminish contraction of the fingers into the palm. In general it may be stated that the application of electricity should not begin till two months from the hemiplegic seizure; that it should be at once left off if it causes headache, giddiness, faintness, sickness, or any unpleasant feeling at the epigastrium; and that the current should never be so strong as to cause pain, nor so long continued as to cause fatigue.

The disease cannot be influenced by drugs, though occasionally laxatives and sometimes hypnotics are required. Nor can one reasonably expect any benefit to result from baths at Gastein, Pfeffers, or anywhere else. To prevent a recurrence of the attack, temperance both in eating and drinking, a loose state of the bowels, and the avoidance of emotional and other excitement, both of mind and body, are the rational and probably the only measures to be advised.

* Heubner relates some remarkable examples of recovery of power in the paralysed limbs under the method of inunction. A striking case was that of a student, aged twenty-six, who after having suffered from headache and giddiness for some weeks was attacked one night with left hemiplegia during sleep. There were indurated glands in the neck and at each elbow, and a scar on the velum palati. At the end of a fortnight he was no better—feverish, prostrate, sleepless, and delirious; bedsores were forming, and spasmodic movements of the right arm had begun. Mercurial ointment was rubbed in for seven days, and then iodide of potassium was given in large doses. Six weeks later he was able to leave his bed, and little by little he regained power in his limbs. Several years afterwards Heubner found him perfectly well, except that he walked with a stick.

TUMOURS AND ABSCESS OF THE BRAIN

"The Eye is the Index of the Brain."

CEREBRAL TUMOURS—*Tubercle—Syphilitic gumma—Meningeal growths—Glioma and other neoplasms—Symptoms: headache and other cephalic symptoms—Vomiting—Temperature, pulse, and respiration—Optic neuritis—Localising symptoms—for the base—the cerebellum—the motor area and other parts—Diagnosis from hysteria, meningitis, &c.—Diagnosis between the several kinds of tumour—Prognosis—Treatment.*

CEREBRAL ABSCESS—*Ætiology—Anatomy—Symptoms: general and localising—Diagnosis—Course and event—Treatment.*

Red softening—Histology—Causes—seldom or never primary.

Inflammation of the brain.—In discussing in the last chapter the symptoms which often enable us to determine the exact seat of the lesions which cause hemiplegia and aphasia, we entered upon one of the most interesting parts of the study of cerebral diseases; but the study was incomplete, because lesions of the arteries rarely occur except in certain regions of the encephalon. There is, however, another important group of local organic affections which are under no such anatomical limitation, and it will be in every way convenient to take them next, so as to place the reader in possession of the chief points that must guide him in what is termed the *localisation* of diseases of the brain. The group of diseases which occupies this chapter is not a pathological one; for it includes syphilitic gummata, tubercle, new growths, and inflammatory changes leading to softening and abscess. They only agree in being sources of increasing local pressure within the skull; but this is clinically all-important, for they produce similar effects; and at the bedside one can often distinguish these several affections only imperfectly upon indirect or collateral evidence. We will begin with a brief account of the lesions which may be included among new growths, taking first *Tubercle*, secondly *Syphilitic Gumma*, and thirdly *Tumour*. Afterwards we will discuss their symptoms in common, their differential diagnosis, and their treatment. Lastly, *Red Softening* and *Abscess* will be described separately.

1. *Tubercle of the brain.*—The tubercular process may affect the brain in two different ways. Sometimes a number of minute tubercles grow into it from the pia mater, or are scattered through its substance along the vessels. This condition is always associated with meningitis, and will be described in the next chapter. In other cases there is a single caseating mass, which may reach a great size. Such masses have been called "solitary tubercles," "tuberculous tumours," and "tubercular nodules." In size they generally vary from that of a pea to that of a walnut; one of the biggest is a specimen in the museum of Guy's Hospital, which was received from Dr Hughlings Jackson by Dr Moxon. Sometimes they have a flattened base towards the pia mater, but more commonly they are more or less

globular in form, and touch the surface only at one spot, or are completely surrounded by brain-substance. They do not adhere to the dura mater lining the skull, but sometimes may to the tentorium when they occupy the cerebellum. Their substance is of a bright yellow colour, firm, or even hard, opaque, and generally homogeneous, except that at the centre it may be softening down into a yellowish liquid, or may be partially calcified. But they also have, in most cases, a narrow, pinkish-grey, soft growing edge, which separates the yellow cheesy material from the brain-tissue around. This edge may obviously consist of an aggregation of miliary tubercles, but more often it has no such appearance. Microscopically it consists of corpuscles, which are sometimes larger than the lymphoid cells that characterise tubercles in other parts. Rindfleisch further insists on the fibrous texture of the marginal zone of the cheesy mass, and declares that many so-called cerebral tubercles are really non-tuberculous, deserving rather to be called fibroid tumours. But a similar transformation of the cellular elements of tubercles is well known to occur even in the lungs, so that there can be no doubt that these solitary yellow tumours of the brain are truly tuberculous.

Among thirty-two cases that have occurred at Guy's Hospital there were only two in which it is stated that no similar lesion could be discovered in any other part of the body. It is true that tubercular meningitis (which was found in several instances) would be regarded rather as an accidental result of infection from the caseous mass than as independent; but in a large number of instances there was chronic phthisis or disease of the mesenteric or mediastinal glands. In no less than eighteen of the thirty-two cases the cerebellum was the sole seat of the affection; three times it was the pons Varolii, once the medulla oblongata, six times one of the hemispheres: in the other four cases there were several tubercles in different parts of the brain.

Twenty-one of the patients were males, the remainder females, a proportion which accords with that given by other writers. In three instances the age was under five years, in six between six and ten years, in twelve between eleven and twenty years, in seven between twenty-one and thirty, in three between thirty-one and forty, and in one forty-two.

In one case, that of a child, aged four and a half, there is said to have been a fall upon the back of the head five or six weeks before the occurrence of a fit, which was the earliest sign of cerebral mischief; the seat of the tubercle was the pons. Dr Crichton Browne mentions ('West Riding Asylum Rep.,' ii) a similar instance; the same part of the head was struck, symptoms speedily showed themselves, and proved fatal in two months, and a mass of tubercles, the size of a walnut, was found in the cerebellum. If it should be thought that in the latter case there was hardly time enough for the development of such a lesion, a suggestion made by Rilliet and Barthez may apply, namely, that when an injury seems to have been the cause of a tubercle in the brain, it may, perhaps, have merely set up acute changes around a tubercle which was there before.

2. *Syphilitic gumma of the brain.*—This affection also is attended with the formation of a firm, dry, yellow, caseous material, which is not always easily distinguished from tubercle. One point of difference is the fact, long ago pointed out by Wilks, that it is constantly seated on the surface of the organ, growing into the cerebral substance from the membranes. Heubner states that an analysis of forty-five cases, recorded by different observers, yielded only three exceptions to this rule, and not one of these was conclu-

sive. Again, when a gumma occupies the convexity of the brain, or the summit of one of the cerebral lobes, the corresponding part of the dura mater becomes thickened and converted into a tough yellow layer and adherent, so as to form with it one inseparable mass. Thus the shape of a gumma is far more irregular than that of a tubercle, which we have seen to be more or less globular. Lastly, the syphilitic affection commonly shows a much more considerable proportion of translucent undegenerated tissue; this appears as a moist greyish-red or grey mass, which is sometimes as soft as jelly; it is especially apt to be abundant at the base, where it may involve several of the cranial nerves and fill up the sella turcica as well as the diamond-shaped space. Microscopically it consists of granulation-tissue, containing some spindle-shaped and stellate elements, and having (according to Heubner) a distinctly alveolar structure in many instances. It is very vascular, and sometimes presents many little extravasations of blood. On the other hand, if antisyphilitic remedies should have been freely administered during life, the only morbid change found after death may be a patch of superficial softening of the cortex, beneath a local adhesion of the dura mater to the brain; this was the case, for example, in a patient of Dr Dreschfeld ('Lancet,' 1877).

It is curious that of ten cases of gumma of the brain at Guy's Hospital all but one were in persons between the ages of thirty-one and forty, the exception being a patient of twenty-six. In five of them there was a definite history of constitutional syphilis, or else indisputable syphilitic lesions were found; only one patient distinctly stated during life that he had never had venereal disease, and in that case the liver contained gummata. As might be expected, males preponderated over females in the proportion of seven to three. In several instances the report of the autopsy is incomplete, an examination of the testes having often been omitted. In one case the patient's illness had been attributed during life to a sunstroke. This was, no doubt, a mistake, but many writers think that a blow or fall upon the head may determine the formation of a gumma in the brain. "Sunstroke" practically often turns out to be cerebral syphilis.

3. *Other tumours of the brain.*—There is no region of the body in which so many different kinds of new growth are found as in the brain and in its membranes; yet histological distinctions are nowhere else of so little practical importance, because the clinical history and symptoms of a case are seldom, and then but slightly, affected by the nature of the tumour. A brief description of the chief varieties will therefore suffice.

a. The *membranes* may present fibrous and some other simple tumours, such as occur almost anywhere, and malignant tumours of all sorts, primary or secondary. A favourite seat for malignant growths is between the dura mater and the bone; or perhaps it would be more correct to say that they begin within the osseous substance itself, and either push inwards the dura mater or penetrate it, protruding upon its inner surface, and invading the cerebral tissue. They generally are *sarcomata.*

Two or three kinds of innocent tumour are peculiar to the coverings of the brain. To one of them Virchow has given the name of *psammoma*, from its containing calcified particles, like those which constitute the brain-sand of the pineal body and choroid plexuses. The lime-salts are deposited in curious little globular structures, made up of elements arranged concentrically, so as to resemble somewhat the well-known "bird's-nest cells." Robin accordingly described this affection as a form of epithelioma; but such a view of

its nature is incorrect. Virchow says it generally appears as a semi-globular mass, of firm consistence, pale red or white, and medulla-like. One examined by the author had a loose texture and a flocculent surface; it grew in such a position as to indent the brain above the left Sylvian fissure. According to Virchow such growths are less frequently seated in the tentorium or in the falx than in the dura mater lining the skull—particularly at the fore-part of the base.

Another special form of tumour peculiar to the membranes is that which was formerly called a *cholesteatoma*, but which has of late been termed "pearl-cancer." It constitutes a dry, hard, rounded mass, occupying the pia mater generally at the base of the brain. Its cut surface has a pearly lustre, and it consists of lobules made up entirely of horny epithelial cells, and supported by a stroma of dense connective tissue.

Lastly, Virchow has described under the name of *melanoma* a fourth kind of meningeal new growth; in a case which came under his observation there were numerous black or brown nodules in the pia mater, the rest of the body being entirely free from them.

β. Of the tumours that have their seat *within the brain-substance* there is one kind that is almost peculiar to it, while the rest are like those found in other parts of the body.

Among the latter, *sarcomata* are the most numerous, especially those of the spindle-cell variety. Sometimes, though very rarely, a myxoma or fibroma is met with. Primary carcinoma of the brain is so rare that only one case in our records at Guy's Hospital is said to have been of that nature. Even secondary cancer is not common; in the majority of cases in which secondary nodules are developed within the cranium, the growth is a form of sarcoma. A peculiarity of secondary tumours in the brain is that they are almost always multiple. On the other hand, it is a rule to which there are scarcely any exceptions, that a primary new growth is solitary. Among forty-four cases collected from our records, in only two was there more than one tumour in the brain, without obvious source of infection elsewhere; and it is quite possible that in those cases there was primary disease of one of the bones, or of some other structure which was overlooked.

The centre of a cerebral tumour now and then softens down into a cyst, and this may become so large that the presence of any solid growth may not be obvious. We have at Guy's Hospital had five cases in which the cerebellum has contained a large thin-walled cavity, filled with a fluid that was highly albuminous or deposited spontaneously a fibrinous coagulum. In three instances no adventitious tissue could be discovered, except the vascular membrane forming the cyst itself; but in each of the other two there was a small tumour situated on one side of the fibrous wall. These cavities are more rare in the cerebrum than in the cerebellum ('Path. Trans.,' 1885, vol. xxxvi, p. 17).

Hydatids are of very rare occurrence in the brain. In a girl, aged nine, Dr Moxon found a large *echinococcus* occupying the middle and posterior lobes of the right hemisphere; she had also a similar affection of the liver. The *Cysticercus cellulosæ* sometimes infects the pia mater or the ventricles.

The one kind of tumour already alluded to as almost special to the brain* is that which Virchow termed *glioma*, because he regarded it as an overgrowth of the neuroglia, or interstitial connective tissue of the brain, which he dis-

* A similar affection sometimes occurs in the retina during childhood, and perhaps also in the suprarenal capsule.

covered to consist of a granular or faintly fibrillated matrix, in which are embedded small nuclei with scanty protoplasm. Such a growth is not very unlike a sarcoma; and many pathologists describe it as a mere variety of that form of tumour. Indeed, Virchow himself admitted that it was often impossible to say of a particular specimen whether it should or should not be called a sarcoma; and he expressly recognised the occurrence of intermediate forms, which he termed glio-sarcomata. But although the distinction may not be of great practical importance, it appears to rest upon a sound theoretical basis, inasmuch as the neuroglia is a completely developed or adult structure, whereas the elements of a sarcoma are embryonic, corresponding with those of connective tissue at an early stage of its growth.*

A glioma may be of a pinkish-red colour, or it may look so exactly like the normal brain-substance that the microscope is required to demonstrate its presence. Its substance is always continuous with that of the surrounding cerebral tissue; for there is never a capsule, as with some sarcomata. Indeed, it often assumes the form of the part in which it grows, so that one might imagine the corpus striatum or the thalamus, or some particular convolution, to have become swollen to three or four times its natural size. But in some cases extensive caseation takes place in these growths; and, according to Klebs, they are also liable to sclerosis, exactly like that which occurs in the normal brain-substance.

A soft glioma often contains a great number of thin-walled vessels, which may rupture and pour out blood into its tissue, tearing it up so that one can hardly discover any trace of the growth. As Virchow long ago pointed out, the disease may then be mistaken for a simple cerebral hæmorrhage.

Perhaps the most puzzling cases of all are those in which repeated extravasations occur, and in which the coagula become converted into tough opaque caseating masses of various colours.

Not only gliomata, but all the less circumscribed forms of cerebral tumour, are apt to set up in the adjacent brain-tissue morbid changes that can only be regarded as inflammatory, and as due to irritation caused by their presence. Such an affection sometimes assumes the form of "red softening." More often it is what Rokitansky first described as "yellow softening"—a state in which the medullary substance has a faint yellow tinge, and looks glistening like blancmange; but nevertheless retains its form when sliced or cut, and is not, in fact, softer than the rest of the brain. The microscope throws no light upon its nature, but it appears to be œdema. Another common result of the presence of a new growth in the brain is the accumulation of an increased amount of fluid in the ventricles, which may become greatly enlarged. It might sometimes be attributed directly to compression of the veins of Galen or of the choroid veins, as when the tumour is seated in the cerebellum or in one of the posterior lobes of the cerebrum. But Virchow has pointed out that hydrocephalus often occurs where this explanation is inapplicable; and his statement is confirmed by several cases in our records.

Statistics of cerebral tumours.—To judge from a series of cases that have

* Prof. Klebs, now of Zurich, maintains ('Prager Vierteljahrschrift,' 1877) that gliomata also contain newly formed nervous elements. Certain cells with many-branched processes, which are well known to occur in these tumours, are regarded by him as ganglionic cells; and he believes that they are sometimes developed out of the axis-cylinders of nerve-fibres.

occurred at Guy's Hospital, there is a remarkable difference in the liability of the several parts of the brain to be the seat of tumours at different ages. We have had twenty-two cases in which there was a primary new growth in one of the *hemispheres*, sixteen on the right side and six on the left. In only five of these was the patient less than thirty years old; the rest were pretty evenly distributed between the ages of thirty and sixty. On the other hand, out of fourteen cases in which a tumour was seated at the *base* of the brain, ten occurred in persons under the age of thirty. The six instances of cysts in the *cerebellum* were all in patients between twenty-one and twenty-seven years old. There were only two cases in which solid tumours were found in children under the age of ten; in each instance the seat of the affection was the cerebellum.

Tumour of the brain is more frequent in males than in females. Among forty-two cases at Guy's Hospital, in which the sex is recorded, the proportion is as 27 to 15. This corresponds very closely with the ratio of 10:6 given by Obernier; but it is interesting that in the cases in which one of the hemispheres was the seat of the affection the preponderance of males is much higher, namely, as 16 to 6; for men are much more liable than women to blows and falls; and Virchow has expressed the opinion that new growths affecting the upper parts of the brain are often caused by injuries to the head. He has further stated that tumours in this position are generally gliomata, whereas at the base sarcomata and carcinomata are more often found.

In the forty-third volume of the 'Guy's Hospital Reports' Dr Hale White collected 100 unpublished cases of cerebral tumour, which were found in the deadhouse from 1872 to 1884 inclusive. The new growth was tubercular in nearly half of these cases (45), and it was a glioma in 24. Two of the remaining cases were glio-sarcoma, 10 sarcoma, 5 carcinoma, 5 gumma, 1 myxoma, and 1 lymphoma; while 4 were cystic.

Of the tubercular cases, 34 occurred in male and 11 in female patients; more than half of these 45 patients were children under ten years old. The hemispheres were the seat of a tubercular tumour in 22 cases, the cerebellum nearly as often (20), the pons in 6 cases, the bulb and the crus cerebri in one each. Nineteen of the tubercular tumours were multiple.

Glioma was widely distributed among patients of different ages, from three years to sixty-two. Here also the male sex predominated in the proportion of 19 to 5, and all the patients under twelve were boys. The seat was the hemispheres in 13 cases, the cerebellum in 4, the pons in 2, the bulb in 1, the pituitarium in 3, and the dura mater in one only.

Of the four cysts, two were situated in the cerebellum. One of the cerebral cysts was a hydatid.

Considering locality only, 48 tumours of all kinds were in the hemispheres, 28 in the cerebellum, 10 in the pons, 7 grew from the meninges, 5 were in the basal ganglia, and 3 in the pituitarium.

Symptoms.—In proceeding to discuss the clinical symptoms of these various forms of cerebral tumour, we must first remark that in making *post-mortem* examinations one occasionally finds a tubercle or a tumour in cases in which there had during life been no suspicion of the presence of any cerebral lesion. Thus we have in the museum of Guy's Hospital a specimen of a very hard growth, nearly as large as a pigeon's egg, attached by a pedicle to the ridge of the petrous bone; it was discovered accidentally in

the body of an aged pauper woman who was said to have had no cerebral symptoms. Another preparation is one of a large carcinomatous mass in the cerebellum, taken by Dr Moxon from a patient who died of cancer of the breast and of the liver. But it may be said that, if she had lived a little longer, the tumour might have manifested signs of its presence; and the same remark applies to the very few instances in which one or more small tubercles have been unexpectedly found in the brain in persons who have fallen victims to phthisis or to some other scrofulous disease. All that is really proved by such cases is that lesions of various kinds may be latent during the early periods of their development. But of this fact there is abundant evidence of another kind. Thus when tubercular meningitis has been the cause of death, and when the patient had appeared to be perfectly well up to the time of the commencement of that disease, one not uncommonly finds one or more yellow tubercles in the brain-substance which must obviously have been of older date. Again, it sometimes happens that a person dies of what appears to be an acute illness, and that the only lesion found at the autopsy is a tumour, with softening of the surrounding cerebral tissue. Some years ago a man was admitted into hospital insensible, livid, and in a high state of fever, so that the diagnosis was typhus; he lived only three days from the time when he was first taken ill, but at the autopsy a small spindle-cell growth, of the size of a bean, was found projecting into the fourth ventricle from the side of the aqueduct. But, with the exception of one or two instances in which psammomata or other meningeal tumours have been quite unexpectedly discovered, our records do not appear to contain an instance in which a tubercle or a tumour has been met with in the brain of a patient killed by accident, or who died from some indifferent disease, such as heart disease, or ileus.

The symptoms may be divided into two groups. Some of them are common to the large majority of cases; others are comparatively seldom observed, their presence being dependent upon the seat of the lesion. It will be convenient to describe the former group of symptoms first; and they may be arranged under three heads:—(1) cerebral symptoms proper, such as headache, giddiness, epileptiform seizures, hemiplegia, loss of memory, mania, stupor; (2) disturbances of distant parts, including vomiting; (3) certain changes in the optic disc, which are revealed by the ophthalmoscope.

1. *Head-symptoms.—Pain* is very rarely absent, and is generally the earliest indication that anything is wrong with the patient. Its seat sometimes, but not always, answers to that of the lesion. The superficial nerves of the corresponding part of the scalp may be tender to pressure or percussion; or (as Romberg first noticed) the act of holding the breath or of coughing may increase the pain—no doubt by producing venous congestion. The museum of Guy's Hospital contains a large tumour, three inches in diameter, found many years ago in the left hemisphere of a girl, a patient of Dr Wilks, who had suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken by Mr Day, of St. Neots, from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains in it, and a sense of coldness in one spot, not larger than a shilling; this corresponded very nearly with the seat of the tumour that was found after her death. The pain caused by a new growth may be of every degree of intensity, from a

dull headache to the most unbearable agony, such as is said to render a patient delirious. It is sometimes constant, but it generally undergoes exacerbation from time to time. In some cases, indeed, it is intermittent or paroxysmal, so that it may closely resemble ordinary migraine. Thus Abercrombie relates the case of a boy, aged six, who began to suffer from fits of severe sick-headache, recurring at first about once a fortnight, and leaving him in good health in the intervals. After five or six months the attacks assumed a different and more persistent character; and two months later he died, when a tuberculous mass was found in the cerebellum. A similar instance has been recorded by Lebert.*

Convulsions are frequently present, and sometimes most severe and frequent. It is not known whether growths situated in any particular region are more apt than others to be accompanied by general epileptiform fits, as distinguished from those seizures to be presently described as "Jacksonian epilepsy" which are limited to the muscles of certain parts, and unattended by loss of consciousness. Dr Reynolds was led by the examination of a large number of cases to the conclusion that convulsions were less common when the disease affected the anterior lobes than when it occupied the posterior lobes or the cerebellum.

Vertigo, varying from a slight feeling of giddiness on standing up, to reeling and inability to walk or stand upright, is an important symptom of a cerebral tumour.

Mental symptoms—including irritability of temper, depression of spirits, loss of memory, a general impairment of intelligence—may be observed even when a tumour lies in the membranes at the base of the brain. Thus we have in Guy's museum a specimen of a cholesteatoma, as large as a pigeon's egg, which compressed the under surface of the pons and cerebellum, and had also insinuated itself into the Sylvian fissure to some extent; the patient was at one time in the hospital with paralytic symptoms, but he became maniacal, had delusions, and was unmanageable, so that he was removed to Colney Hatch Asylum, where he died. On the other hand, the mental faculties are not infrequently retained nearly to the last when one of the hemispheres is the seat of the disease.

Friedreich and Obernier speak of excessive *sleepiness* as a principal symptom in two cases which severally came under their observation. The most usual termination of cases of tumour and cerebral abscess, as of the vascular lesions described in the last chapter, is by stupor, which gradually passes into *coma*. Sometimes the patient lies for weeks without taking the slightest notice, passing all his evacuations under him, and showing no sign of intelligence, except that he slowly swallows the food that is put into his mouth. Some years ago Sir William Gull had under his care a boy

* These cases seem to have an important bearing upon the general theory of the production of "cerebral symptoms" by local affections of the brain. I shall hereafter have to describe a group of functional disorders of the nervous centres, including migraine, vertigo, epilepsy, and some others—under the name of "nerve-storms" or "explosive neuroses." And I shall endeavour to show that each of these is liable to be set up by a variety of exciting causes. Now, my hypothesis is that a tumour or a tubercle causes a transitory vertigo, or an epileptiform fit, or an attack of sick-headache in exactly the same way as any other disturbing agent. I conceive that the nerve-storm so produced has precisely the same seat as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If this be granted, it ought to follow that pain in the forehead should point less directly to the anterior part of the brain as the seat of a tumour than occipital pain to the cerebellum or posterior lobes.—C. H. F.

who manifested a remarkable retardation of intelligence. If a question was put to him he seemed not to heed it; but after many seconds, when the questioner had passed on to talk to someone else, he would deliver, word by word, a reply which showed that he perfectly understood what had been said. In other cases the mental condition undergoes changes which may perhaps depend on varying degrees of pressure by ventricular effusion. After lying in a stupor for two or three weeks the patient may recover his senses, remain conscious for several days, and then relapse into his former state.

2. *Vomiting* is a frequent and most important symptom—commonest perhaps with cerebellar tumours. The bowels are usually constipated as in other chronic cerebral diseases, and the abdomen is often hollow, the walls retracted, and the intestines empty. As a rule, there is loss of flesh, and extreme emaciation sometimes occurs. But a boy of fourteen, who was in Guy's Hospital in 1867, became remarkably fat during his illness, and remained so until he died; and two or three years previously the body of a young woman in a similar condition was brought down into the *post-mortem* room. In each case there was a tumour at the base, growing upwards into the third ventricle.

The *temperature* of the body is often one or two degrees below normal; but before death fever may develop itself. Obernier speaks of a rapidly advancing case in which every exacerbation of the headache was associated with a marked fall in the rate of the *pulse*. The mode of death is often by cessation of *breathing*, the heart continuing to beat for some little time; in one instance it went on for thirty-five minutes, while artificial respiration was vigorously kept up. In such cases it is wonderful how quietly life departs, without a gasp or a moan, or the movement of a limb.*

3. *Changes in the optic disc and retina.*—In 1860 von Graefe communicated to the German 'Archiv für Ophthalmologie' a short paper, in which he stated that inflammation of the optic nerves within the eyes sometimes occurred as a complication of cerebral diseases. It was already known that *blindness* was a frequent effect of tumours of the brain, even when their seat was not such as to involve any of those parts which were supposed to be functionally related to the sense of vision, but the belief had been that paralysis of the nerves of sight was the cause of the amaurosis, and that if any anatomical change was to be found it would be a simple atrophy from disuse. Thus von Graefe confesses the surprise that he felt when he first discovered (about the year 1857) that the optic discs were swollen and reddened in a case of this kind. His observations were soon confirmed by others, and after a while the remarkable fact was elicited that precisely similar appearances could often be detected when the patient was not conscious of any visual defect. Thus the ophthalmoscope has acquired great value in the diagnosis of diseases of the brain.

It was suggested by von Graefe himself, in his earliest communication, that there were two separate ways in which changes in the discs could arise, and that these were characterised by different appearances. On the one

* Some years ago I was called to see a female patient, whose symptoms pointed to the presence of a cerebral tumour, with Dr. Blades, of Kennington. On my arrival at the house he came out of her room to speak to me, leaving her husband and two women with her. When we went to her bedside a few minutes later we found her dead, except that a slight flickering of the pulse was still perceptible. Not the slightest change had been observed.—C. H. F.

hand, an increase of intra-cranial pressure, acting upon the cavernous sinuses, might mechanically obstruct the return of blood through the retinal veins; on the other hand, an inflammatory process at the base of the brain might be propagated as a "neuritis descendens" along the optic nerves to their terminations within the eyeball. Subsequent writers have to some extent modified the interpretation of these two conditions, and it has been shown that they very often co-exist. The one is known to the Germans as the "Stauungs-papille;" following Dr Allbutt, English writers term it the "Choked Disc," or (with less propriety) "Ischæmia of the Disc." The other is called "Optic Neuritis," "Papillitis," or "Neuro-retinitis descendens." There is also a third change, "Atrophy of the Disc," which may either arise independently or be consecutive to neuritis.

The choked disc.—There is no better description of this affection than that which von Graefe gave in his account of the first case that came under his observation. "The papilla," he says, "was greatly and irregularly swollen, rising steeply on one side and falling gradually on the opposite side to the level of the retina. . . . . Instead of being transparent its tissue looked grey and opaque, with an extremely deep reddish tint, and the adjacent part of the retina had the same appearance, so that the choroidal margin was completely hidden. The opacity was diffused, except that with the direct method of examination one could perceive a striated appearance following the course of the fibres of the optic nerve. The retinal veins were dilated, exceedingly tortuous, and obscured here and there by dipping into the opaque tissue; the arteries were comparatively small. The turbidity of the retina diminished gradually from the disc outwards over a zone of rather more than 2 mm. in breadth, so that it occupied an area 5 mm. in diameter (including the disc itself)." In a second case there were ecchymoses in the part of the retina near the disc.

The prominence formed by the optic disc in a case of this kind appears to be easily recognised after death when the eye is removed and laid open; an excellent illustration of it is given by Dr Allbutt. In one instance in which an intercurrent attack of smallpox destroyed life at an early stage of the ocular affection, Cornil found that the microscopical appearances consisted in an infiltration of the connective tissue with serum, and in a few hæmorrhages into the adjacent part of the retina ('Arch. Gén.,' 1868, ii, p. 679). The absence of grave morbid changes is proved by a case of Mr Lawson's, in which the presence of a hydatid cyst within the orbit caused an extreme state of "choked disc;" four days after puncture of the tumour the engorgement had almost entirely disappeared. A drawing in 'Pagenstecher's Atlas' (Pl. xxxi, fig. 7) shows the nerve-fibres bulging outwards, so as to separate the peripheral layers of the retina from the choroid. In some more advanced cases of von Graefe's, examined by Schweigger and Virchow, the connective-tissue elements, the vessels, and the nerve-fibres were all found swollen, and the latter were beginning to degenerate, while in the outer coat of the vessels there was an overgrowth of nuclei.

The choked disc was attributed by von Graefe to compression of the cavernous sinus, aided by what he termed the constricting action of the sclerotic ring. He argued that if from any cause the flow of blood along the retinal veins was obstructed, the unyielding structure would give rise to a sort of *strangulation* of them at the point where they pass through it, and so he explained the fact that the congestion was limited to the intra-ocular termination of the optic nerve; but (as Dr Hermann Schmidt has

urged in vol. xv of the 'Arch. f. Ophth.') the ophthalmic vein communicates so freely with the facial vein that it is difficult to see how pressure upon the cavernous sinus could appreciably interfere with the escape of blood from the eyeball, nor does it appear probable that the wall of the sinus would yield to any moderate force. These considerations are confirmed by a case under the author's observation, in which the sinus on one side was completely obstructed by softening thrombus; the corresponding optic disc had been noticed during life to be perfectly normal. In all probability Schmidt's explanation is the correct one. He finds experimentally that an injection of Prussian blue into the arachnoid cavity (subdural space of the brain) makes its way through the optic foramen into the space between the two sheaths of the optic nerve, and fills a fine network of lymphatic channels within the lamina cribrosa (formed by the sclerotic ring) between the very fibres of the nerves themselves. He therefore suggests that some of the fluid which is always present in small quantity in the arachnoid cavity is driven into the optic nerve in a similar manner whenever the intra-cranial pressure is from any cause increased. As a matter of fact, a watery liquid has more than once been found distending the sheath and giving it a bulbous appearance, and this liquid readily escapes as soon as a puncture is made. One difficulty is in explaining on Schmidt's theory why cerebral hæmorrhage does not cause choked discs. Is it because the great force suddenly exerted flattens the sheath of the nerve, and closes the channel through it?

Optic neuritis.—The distinctive features of this affection, as compared with choking of the disc, are that its swelling and redness are less marked, but that it looks more opaque, and that the morbid process extends further into the retina itself, and (according to von Graefe) involves its middle and outer layers, as well as its inner layer. Dr Allbutt endeavours to describe the appearance of the disc by saying that there is not "a circumscribed intense redness, or brownish grey, but rather a wash of reddish lilac or a grey tint." And he adds that one does not perceive "a multitude of minute branches and capillaries," such as give a "mossy" look to the choked disc, but that the vessels which become distended and tortuous are rather the main trunks. Moreover, these are often completely concealed in part of their course by the opaque tissue, beneath which they dip.

One of the chief microscopical appearances seems to be that the optic nerve-fibres within the disc are enlarged and beaded, presenting a series of fusiform swellings. The connective-tissue bundles are said by Virchow to present no excess of cells or nuclei, but the coats of the vessels are thickened. The trunk of the nerve in its whole length shows an accumulation of cells and nuclei within its sheath, and also between its fasciculi.

As stated above, von Graefe ascribed neuro-retinitis to the direct extension of inflammation downwards; as, for instance, from a basal meningitis. We shall find that this explanation is not always applicable. The probability is that the affection is then secondary to a choked disc, being developed in the same way as those chronic affections which are well known to occur in the lungs and liver and kidneys when there is obstructive disease of the heart. It is not surprising that (as Mr Hulke has remarked) mixed forms of ophthalmoscopic changes are more commonly seen than typical specimens of a "Stauungs-papille" or of neuro-retinitis.

Dr Gowers holds that congestion of the disc is not the result of intra-cranial pressure, that it is always inflammatory or irritative in origin, and

that the distinction between the choked disc and optic neuritis is one of degree only.

Atrophy of the disc.—Neither congestion of the disc nor neuro-retinitis is a permanent affection. They are not even stationary; for the process, if the patient lives long enough, must subside or quickly end in atrophy. The latter is by far the most common termination. The disc becomes less and less swollen, and at length is quite flat, or even sinks below the level of the surrounding retina. Its red and grey tints grow into a dirty white colour. The tortuous veins diminish in size; spots of hæmorrhage fade and are absorbed. The outline of the disc remains for some time blurred and irregular, its margins are ragged, and streaks of exudation are to be seen in the course of the retinal vessels. This description of "consecutive atrophy" is taken from Dr Allbutt, but it accords with those given by all other writers. He goes on, however, to say that these appearances also are transitory. Little by little the disc clears up; its edge becomes sharply defined; its surface dead white and glistening. Its condition is then undistinguishable from one which has been preceded by no swelling or inflammation of the disc—the "simple white atrophy" of writers. Pagenstecher, indeed, stated that in microscopical sections of the dead tissues the origin of the affection may be recognised by the permanent displacement outwards of the two external layers of the retina from their normal commencement close to the edge of the choroidal ring. As to the histological changes in the disc itself, he shows that all the nervous elements may disappear, and be replaced by coarse connective tissue.

It is surprising how little impairment of vision attends these changes in the optic discs in many cases. Pain and over-sensitiveness to light are constantly absent. If there is any affection of sight, it is that the patient either cannot see at all, or sees objects more or less indistinctly, as if through a mist. Dr Allbutt speaks of several patients with choked discs who could read a badly printed news-sheet with ease. One should therefore make it a rule to examine with the ophthalmoscope all cases in which symptoms of cerebral disorder are present; unless, indeed, an exception may be made for some of the simple uncomplicated neuroses. On the other hand, if a patient is found to present morbid appearances in the fundus of the eye, one should not be contented with his statement that his sight is perfect. The extent of the visual field in all directions should be accurately explored; and perhaps it may turn out that although vision is good at the centre, it is yet very defective at the periphery or towards one side. When blindness sets in, it sometimes seems to come on almost suddenly, although the changes in the discs were no doubt developed very gradually. This occurred, for instance, in a patient under my care in 1878; about three weeks before her admission she went to sleep one afternoon, and on waking up found that she was totally blind. If perception of light is completely lost, the irides are widely dilated and motionless. But in those cases in which the impairment of sight is partial, the pupils often appear to be of normal size, and their movements are sometimes not even sluggish. The suggestion of M. Jaccoud, that the excitation of reflex contraction of the iris by light is a proof that blindness is due not to any affection of the optic nerve within the eye itself, but to a lesion situated above the corpora geniculata, seems to be altogether fallacious.

When a choked disc, or one affected with neuritis, passes into a state of atrophy, the patient's sight often becomes progressively worse; but some-

times, on the other hand, it shows a marked improvement. Thus a patient of Dr Goodhart's, who at one time could see absolutely nothing with her right eye, was ultimately able to read Snellen's $2\frac{1}{2}$, though with difficulty, at a distance of about a foot. When the power of vision has been but slightly impaired during the early stage of the disease, it is sometimes perfectly regained; but even in such cases, if actual neuritis has been present, the disc seems invariably to become more or less atrophied; it never resumes its normal appearance.

Localisation of organic cerebral lesions.—We now turn to other symptoms, which not only indicate in general the *presence* of some local lesion—whether a tubercle, gumma, or tumour,—but also point more or less definitely towards its exact *seat*; they constitute what Dr Jackson terms "localising symptoms."

In the interpretation of these symptoms there is this preliminary difficulty. When one finds a tumour, or a tubercle, in some particular region of the brain, one cannot tell whether the disturbances of function to which it gave rise were the results of *irritation* of the structures around it, or of *destruction* of those among which it grew. In the latter case the symptoms would be those of the uncontrolled or unbalanced action of the corresponding parts on the opposite side of the brain, or of some other parts physiologically antagonistic to those containing the lesion. It is precisely this difficulty which makes it impossible to attach an exact meaning to inequality of the pupils; and several other instances will present themselves further on. Dr Jackson distinguishes cerebral lesions as "destroying" or "discharging." It is remarkable how great a size may be reached by a mass in the brain, without its depriving the structures in which it is embedded of their function; while, again, a tumour sometimes compresses adjacent parts, so as to annihilate their activity. Without attempting to lay down a positive rule, it may be said that, for the diseases now under consideration, the presumption is in favour of irritation, rather than destruction, when either explanation is admissible.

The base.—Beginning at the base of the brain, and passing from before backwards, one meets with the simplest of all localising symptoms, namely, those which depend on interference with the various cranial nerves.

It is easy to see that complete *anosmia* points to the presence of disease near the ethmoid bone, or involving the inner and lower parts of the two anterior lobes; while loss of smell on one side may be due to a lesion implicating one olfactory bulb, or the root of the nerve extending outwards to the Sylvian fissure.

A growth pressing upon the optic chiasma produces various effects according to its exact situation. Thus there may be more or less complete loss of sight in one eye, or even total blindness; but as changes in the discs are almost certain to be present, it would be scarcely possible to draw any inference from this symptom. What is really significant is the limitation of a visual defect to half the field in each eye, or *hemiopia* (cf. p. 567). Of this there are three different forms, but their interpretation is still somewhat doubtful, because opposite views are held as regards the structure of the chiasma. On the theory of semi-decussation, which appears the more probable, they may be described as follows:

(1) Compression of the centre of the chiasma, cutting off the decussating inner fibres of each optic nerve, will cause loss of vision over the temporal

field in each eye (*double temporal hemiopia*); (2) compression of one optic tract will deprive the patient of the nasal field in the corresponding eye, and of the temporal field in the opposite eye (*homologous lateral hemiopia*); (3) loss of vision in the nasal fields of the two eyes (*double nasal hemiopia*) can only be due to a double lesion, symmetrically placed so as to interfere with the non-decussating outer fibres on each side. Examples of all these forms of hemiopia are known to occur. Homologous lateral hemiopia is very common in attacks of migraine; of double temporal hemiopia cases have been recorded by Sämisch and by E. Müller, in each of which a large tumour lay in the middle line, involving the chiasma, and a third very singular one by Dr Weir Mitchell due to pressure of an aneurysm of an abnormal anterior communicating artery ('Journ. Nerv. and Mental Dis.,' Jan., 1889); of double nasal hemiopia, one by Knapp, in which the pressure at the necessary points was effected by dilated and atheromatous branches of the circle of Willis.*

Intra-cranial growths sometimes produce *deafness* when they occupy, or extend into, the space between the internal auditory meatus and the side of the pons. Dr Hughlings Jackson has met with one case in which there was loss of hearing from this cause without any facial paralysis. In another instance a complete bilateral destruction of the auditory sense was imperfectly explained by the autopsy, for the only growth at the base was one of the size of a hazel-nut on the *left* side. It might be suggested that this symptom ought sometimes to be referable to an affection of the brain itself; but Dr Jackson's experience appears to negative such an opinion.

With regard to affections of the other cranial nerves, there is little to add to the remarks made when describing their symptoms (see pp. 407—420). An anatomist can sometimes infer the seat of a growth with great accuracy from the implication of some nerves while others escape. Thus, in one instance, the fact that all the muscles of the eyeball were paralysed, while no part of the fifth nerve was interfered with except its ophthalmic branch, seemed to prove that the disease occupied the sphenoidal fissure. On the other hand, it is sometimes possible to draw the conclusion that no single lesion will explain every feature of the case, a conclusion which (as we shall presently see) may be of considerable diagnostic importance. Paralysis of the third nerve, if due to an intra-cranial tumour, points particularly to a syphilitic gumma of the base.

The cerebellum.—The localisation of a tumour or tubercle in the cerebellum is often attempted, and is not infrequently successful. But one must not forget that this part of the encephalon is a very common seat of such growths, so that the result of the diagnosis may possibly sometimes be due to good luck rather than to skill. Beside occipital headache, contraction of the muscles at the back of the neck, vomiting, convulsions, blindness, and vertigo—no one of which symptoms is special to lesions of the cerebellum—there are some others which are believed to be more characteristic; particularly a *reeling, staggering gait*, like that of a drunken person, but with a tendency to fall in some particular direction. It was long

* On the theory of complete decussation, "double temporal hemiopia" would be caused only by pressure on the *front* of the chiasma, while pressure on the *back* of the chiasma by a single growth would account for "double nasal hemiopia." So far that theory might hold, although both these kinds of hemiopia are very rare, but it gives no explanation of the far more common "homologous lateral hemiopia," excepting the untenable one of a lesion being placed *on one side* of the chiasma, in such a situation as to compress a part of the optic nerve, and also a part of the optic tract on that side.

ago shown by the experiments of Flourens on pigeons that excision of the cerebellum rendered the bird unable to walk or to fly, or to perform regular determinate movements; and he suggested that the function of the organ was one of co-ordination. Subsequent investigations have proved that a large part of the process by which the actions of the individual muscles are harmonised and combined takes place in the spinal cord. But this fact does not exclude the possibility that a higher co-ordination may be effected by the cerebellum; and an hypothesis has been suggested by Dr Broadbent, namely, that what it does is to bring muscular action into relation with visual impressions, as when movements have to be guided by sight; whereas in the spinal cord they are arranged for tactile and other cutaneous impressions. However, a patient suffering from disease of the cerebellum may still stagger, although, in consequence of his optic discs having undergone atrophy, he can no longer see anything.

When the staggering is accompanied by a tendency to fall in one particular direction, we might expect that this circumstance would not only indicate the presence of disease in one side of the cerebellum, but also show which side is affected. But tumours of the cerebellum often attain a great size, and destroy a large part of the organ, beside compressing other parts or interfering with their supply of blood. Moreover, the same affection may produce diametrically opposite effects, according as its action is that of a "destroying" or of a "discharging" lesion.

In 1877 an autopsy in a case of Dr Frederick Taylor's showed a cheesy tubercle in the cerebellum, rather to the right of its centre; the right lateral lobe was universally pale and softened, and full of granule-masses. The patient was a boy, five years old. It so happened that the exact seat of the mischief had been indicated several months before his death, by a bulging of the right side of the occipital bone, which was so much thinned at one spot that it would yield on pressure, and rebound like a piece of tin or cardboard. At the same time it was noticed that when he sat up in bed he had no balancing power; but he did not roll over on one side rather than on the other. The eyes were affected with a peculiar form of nystagmus, which might perhaps have been turned to account as a localising symptom. "It came on only when he lay on the left side; the eyes would then gradually fall over to the left, and be suddenly jerked back to the right; and these movements would be repeated rhythmically. When he sat up in bed there was a continuous rhythmical movement of the head from left to right." Afterwards, six weeks before he died, "he lay on his back, with his head turned to the left; his eyes were then directed to the left side, and jerked from time to time upwards." Now, Hitzig, following Purkinjé, has shown ('Reichert's Archiv,' 1871) that by passing a galvanic current through the head of a healthy person, from one mastoid process to the other, one can obtain oscillatory movements of the eyes exactly like those observed in Dr Taylor's patient; and in a subsequent paper he accounted for those effects by referring them to disturbances of the cerebellum. Dr Ferrier, too, found that galvanising the exposed cerebellum of monkeys and other animals caused the eyes to deviate in different ways, according as the poles were applied to different regions of its surface. Indeed, his experiments seem to indicate a very simple law of localisation, namely, that when any part of the cerebellum is the seat of irritation the eyes become turned in the corresponding direction, whether to the right or to the left, upwards or downwards, as the case may be. In many cases analogous movements of the head accompanied

those of the eyes. Thus a tendency to fall backwards, which has sometimes been noticed in cases of cerebellar disease, would point to the upper surface of the organ as its seat; for Dr Ferrier found that galvanising that part in monkeys led to upward movements of the eyes, and to throwing back of the head. Dr Taylor's patient had also left hemiplegia, which was no doubt due to compression of the right side of the pons by the tumour.

Hemiplegia in cerebellar disease appears to be always produced in that way, and it is usually "crossed," affecting the limbs on the opposite side to the cerebellar lesion. But the functional relations of each half of the cerebellum are nevertheless chiefly with the limbs of the same side of the body through the connections of the fibres of the middle peduncle with nuclei in the opposite half of the mesencephalon. This is proved, not only by anatomical and physiological investigations, but also, and still more convincingly, by the pathological fact that when there is congenital unilateral atrophy, affecting one hemisphere of the brain, one crus cerebri, and one side of the deep part of the pons, the wasting is found in the opposite side of the cerebellum, as it is in the opposite side of the spinal cord.

On the whole, it may be said that when there are the general symptoms of an intra-cranial tumour (including tubercle, gumma, cyst, and abscess), namely, headache, vomiting, and optic neuritis, especially optic atrophy and amaurosis—and when there is no hemiplegia, but only general muscular weakness, when there is spasm of the muscles at the nape of the neck, and decided vertigo and loss of power to maintain equilibrium—the diagnosis of the cerebellum as the seat of the lesion is almost certain.

This last symptom, disturbance of equilibrium, is said by Nothnagel to depend on lesion of the superior vermiform process, and this conclusion is confirmed by other observers. Tumours of the lateral lobes of the cerebellum appear to have no special symptoms; but they may sometimes be more than guessed at by the effects they produce through pressure on the pons or other adjacent structures.

The motor area.—Passing now to the regional diagnosis of lesions involving the motor tract, we may observe that the greater part of the description of hemiplegia given in the last chapter applies to the same symptom when it is produced by a new growth. But whereas we have seen that certain parts of the brain are far more liable than others to suffer from the effects of embolism, or of rupture of a cerebral artery, the distribution of tumours is comparatively irregular and capricious. They may interfere with the fibres which pass down to the spinal cord at any point in their course, and may destroy them either in whole or in part. On the other hand, it is wonderful how large a tumour may be found in the very substance of the motor tract, without completely abolishing its functions. In 1869, a boy, aged four years and a half, died in Guy's Hospital under the author's care, after an illness of fourteen months' duration. Three months before his death he could sit up in bed, although he rolled about and his balance was easily upset. A month later he "could move his legs a very little." Dr Moxon found that the pons was occupied by an immense caseous tubercular mass, which consisted of two halves fused together, and preserving almost exactly the normal shape of the part; only a thin shell of nervous matter remained. It seemed marvellous that life could have been maintained while the mass was growing to such a size. In another case a minute tumour was found in one of the corpora dentata of the bulb, which had given rise to no symptoms of disease of that part.

Then, again, paralysis of the limbs on one side of the body, or even of all four limbs, may be due to a mass pressing on the motor tract from without; for instance, to a sarcoma connected with the base of the skull, and compressing the medulla oblongata or the pons; or to a glioma in the hemisphere above the lateral ventricle, pushing downwards and flattening the basal ganglia. In other cases, when the tumour lies altogether outside the motor region, hemiplegia is caused by the yellow softening that so often develops itself secondarily.

A growth situated in the superficial convolutions of one *hemisphere* may affect the movements of the opposite side of the body, without either disturbing the corpus striatum mechanically by pressure, or involving it in any morbid action. The symptoms so produced are among the most interesting that come under the observation of physicians. They are generally convulsive in character, but the spasms are often followed by a transitory paralysis; and it is still a question whether a loss of power may not occur primarily and alone. Bright was the first to point out, in the earliest volume of the 'Guy's Hospital Reports' (1836), that some cases in which fits were due to a local lesion presented the peculiarity that consciousness was not lost. But this form of convulsions was never systematically investigated until, about 1865, Dr Hughlings Jackson took up its study, in the hope that it would throw important light upon the nature and seat of epilepsy in general. He insisted on the fact that *hemispasm*—the "mobile counterpart" of hemiplegia—must indicate a condition of "instability" in the convolutions which discharge through the corpus striatum, and must therefore be a symptom of disease in those convolutions or (possibly) in the corpus striatum itself. And he set to work to determine, so far as pathological opportunities might offer themselves, the exact seat of the lesions found where this symptom had been present. His theories have since received remarkable confirmation by direct experiment, and the particular form of convulsions in question is now commonly known abroad as "Jacksonian Epilepsy."

It was in 1870 that Fritsch and Hitzig made known the fact that, instead of the surface of the brain being insensible to galvanic currents, as Schiff and other physiologists stated, there were in the dog certain parts of the convolutions which reacted to such currents in a very definite way, each setting in motion some particular limb, or even some special set of muscles. Thus they laid down the position of "cortical centres" for the movements of the neck, face, fore- and hind-leg respectively. Three years later Dr Ferrier repeated these experiments with the faradic current, employing various animals, and at length monkeys, in which the principal convolutions are comparable, one by one, with those of the brain of man. Afterwards Hitzig himself operated on a monkey. Between the results of these two observers there were some discrepancies in details. Hitzig used a comparatively weak galvanic current; thus he obtained movements over a far more limited area; indeed, his main object was to discover what parts of the cortex would yield movements limited to isolated groups of muscles on the most feeble stimulation. He maintains that in the monkey the motor centres for all parts of the body are situated in the anterior central (ascending frontal) convolution. That for the leg is nearest the falx cerebri, at a distance of about three millimetres from it; that for the arm lies three millimetres further outwards; that for the upper part of the face, supplying the muscles of the ear and eye, is situated rather more

behind and externally; that for the lips, tongue, and jaws is six millimetres inwards from the fissure of Sylvius. Hitzig did not deny that irritation of adjacent convolutions by more powerful (especially by induced) currents gives rise to movements in distant parts; but he attributed them to the action of the current upon parts beneath the cortex.

Ferrier placed his motor centres in the posterior central (ascending parietal) as well as in the anterior central (ascending frontal) convolution, and also in the postero-parietal lobule; and his sensory centres in the back part of the third frontal (speech), in the angular (vision)* and in the superior temporo-sphenoidal convolution (hearing). He agreed with Hitzig in placing the centre for the leg close to the median line, but he divided it into two,—one in the postero-parietal lobule, for advance of the hind limb, as in walking; the other in the upper parts of the ascending parietal and ascending frontal convolutions, for climbing and other complex movements of arms and legs. He made the centres for the arm and hand occupy the ascending parietal convolution nearly as far outwards as the fissure of Sylvius, as well as the ascending frontal and the superior frontal convolutions outside and in front of the leg-centres. The movements of the lips, tongue, and mouth he connected with a series of centres occupying the lower part of the ascending frontal, and with one in the supra-marginal convolution. Centres for the movements of the eyes were believed to occupy an extensive area in the two upper ("first" and "second") frontal convolutions. Stimulation of the angular gyrus, or of the superior temporo-sphenoidal convolutions, also caused the eyeballs to move, with dilatation of the pupils; but he supposed these parts to be centres for the senses of sight and hearing, and the movements to be reflex.

Since the publication of Ferrier's book in 1876, Nothnagel, Munk, Goltz, and other experimenters in Germany, and Burdon-Sanderson, Yeo, Schäfer, and Horsley in England, have repeated and varied his experiments. The stimulation of definite cortical areas has been controlled by destroying corresponding parts of the hemispheres. At the International Medical Congress which met in London in 1881, Prof. Goltz brought a dog from Strasburg which had survived extensive destruction of successive portions of the cerebral cortex, and this living witness was confronted by a monkey which was brought forward by Dr Ferrier. After observation of the functions of both animals they were destroyed, and the brains were subsequently submitted to most exact and thorough anatomical examination by Dr Klein, Mr Langley, Mr Schäfer, and Dr Gowers. The account of the discussion in the 'Transactions' of the Congress (vol. i, pp. 218—240), and the detailed reports by Langley and by Schäfer, published in the 'Journal of Physiology' (vol. iv, pp. 248—326), furnish an admirable example of the difficulty of ascertaining and interpreting scientific facts, and of success in at least the former task.

The practical difficulties have been—in faradic stimulation, the danger of a current spreading, either too widely or too deeply; in destructive operations, the appearance of much wider and more complete ablation than proved to be the case; in pathological observations, the infrequency of locally circumscribed, single and uncomplicated lesions; and, generally, the fallacies resulting from vaso-motor disturbance, from inhibition of distant ganglionic centres, and from direct or indirect mechanical pressure.

* In the second edition of Dr Ferrier's work, 'The Functions of the Brain,' 1886, he includes the occipital lobes in the visual area (pp. 271 *et seq.*).

Nevertheless, pathological cases completely observed, completely examined after death, and free from complications, have gradually accumulated. A new series of experiments on the monkey's brain has been carried out by Profs. Ferrier and Yeo, and an independent series, with no less skill and care, by Profs. Schäfer and Horsley. In the congress of physiologists which met at Bâle in September, 1889, Mr Horsley and Dr Beevor demonstrated on a monkey under anæsthetics the accuracy with which predicted movements of the thumb, forearm and shoulder, the eyes and the tongue could be produced by faradic stimulation of definite areas of the cortex.

It may now be taken as certain that there are excitable areas in the neighbourhood of the fissure of Rolando, which are connected with definite movements. The exact locality of the motor areas has been accurately ascertained, and has been confirmed by the crucial proof of operation upon human patients. Other portions of the cerebral cortex are not thus excitable. The precise localisation of sensory areas is still doubtful. Disregarding, therefore, the anterior frontal, the posterior parietal, occipital, and temporo-sphenoidal regions of the cortex, we have a "motor area" around the fissure of Rolando, which includes the ascending frontal with the closely adjacent part of the third, second, and first frontal convolutions, and the ascending parietal, together with the inner aspect of the same, which forms part of the marginal gyrus, and the front of the superior parietal lobule (postero-parietal of Huxley and Turner). Irritation of these parts is capable of causing definitely distributed clonic spasms of the muscles on the opposite side of the body—a "discharging" lesion; destruction of the same parts is capable of causing definitely distributed loss of power in the same muscles on the opposite side of the body—a "destroying" lesion.

The area which corresponds with certain movements of the *leg* comes first, occupying the borders of the fissure of Rolando close to the falx cerebri, and extending over the adjacent parts of the ascending frontal (or præ-central), the ascending parietal (or post-central), and the inner surface of the same part of the hemisphere, which corresponds to the marginal convolutions immediately behind the calloso-marginal fissure. The area which corresponds with movements of the *arm* is below and external to the last, and occupies the ascending frontal and parietal convolutions opposite the second frontal gyrus in front and the angular gyrus behind. The area which corresponds with movements of the *facial muscles* appears to be lower down in the same direction, in front and behind the lowest point to which the fissure of Rolando reaches. Lower still, at the angle between the ascending and horizontal branches of the Sylvian fissure, opposite the third frontal convolution, this region marches with the gyri operti, the insula, and Broca's aphasic region.

Lastly, high up and internal is a motor area which lies in front of that for the lower extremity, occupying the back part of the first frontal convolution, which is believed to be associated with movements of the trunk on the opposite side.*

* Centres for the muscles of the eyeballs, for movements of the tongue and jaw, and for movements of the tail, have been ascertained with more or less exactitude in the monkey and the dog. The difficulty of comparing the results, even in the former animal, with its different mode of progression and of prehension from human physiology, is obvious, and is fully recognised by Ferrier. Disturbance of the ocular muscles in man appears to be more often connected with lesions of the motor nerves, of the bulb or of the cerebellum, than with those which affect the cerebral cortex.

When we apply the knowledge thus obtained with a view to the diagnosis of circumscribed lesions of the cerebral cortex, we are met by certain clinical difficulties.

In the first place, it is to be noted that the occurrence of convulsive attacks without unconsciousness beginning in, or limited to, some particular part of the body, is not of itself a proof that a recognisable local lesion exists in the brain at all. We shall find several instances of epilepsy in which attacks of the kind were almost certainly "functional." Thus in a case of Dr Jackson's, the only morbid change discovered at the autopsy was atrophy of both hemispheres, although the fits which had occurred had begun in the right index finger and thumb, and had often been unattended with loss of consciousness ('Med. Times and Gazette,' 1872).

Moreover, according to Dr Jackson, the seat of localised convulsive seizures, or of general seizures beginning with local spasms, is in the majority of cases some part of the hand, generally the thumb or the forefinger; the next most frequent seat is the cheek or the tongue, and it is much more rarely the great toe.

The results of experiment have, however, been confirmed by clinical experience. Some of the earlier cases may be briefly cited. (1 and 2) Two cases of Jackson's (one recorded in the 'Med. Times and Gaz.' for 1872, the other quoted by Ferrier), in each of which the spasms always began in one thumb, and a lesion was found in the opposite first frontal convolution. (3) A case at Guy's Hospital where we found in the back part of the first frontal gyrus a small glioma on the right side; there had been repeated fits without unconsciousness, starting mostly in the left foot, but occasionally in the left arm. (4) A patient of Griesinger's, who was attacked with transient spasms in the right leg, afterwards affecting the right arm, the face, and the tongue; a cysticercus lay close to the left side of the falx cerebri, in such a position that its anterior extremity coincided with a line drawn vertically upwards from the ear. (5) Recorded by Dr Dreschfeld, of Manchester ('Lancet,' 1877). The convulsive movements began with a sudden clenching of the left fist, flexion of the wrist, and pronation of the forearm, while the left angle of the mouth was at the same time strongly drawn downwards; a local syphilitic lesion was found in the adjacent parts of the ascending parietal and supra-marginal convolutions. (6) A case observed by Hitzig. The patient was a French soldier who was wounded on the right side of the head near Orleans on December 14th, 1870. On February 4th, 1871, he was attacked with clonic spasms affecting the left side of the mouth and nose, the eyelids, and afterwards the fingers on that side. After death there was found to be a local necrosis of the parietal bone, and an abscess in the right ascending frontal convolution. (7) Another instance, very similar, has been recorded by Wernher ('Virchow's Archiv,' lvi, p. 289). The patient, who had fallen from a railway truck, was attacked with convulsions limited to certain muscles on the right side, especially those of the angle of the mouth, the ala nasi, the eyelids, and the tongue; after death the left temporal bone was found fractured, and the surface of the brain crushed on each side of the Sylvian fissure at the lower end of the fissure of Rolando. (8) A case of Dr Gowers ('Brit. Med. Journ.,' 1874) in which spasms began in the left angle of the mouth, involving afterwards the frontal muscle on each side, but in which the only local lesion appeared to be a clot of blood situated above the right lateral ventricle, just inside the gyrus fornicatus. This case certainly does

not correspond with the observations of Hitzig and Ferrier; but it does not contradict them, as if, for example, a small tumour were to be found in the occipital lobe when limited spasms had been present during life.* (9) A case reported by Ferrier ('Brain,' April, 1880) of convulsions in the left arm and leg following hemiplegia, in a man the subject of phthisis. At the autopsy a tubercular growth was found occupying both sides of the fissure of Rolando on the external aspect of the right hemisphere, and extending into the internal aspect as well.

During the last ten years many similar cases have been recorded by Jackson, Gowers, Bastian, Hadden, and other observers in this country; and a still larger number in France, Germany, and America.

We are now justified in stating that lesions of certain particular convolutions are capable of causing limited paralysis instead of spasms. Dr Hughlings Jackson long ago pointed out that unilateral convulsive seizures were often followed by a more or less complete hemiplegia; but he then believed that this form of paralysis was always transitory, passing off in a few days or weeks. No doubt persistent local paralysis is seldom or never caused by a cerebral tumour, unless it interferes directly with the corpus striatum, or with some part of the motor tract. But Dr Jackson's distinction between "discharging" and "destroying" lesions is in harmony with the fact that although paralysis may not be produced by a new growth in a convolution, it may yet result from softening in the motor region; and almost every instance of aphasia is a case in point. Hence the most careful scrutiny of the convolutions near the fissure of Rolando should be made whenever a patient has had unilateral ptosis, or paralysis confined to part of the face, or to a finger, or any part of the upper limb on one side. The results of Ferrier's and subsequent experiments are decisive of the physiological fact, and clinical observations to the same effect are recorded; two early ones by Löffler were quoted by Hitzig. One is the case of a man whose two parietal bones were both fractured at the vertex by a gunshot wound; he had paralysis of both legs. Another is that of a man who was wounded at the upper and anterior angle of the left parietal bone; his right leg instantly became paralysed, and he fell to the ground. On the seventh day the loss of power extended to the right arm also, but this quickly got better, while in the leg the recovery was very slow. Judging from experiments on dogs and monkeys, paralysis caused by a cortical lesion ought to be limited to some special movements of the affected

* There is, however, a case of Sir William Gull's in which a large abscess in one posterior lobe was attended with attacks of spasm limited to one arm; and a few other cases have been recorded which would appear inconsistent with the experimental results given above, were it not that legitimate criticism seems to deprive them of decided significance. One of these was observed by Dr Jackson. The foot had been the starting-point of the spasms, and a tumour was found which involved the lower part of the ascending frontal convolution; but the brain also presented other lesions. Another is a case of Dr Gowers', in which there was thrombosis of the superior longitudinal sinus and of some of its afferent veins, with hyperæmia of parts of the three frontal convolutions, situated further forwards than any recognised "cortical motor centres;" but in that instance the fits were general, although they began with a slow movement of the hand to the head; and death occurred within two hours from the time of their commencement. Again, in a patient of Dr Buzzard's, a girl of eighteen, a tumour of the size of a walnut was found in the white substance of the *left* hemisphere, extending as far as the grey matter of the gyrus fornicatus; the cause of death was phthisis. The symptom which had suggested the idea that a local lesion might be found was that she had fits beginning with an aura in the *left* wrist; but this is quite a different thing from the occurrence of spasm in the same part. Moreover, the application of a blister to the seat of the aura sufficed to transfer it to the opposite arm.

muscles. An instance of this is afforded by Hitzig's case of the French soldier referred to above (No. 6). His spasms were followed by a partial paralysis of the lower part of the face on the left side; and it is expressly stated that he could voluntarily bring the muscles into action as well as on the opposite side, although that half of the face remained almost motionless when it should have moved in common with the right side for the purpose of expression. This is exactly the opposite of what occurs when disease of the corpus striatum or other lesion of the motor tract causes paralysis of the face (cf. p. 563). In most of the cases reported by Ferrier, Jackson, Bourneville, and other writers quoted by Bastian, there were first spasms of the thumb, arm, foot, or leg, and afterwards paralysis. Sometimes, however, circumscribed convulsions supervened in a paralysed limb.

Lastly, instances have been observed by Gowers of congenital absence of one hand, and by Bastian of a wasted arm, associated with atrophy of the opposite ascending parietal gyrus; also of an amputated limb with similar atrophy of its motor area in the cortex.

The cerebrum outside the motor tract.—We have still to consider what "localising symptoms" may be present when a lesion is seated in the substance of one hemisphere, or in the superficial convolutions beyond the motor area. As we have seen, the cortical centres for the special senses are still imperfectly known, and tactile sensibility is almost always less affected than movement. Hence it is not surprising that no special localising symptoms can as yet be connected with cortical lesions of the front part of the frontal lobes, the temporo-sphenoidal or the occipital. In many cases the only symptoms are lethargy, listlessness, an oddness of manner, taciturnity, and unwillingness to speak, or loss of memory and impairment of intelligence—symptoms that we are accustomed to associate with diffused morbid changes affecting both hemispheres. These, it is interesting to remark, are the kind of symptoms which are found, in monkeys and in dogs, to result from ablation of the cortex outside the motor area.

The general symptoms of headache, vomiting, and optic neuritis may point to the presence of a tumour, but unless it happens to press on some nerve-trunk we have no means of determining its seat.

It is well to remember that cerebral hæmorrhage, the commonest of "destroying" lesions, is, as we found (*supra*, p. 557), rare in these "non-motor" regions compared with the corpus striatum, the pons, and the cerebellum. But tumours and cerebral abscesses are much less rare, so that we have sufficient evidence of their negative effects.

In a case under the writer's care in which the symptoms pointed to a cerebral tumour, its locality was ascertained by signs of pressure on the left orbit; the skull was therefore trephined, and a large growth was found in the anterior part of the frontal lobe. While these sheets are passing through the press, a patient under the care of Dr Shaw, whose sense of smell was noted to be decidedly deficient during life, died with symptoms of organic cerebral disease, and a cyst was found in the temporo-sphenoidal lobe.

The basal ganglia.—We have seen that ordinary hemiplegia does not, as was formerly supposed, depend upon lesion of either grey nucleus of the *corpus striatum*, but upon interruption of the motor tract as it passes between the caudate nucleus and thalamus (the internal capsule behind the genu), or upon severing of the white fibres of the external capsule or corona radiata outside the lenticulus, as they run from the motor area of the cortex.

Destructive lesions confined to the lenticulus or the cauda may produce no special symptoms.

The *thalamus* is more directly in the motor tract, but it is rarely the seat of disease, and the symptoms then produced are not characteristic. In the case of a boy in Guy's Hospital, which was published in the 'Pathological Transactions' for 1884, a tumour occupied the right thalamus. There was headache and double optic neuritis, with ptosis and slight motor paralysis of the opposite side affecting the arm more than the leg; no marked anæsthesia, if any, and no blindness or strabismus.

Lesions of the back of the thalamus have caused hemiopia, but there was probably pressure exerted on the optic tracts or the corpora quadrigemina.

The *pituitary body* is not infrequently the seat of a cancer, glioma, or other tumour. Three cases are noted in Dr White's list (p. 602, *supra*): one of these patients, a man of forty-five, with symptoms of tumour of the base, had a secondary growth in the lung. A fourth was a cystic tumour.

The *pinea* or conarium may contain a psammoma or sarcoma. Dr Heinrich Reinhold has collected four or five cases in a monograph ('Tumor der Zirbeldrüse,' 1886). In his case it was a glio-sarcoma.

Diagnosis.—The diagnosis of the affections described in this chapter involves two distinct questions. First, they have to be distinguished from other diseases of the nervous centres or of distant parts; secondly, they have to be differentiated from one another. The answer to the former question is often wonderfully positive and exact. To the young student—who perhaps knows the difficulty of distinguishing between tumours and other surgical affections of parts that can be seen and handled—nothing is more striking than the confidence with which the physician can sometimes assert the existence of a tumour in the cranial cavity.

Cases do, indeed, occur which are only cleared up in the *post-mortem* room. A man of whom little is known, or who has hitherto shown no marked symptoms of cerebral disease, may die in a succession of fits, or in coma of a few hours' duration, and it may be difficult to decide between tumour, cerebral hæmorrhage, thrombosis of an artery, and uræmia. Or, if he lives for two or three weeks, between tumour, abscess, and meningitis.

But, as a rule, the illness caused by a tumour, or a tubercle, or a syphilitic gumma in the brain begins gradually, and goes on for several months. In the cases that have occurred at Guy's Hospital within the last few years the duration of well-marked symptoms has generally been from three to nine months, but one patient had had fits for four years. Jackson has recorded the case of a woman who had optic neuritis and staggered in walking in 1865, and who did not die until the summer of 1872, when she had become insane—there was a growth springing from the "floor of the sphenoidal fossa." It may be affirmed that when cerebral symptoms of the kind described in the present chapter have lasted for more than five or six weeks, they are due either to a tumour or to red softening. Abscess is secondary to injury or some source of suppuration, and primary red softening is very rare.

There still remains another important source of difficulty. In women the symptoms of a cerebral tumour are sometimes so vague that for a long time they are mistaken for those of *hysteria*. The writer remembers a young woman in Mary Ward who had every symptom of hysteria, and who no doubt was highly hysterical. But Dr Willis, whose patient she was,

always asserted that there was more than hysteria. One morning, to the astonishment of nurses and students, she was found dead in her bed, and at the autopsy we found a cerebral tumour. A painter once died in Guy's Hospital of drowsiness and epileptiform fits, which were believed to be the effects of lead-poisoning; but a spindle-cell sarcoma of the size of a marble was found in the right hemisphere, with extensive yellow softening around it.

The ophthalmoscope.—In such cases, as in many others, the routine use of the ophthalmoscope is a great safeguard. The discovery of choked discs, or of optic neuritis, or of atrophy, goes far towards establishing the presence of an organic lesion. No doubt at first the significance of these appearances was rated too highly. When neuritis, or choking of the disc, affects one eye only, it is sometimes a sign that the cause lies in the orbit rather than within the skull. Mr Lawson brought before the Clinical Society in 1876 a case in which a hydatid cyst pressed upon the nerve behind the eye, and so caused great swelling of the disc on that side. Again, unilateral optic neuritis may be due to pressure upon the optic nerve between the chiasma and the optic foramen; a gumma at the base seemed to have acted in this way in a case recorded by Mr Hulke in the 'Ophthalmic Hospital Reports.' Dr Jackson has met with two cases in which the ophthalmoscope revealed an affection of one eye, dependent upon the presence of a tumour in the opposite cerebral hemisphere. In a patient of the writer's there was much more severe optic neuritis on the same side as a cerebral abscess than on the other. But it is a rule to which there are very few exceptions that both optic discs suffer from disease of the brain.

In some rare instances ophthalmoscopic changes seem to precede all other symptoms. A patient of Dr Allbutt had amaurosis from atrophy for three years before any signs of cerebral tumour began to manifest themselves, although these afterwards became well marked. But optic neuritis alone is not enough to indicate organic disease. In a series of unselected cases, recorded by Mr Hulke in the 'Ophthalmic Hospital Reports,' there are several in which optic neuritis, terminating in atrophy, seemed to be either spontaneous, or attributable to such vague causes as childbearing, lactation, leucorrhœa, sexual excesses, or to an antecedent attack of diphtheria or rheumatic fever; and in 1866 von Graefe spoke of it as sometimes due to menstrual disorders.

Even when unmistakable indications of cerebral disturbance are present, it is a question how far one is justified in concluding from ophthalmoscopic appearances that there must be organic lesion in the brain. In many of Mr Hulke's cases the other symptoms did not support the diagnosis of organic lesion of the brain. Three of them, all of which terminated in recovery, were thought to be probably examples of meningitis; but the evidence was imperfect. Another case would certainly have been regarded as one of ordinary epilepsy if the eyes had not been examined. Two patients, both advanced in years, had hemiplegia, and ultimately died of apoplexy: one, a man aged thirty-eight, attributed the affection of his eyes to watching a solar eclipse; another, a woman aged twenty-four, ascribed hers to standing in the garden with her head uncovered.*

* Dr Hughlings Jackson, who believes that double optic neuritis is almost certain evidence of what he terms "coarse disease" within the cranium, admits that he has himself met with a few instances in which, after death, no such disease could be found. Of one case of this kind he has published full details. A woman, aged thirty-four, had for about a year been subject to attacks of severe headache accompanied with vomiting; for three

In describing meningitis we shall mention cases in which symptoms very like those of that disease have been associated with double optic neuritis, but in which, although they terminated fatally, no lesion could be discovered either in the membranes or in the cerebral substance. Surely it is but common sense to suppose that, if one could see the state of the brain in cases which recover, it would be found equally free from obvious morbid changes. Ophthalmoscopic appearances have great value along with other symptoms in the diagnosis between organic and functional diseases, but they are not more pathognomonic than the rest.

Another question is whether any special inference can be drawn from the exact character of the ophthalmoscopic appearances in a case believed to be one of local organic disease of the brain. Meningitis often accompanies a gumma of the base; and a tumour in any part may set up extensive yellow softening. It might be thought that neuro-retinitis, as distinguished from choking of the disc, would indicate the presence of such secondary affections. But one of Mr Hulke's cases seems to show that the former change may depend upon the presence of a sarcoma attached to the floor of the skull, without there being any evident inflammation of the membranes or of the brain itself. Nor can simple atrophy of the disc, without antecedent neuritis, be taken as a proof of the existence of hydrocephalus.

To conclude, double optic neuritis certainly most often indicates a tumour, less commonly meningitis or hydrocephalus or tabes, rarely or never cerebral embolism, thrombosis, or hæmorrhage. It may occur in Bright's disease or after fever. Single optic neuritis rarely points to a cerebral lesion. Acute optic neuritis is often caused by a chronic lesion, as a tumour. Primary optic atrophy is not so often a symptom of local cerebral lesions as of more general diseases, such as tabes, general paralysis, and insular sclerosis; but when secondary it has the same significance as the descending optic neuritis, of which it is most often the effect. Atrophy, like neuritis, of one optic disc seldom points to cerebral disease; much more often to a "peripheral" lesion affecting the optic nerve or tract.

The *differential pathological diagnosis* between the several forms of local organic disease of the brain must usually rest on our knowledge of the most frequent, and therefore the most likely lesions in a given case. In a child there is a strong presumption in favour of *tubercle*. The older the patient,

months she had been blind. Her illness began by her being seized with vertigo and momentary unconsciousness, after which she had headache for four days. At another time the pain lasted for three weeks. She was admitted on December 19th, 1874. On January 6th, 1875, she had an attack of pain so intense as to make her toss her head from side to side, holding it in her hands, and crying, "Oh, my head! I don't know what I shall do." She retched and vomited frequently. Both optic discs were greatly swollen, and the veins in them were dilated and tortuous. After the 10th she sank gradually into what appeared to be natural sleep, which, however, passed into coma, and on the 12th she died by failure of the respiration. A tumour or some similar disease was confidently anticipated, but Dr Sutton, who made the autopsy, found only certain microscopical changes in the cortex.

Compare with this case one that was brought before the Clinical Society in 1876 by Dr Goodhart and Mr Higgens. A girl, aged twenty-one, was attacked with intense headache and vomiting on December 21st, 1874, at the very time when Dr Jackson's patient had just entered the London Hospital. Some months previously she had been stunned by a severe blow from a stone on her right temple. On the 24th there was well-marked double optic neuritis, her pulse was irregular and only 52 in the minute, and she had no fever. Afterwards she had paralysis of both sixth nerves, suffocative attacks in which she could hardly breathe, constipation, transient hemiplegia, delirium, and an affection of the speech, so that her mother could not understand her. But between the 6th and the 14th January, 1875, all her symptoms began rapidly to subside, and before long her impaired sight seemed to be the only thing that troubled her, except that she was unable to take solid food without vomiting.

the greater the chance that the lesion is some other form of tumour than tubercle; and above the age of forty the latter is very unlikely. To distinguish between tubercle and glioma or sarcoma is, however, of little practical consequence; what is really important is that one should never overlook *syphilis*. It is particularly to be noted that in many instances in which gummata are developed in the brain no nodes upon the bones can be discovered, nor any indications of past or present orchitis, iritis, or cutaneous eruptions. In only three or four of the ten fatal cases of this kind recorded by the author was it ascertained during life that the patient had suffered from any venereal disease. Dr Buzzard lays stress on the presence of a muddy complexion, and other signs of cachexia not traceable to any definite visceral disease, as pointing to syphilis. It is well known that in most cases the pains caused by this disease regularly recur, or at least undergo a marked aggravation in the evening. What seems to have more weight than anything else in suggesting syphilis as the cause of obscure nervous symptoms is the impossibility of referring them to any single lesion. Dr Jackson long ago insisted on the value of this principle, and Dr Buzzard has illustrated it by cases in point. Thus two of his patients had paralysis of one arm and of both legs at the same time; the inference was that there was an affection of the spinal cord as well as of the brain. Still secondary tumours are often multiple, and the presence of a primary new growth elsewhere is very apt to be overlooked. Thus a patient coming to a physician for headache and paralysis may very likely never mention that there has for years been a tumour in the breast, or that one testicle was excised some months before. Again, the original seat of the malignant disease may be some internal organ, where such an affection is difficult of detection. Out of sixteen cases at Guy's Hospital of secondary growths in the brain there were no less than six in which the starting-point of the mischief was a sarcoma surrounding the root of one lung, and in five of them this was not discovered during life.

Prognosis.—All that we know of the progress of cases of cerebral tumour would tend to show that they are inevitably fatal.*

Even when the local lesion in the brain is a syphilitic gumma the prognosis is not very favourable, so far as concerns the patient's ultimate restoration to health. For a time active treatment is generally followed

* The nearest approach to a recovery that I remember to have heard of occurred in a boy, aged fourteen, who was under my care in Guy's Hospital in 1867. About two years previously he had been taken ill with "pain in the back of the head, loss of sight, and fits in which he used to clench his hands." On May 1st, when he had been in my ward for three weeks, it is noted that "he lies apparently unconscious of everything, and cannot be roused. His head is constantly thrown backwards; and when he is touched there is a sort of opisthotonos. He is completely amaurotic; his pupils are equal and slightly dilated." On May 4th the report is, "He is slightly more conscious, he recognises his mother, and will raise his hand into the air when told to do so. His evacuations are passed involuntarily." During the next three months the chief change was that he slowly wasted away, until he was reduced to a mere skeleton; the only sign of intelligence that he ever manifested consisted in lifting his hand, as already described. But one day, in going round the ward, I spoke to him; and to the astonishment of everyone he slowly articulated a few words in reply. From that moment he began to improve. He took food well, regained flesh, talked more and more every day, got up, walked about the ward, and at last was discharged perfectly well except that he was blind. Afterwards he attended among my out-patients. I have no notes of his symptoms during this period, but I remember that he complained of paroxysmal headache, and of epileptiform fits. He was readmitted, and died. The notes of the autopsy have unfortunately been mislaid; but I recollect that there was an irregular calcareous mass, of about the size of a marble, embedded in the floor of the third ventricle, with some mucoid fluid round it.—C. H. F.

by very striking results; consciousness is regained, paralysis is recovered from, headache subsides, epileptiform convulsions cease to recur. But after an interval the symptoms return; perhaps the opposite limbs are now paralysed, or the affection may assume a paraplegic instead of a hemiplegic form. A second course of medicine may again be successful, but at last our efforts are baffled, and the patient succumbs. Such cases are often prolonged over a period of several years. On the other hand, it sometimes happens that the cure is permanent; and more often that each return is milder than the last, until the disease gradually wears itself out.

Treatment.—In the treatment of cerebral gummata one should not trust wholly to iodide of potassium, though it is desirable to give it at first, and often in very large doses, for its action is more rapid than that of mercury. But afterwards a mercurial course should always be prescribed. The bichloride may be given internally, or recourse may be had to calomel-vapour baths or to inunctions with blue ointment.

Such a line of practice often proves brilliantly successful, even when there was no proof that the symptoms were due to a syphilitic lesion. The question then arises whether the proof is supplied by the result of the treatment.

The palliation of symptoms is always possible. It there be epileptiform convulsions, full doses of bromide of potassium should be given. The same remedy will often relieve headache or giddiness. Or it may be necessary to administer opium or morphia by the mouth, or to inject the alkaloid subcutaneously. Dr Reynolds speaks highly of Indian hemp as sometimes altogether removing pain in the head. He also recommends the local application of ice.

The recent achievements of antiseptic surgery, and the power of localisation which we have seen that experiment and observation have now conferred on the physician, have led to the bold attempt to remove a cerebral tumour by trephining and enucleation. Mr Victor Horsley has published a remarkable series of not less than ten cases in which the seat of a cortical lesion was diagnosed and its removal accomplished ('Brit. Med. Journ.,' April 23rd, 1887). In half of these cases the morbid condition was not a new growth, but some inflammatory or degenerative change in the brain or meninges. However, in one it proved to be a tubercular tumour, in a second a glioma, in a third a large Pacchionian body, in a fourth a tumour weighing 4½ oz., and in a fifth a tubercular tumour. In the last of these cases the operation was fatal, in the second the glioma returned and proved fatal six months afterwards; in the three others the patient recovered from the operation, and the convulsions were rendered less frequent or the pain was removed. Mr MacEwen, of Glasgow, has also had successful cases, some of them as early as 1883 (see his address, *ibid.* August 11th, 1888).

It must be confessed that only a small number of cerebral tumours would be amenable to surgical treatment even if we could always accurately determine their situation. Many are too deeply seated, others are multiple; others not sufficiently circumscribed, others of malignant nature, and others only come under notice too late for treatment. The syphilitic growths, moreover, may be better cured by drugs.

A hundred cases from the records of Guy's Hospital were reviewed, with reference to the possibility of operation, by Dr Hale White ('Guy's Hosp.

Rep.,' vol. xliii, p. 117). He concluded that not more than three tubercular tumours, not more than four or five gliomata, and only one sarcoma, two cysts, one myxoma, and two tumours of doubtful origin—in all ten certainly, and four probably, of the 100—were removeable by operation, provided that their exact seat had been previously ascertained.

CEREBRAL ABSCESS.—This disease, though so different pathologically from a cerebral tumour, resembles it in its clinical characters, and hence is for practical purposes treated of here.

It is not a common malady, but is very dangerous, and affords a most instructive example of the unforeseen practical results which follow from thorough scientific investigation of diseases.

Ætiology.—The most important fact which was established by Sir William Gull in his papers on cerebral abscess in the 'Guy's Reports' for 1857 is that this affection is never primary nor idiopathic; it is due either to general pyæmia, or to injury or disease of the skull involving local purulent infection.

Its most frequent origin is from *diseases of the ear*, especially suppurative inflammation of the tympanum. Otorrhœa, if prolonged, always involves the risk of extension of mischief through the bone to the internal surface of the skull. Hence, no one with a chronic discharge from the ear should be accepted for life assurance at ordinary rates. In the 'Medical Times and Gazette' for 1863 a case is recorded of a patient who for several years had a discharge from the ear, but who lived to the age of sixty-six, and then died of a cerebral abscess; and it often happens that an aural affection which had been present from early childhood kills a grown-up man or woman. In many instances the several stages of the morbid process are plainly traceable after death. The tympanic cavity is found to be bare and carious, or necrotic; the dura mater over its roof is raised from the bone by pus, or it is softened and sloughing; the arachnoid and pia mater are adherent at this point, and close to it an abscess is found in the temporo-sphenoidal lobe. Or, the caries may pass from the mastoid sinuses or from the petrous bone to the posterior fossa of the basis cranii, and then the abscess occupies the corresponding half of the cerebellum. Among eighteen successive cases at Guy's Hospital there were twelve in which the temporal lobe of the cerebrum was the seat of the abscess, three in which it lay in the cerebellum, two in the centrum ovale, and one in the pons.

The late Mr Toynbee believed that abscess of the cerebrum usually follows from caries of the tympanum, abscess of the cerebellum from phlebitis of the lateral sinus, and abscess of the bulb from caries of the labyrinth. The third situation is too rare to be taken into account; but Gull, and subsequent German writers, confirm Toynbee's statement of the connection between disease of the middle ear and abscess of the cerebrum, and between the disease of the mastoid sinuses and abscess of the cerebellum.

According to Huguenin and Meyer, the right side of the encephalon is much more apt than the left to be affected with abscess from disease of the ear; and, among twenty-four cases collected by Gull and Sutton, the right side was affected in no less than eighteen. But it is curious that of the eighteen cases that have since occurred at Guy's Hospital, in nine the abscess was on the left side, and in nine on the right.

Aural surgeons formerly laid stress on the diagnosis of caries of the

tympanum, as indicating the danger of extension to the brain, and they did not hesitate to pass a probe in search for rough and denuded bone. But apart from the risk of breaking through the wall, and so of setting up the very mischief which is dreaded, experience has shown that the detection of caries is less important than was supposed. In many instances of cerebral abscess set up by inflammation of the middle ear, the bone is found after death to be healthy. There is more than one route by which the morbid process may reach the brain. It may pass along the bony canals which transmit the superficial petrosal and other veins, or through the spaces in the diploë which convey vessels from the tympanum to the dura mater. A curious circumstance is that in many instances there is a tract of apparently healthy cerebral substance between the wall of the abscess and the petrous bone.

When the suppuration of the ear assumes a putrid character the danger of meningitis or cerebral abscess is the greatest. The suppression of the discharge from the external meatus and a sudden increase of pain in the ear are bad indications, for they denote swelling of the mucous membrane and increased tension in the tympanum. But in some cases there is no pain at all: there may even be no otorrhœa, for the secretion may be retained behind a perfect membrana tympani; and thus, unless one carefully tests the patient's hearing, we may easily overlook the fact that he has anything the matter with his ear.

Another, but a far less frequent cause of abscess of the brain, is chronic *disease of the nose.* Two such instances were recorded by Gull in the 'Guy's Hospital Reports' for 1857. Each patient had suffered from a discharge from the nostrils; in one case the abscess was in the middle lobe, in the other in the anterior lobe. Other writers have given cases in which a *nasal polypus* was the starting-point of the mischief. The author once made an autopsy in which an ulcerating *epithelioma* of the lip and cheek extended to the base of the skull along the third division of the fifth nerve, and set up an abscess in the middle lobe of the brain. Necrosis or *caries of the calvaria* from any cause, if attended with sloughing of the dura mater, may have a like effect. Another source of suppurative meningitis or of cerebral abscess is *carbuncle* of the face, particularly of the upper lip.

General *pyæmia* sometimes leads to the formation of one or more abscesses in the brain, as in other parts of the body. In records of examinations at Guy's Hospital (during the same period within which occurred the eighteen cases above mentioned of cerebral abscess from disease of the ear) there were nine instances of abscess as a part of general pyæmic infection.

There is a curious group of cases of cerebral abscess, which are secondary to pre-existent suppurative inflammation in a remote part of the body, but in which there are at no period any symptoms of blood-poisoning, and in which no pyæmic abscesses are found anywhere but in the brain. Sir William Gull first showed the ætiology of such cases, although a similar instance had before been recorded by Abercrombie. The most remarkable point is that the *lung* is generally the seat of the primary lesion which leads to the abscess in the brain. At Guy's Hospital within a few years we had six cases of this kind; six others are given by Gull and Sutton in the second volume of 'Reynolds' System of Medicine,' and some have been recorded in Germany by Biermer, Huguenin, and Meyer. The supposition has generally been that the cerebral inflammation is set up by a portion of thrombus washed out of a pulmonary vein, and

carried to the brain in the blood; and Böttcher is said to have found in the floor of an abscess of the brain, which was secondary to a pulmonary abscess, some pigment which he was able to identify as having come from the lung. The nature of the thoracic disease has varied in different instances. Adding the cases which have recently occurred at Guy's Hospital to those related by Gull and Sutton, we obtain a series of twelve examples of this form of abscess of the brain. Among them there are three in which the primary affection was empyema (cf. 'Path. Trans.,' vol. xxviii, p. 4), two of tubercular phthisis, two of some form of acute pneumonia, one of simple bronchitis; in each of the remaining four it seems to have been pulmonary cirrhosis (chronic interstitial pneumonia), with dilatation of the bronchial tubes, or a sloughing cavity in the indurated tissue. Huguenin places bronchiectasis with stagnant putrid secretion at the head of the list of pulmonary diseases which give rise to cerebral abscess. It is important to note that the mischief in the lung may be altogether latent. In one of the above cases Dr Moxon says that he almost despaired of finding a primary lesion, until at last he discovered that the mucous membrane of the right bronchus was extensively ulcerated, with its cartilages exposed and necrosed.

We must remember that general pyæmia may have an internal source: as ulcerative endocarditis or acuto osteomyelitis. Either of these sources of infection may lead to abscess of the brain with or without meningitis.

Another cause of suppuration within the substance of the brain is direct *injury to the head*, as from a fall or blow. Generally the skull is fractured, and serious symptoms are present from the time of the accident until death releases the patient from his sufferings. Such cases come under the care of the surgeon, and do not require further mention here; but in exceptional instances the fact that the head has been injured may be overlooked, or the accident may have occurred some time previously, and been forgotten; and thus the physician may find himself in attendance.

Sir William Gull records the case of a boy, aged sixteen, who was in 1844 taken into Guy's Hospital for what appeared to be slight fever. After he had become convalescent he was attacked with cerebral symptoms, and ultimately fell into a comatose state. His friends then for the first time mentioned that three weeks before his admission he had been stunned for a minute or two by falling backwards from a cart, so as to strike his head upon the ground. There was neither wound nor bruise, but it seems that he complained of nearly constant pain in the head up to the time of his coming into the hospital. He died about two weeks after the accident; and a large abscess was found in the left hemisphere. There was also an abscess in the situation of the sphenoidal sinuses beneath the carious olivary process of the sphenoid bone. Could this have been caused by the fall? and, if so, was it not the cause of the cerebral abscess?

Another case occurred to Dr Tuke ('Med. Times and Gaz.,' 1861, i, p. 196). It is that of a man, aged forty-seven, who died after a week's illness with cerebral symptoms. A few months previously he had fallen from a ladder and struck his head, without any severe symptoms immediately following. Dr Tuke believed that the accident was the cause of the cerebral symptoms; but it should be noted that, although the patient had suffered from "chest symptoms" a year previously, there is no mention of the state of the lungs at the autopsy.

One would be the more ready to attribute a cerebral abscess, for which we could find no other cause, to an injury of the skull that occurred

within a year or two previously, because it appears that there is scarcely a case to be met with in which one is absolutely driven to admit that the abscess is primary and spontaneous. Among seventy-six cases collected by Gull and Sutton for the 'System of Medicine' there are, indeed, a few in which no cause was found; but the only one of which it can be said with certainty that the autopsy was complete is the last of the series, that of a man who died in Guy's Hospital in 1863, and whose body was examined by Dr Wilks. He was employed at a music-hall, and some years before his chest had been crushed in an accident, but he was not known to have injured his skull. However, he had led an irregular life, and might easily have received and forgotten such an injury.

Of 100 cases collected by Mr Barker ('Lancet,' June 11th, 1887), 29 were due to disease of the ear, 27 were traumatic, 20 were associated with suppuration in the lungs or liver, and 7 were due to general pyæmia.

In his recent 'Gulstonian Lectures' (March, 1890) Dr Pitt has recorded 56 cases of cerebral abscess from the records of Guy's Hospital, and of these 18 were due to disease of the ear, 1 to disease of the nose, 10 to injury of the skull, and 3 more to cranial periostitis or caries; 3 to invasion of tumours of the skull, 8 to primary disease of the lung, 9 to general pyæmia, and in 4 no certain origin could be ascertained.

Cerebral abscess, consecutive to disease of the ear, is about equally frequent in males and in females; of the cases secondary to chronic pulmonary affections the larger proportion seem to occur in men.

The great majority of cases, from whatever cause, are met with in persons between fifteen and thirty years old.

Of Dr Pitt's 56 cases, only 4 of non-traumatic origin occurred in children under ten, and only 9 in patients above forty.

Anatomy.—Abscesses of the brain arising from different causes are to some extent different. Those which result from injury are almost always solitary, and so are those which are secondary to affections of the ear or nose. Out of twenty-seven cases due to aural disease, collected by Gull and Sutton, there are only two in which more than one abscess was present; in one the cerebellum contained three abscesses, in the other there was one in the cerebellum, as well as one in the cerebrum. On the other hand, fourteen cases of general pyæmia, in which the brain was the seat of suppuration, yield eight in which the abscesses were multiple, and in most of them there were four, five, or more in different parts of the cerebral substance. So, again, in seven out of eleven cases consecutive to affections of the lungs, the abscess was multiple. The presence of numerous centres of suppuration may therefore go far towards determining the real origin of an abscess in an autopsy, and this might be of juridical as well as of pathological interest.

The cerebral abscess caused by disease of the ear or nose is generally ill-defined, its wall shreddy, and surrounded by softened cerebral substance; but sometimes it is enclosed in a thick capsule. In pyæmia the abscesses seem to be very rarely circumscribed; but in Dr Moxon's case, which lasted eighteen months, the limiting membrane was so firm that it could be lifted out of the brain-tissue in which it lay. When the affection is secondary to disease of the chest there is almost always a well-marked capsule. Sir William Gull stated that the cyst wall is made up to a great extent of spindle-cells. Rindfleisch has since shown that they constitute its middle layer, there being outside them a plane of fibrous tissue, while

the cavity is lined with embryonic tissue, the superficial cells of which are opaque and yellow from fatty degeneration. Even in abscesses of recent formation the pus is commonly greenish and viscid, although it has an acid reaction; but in those of long standing it is often as thick as mucus, of a bright green colour, and alkaline. It may be odourless, or have a nauseous sickly smell, or (when arising by extension from bone disease with necrosis) be horribly fœtid. Mixed with the pus corpuscles is much granular matter and fat, probably derived from the brain-tissue replaced by the abscess; and in very old cases one can hardly recognise any pus-cells; they have undergone complete degeneration, and nothing is left but débris.

It is only in cases arising from injury that the commencement of the cerebral suppuration can be fixed with sufficient accuracy to decide as to the length of time required for the production of a capsule. The evidence collected by Lebert and Meyer on this point goes to show that by the end of the third week the abscess cavity may be found circumscribed, but that a definite membranous cyst wall is not found before the sixth or seventh week, and sometimes not until a far longer period has elapsed. Gull observes that this might become a medico-legal question in some cases of a man dying of cerebral abscess after receiving a blow, if an interval had elapsed so that no direct clinical connection could be traced between the supposed cause and the effect. He cites one instance in which the absence of a limiting membrane was taken as proof that the disease could not have been the result of a severe fall eighteen months previously; and another, in which its presence showed that the suppuration in the brain was not due to an attack of smallpox which occurred within the last three or four weeks before death. Unencapsuled abscess may form in seven days.

An abscess of the brain, when solitary, generally reaches a considerable size before it destroys the patient. It is frequently as large as a hen's egg, and sometimes larger. It often reaches close to the surface of the hemisphere, and is said sometimes to break through beneath the pia mater. It is also said that pus from an abscess in the brain may be discharged through the auditory meatus producing "cerebral otorrhœa." So, again, an abscess set up by caries in the nose may break through the ethmoid bone into the nostrils. Usually a large abscess within the hemisphere pushes inwards towards the lateral ventricle. At Guy's Hospital this was found in four or five cases of suppuration following caries in the ear; and it is by no means infrequent in cases which are secondary to empyema or bronchiectasis. The pus may be found collected in one of the cornua, into which it has fallen by gravitation, or the whole of both lateral ventricles may be full of pus, with the ependyma intensely inflamed, thickened, grey, and velvety; the morbid action may even spread through the third and fourth ventricles to the subarachnoid spaces of the brain and spinal cord.

Symptoms.—The symptoms of abscess of the brain vary widely in different cases. It has been said that there may be absolutely none, and the affection be accidentally found after the death of the patient from some other cause. This, however, may be justly doubted. In the large majority of cases, symptoms are present for a much shorter period than that during which the abscess in the brain must have been forming; in other words, the disease is commonly latent during part of its course. This latent period may be only a week or a fortnight, but occasionally it extends to several months. In cases of general pyæmia, the occurrence of suppuration in the

brain may be masked under the delirium and stupor which so often accompany severe blood-poisoning.

The earliest symptom of cerebral abscess is, as a rule, *pain*. This varies greatly in severity. In cases secondary to otorrhœa it is the most agonising, in cases of pyæmia least so. Gull and Sutton speak of one patient as continuously holding his head with both his hands, and of another as walking about with his hands pressed against one side of his head, and calling out, "Oh my head! oh my head!" Another could not help screaming; and although perfectly sensible, would tear and bite anybody or anything near him, at the same time expressing contrition for what he was doing. The pain is generally continuous; but it is sometimes intermittent, especially at first, and is usually increased by drink or by violent movements. Anstie mentions the case of a boy who for three months complained of no symptom whatever, except of a pain which came on in attacks very closely resembling those of migraine, not oftener than once in ten days or a fortnight, and lasting for some hours at a time, nearly always ending in vomiting, and disappearing after sleep. In some instances the seat of the pain corresponds closely with that of the abscess; Gull and Sutton record the case of a boy who had almost constantly a burning pain over the front and right side of the head, and in whom the disease was in the anterior lobe of the right hemisphere. But they go on to speak of a patient who had an abscess in the cerebellum with pain in the forehead, and of another who complained of the left side of his head, but had an abscess in the right middle lobe.

Next to pain, *vomiting* is the most important symptom—occurring frequently, without relation to the ingestion of food, and without any symptoms of gastric irritation.

Much less frequently an *epileptiform seizure* forms the starting-point of the symptoms; and such seizures may be repeated at intervals for a considerable period, before any further sign of illness manifests itself. Or the first thing noticed may be a dulness of expression, a change of disposition from cheerful to morose or melancholy, a disinclination to speak, loss of memory, or inability to sleep. Gull lays stress on rapidly increasing emaciation as having been a principal symptom in some cases. *Rigors* may occur, sometimes with such regularity that the case might be mistaken for one of ague. The pulse is sometimes slow; Huguenin relates a case in which it fell to fifteen and even to ten in the minute.

The *temperature* is often normal or subnormal, rarely raised unless there is meningitis or pyæmia as a complication. In a case under the writer's care in January, 1886, the patient, a girl about twenty, had a temperature of 106°, with symptoms of pyæmia and basal meningitis. There were found, beside suppuration of the liver and other organs, a large abscess in the left hemisphere, which had opened into the ventricle, a second in the lower part of the ascending parietal gyrus, and a third in the præcuneus. There had been no motor paralysis.

The *pupils* are often sluggish or unequal, sometimes dilated. The *optic discs* are sometimes seen with the ophthalmoscope to be "choked," or œdematous, or in a state of acute optic neuritis. In a case of the writer's this was much more marked on the side of the abscess, but this is exceptional; indeed, the fundus in several cases of cerebral abscess has been normal. Dr Pitt found optic neuritis more common from thrombosis of the sinuses.

Localising symptoms.—In the symptoms hitherto mentioned there has been little or nothing to indicate that one part of the encephalon rather than another is the seat of disease. But in exceptional cases "localising" symptoms are not wanting. Aphasia may be present, which shows that the abscess is in the back part of the left frontal lobe. Much more frequent is hemiplegia, partial or complete. This indicates that the part affected is near the fissure of Rolando; or that the inflammatory process extends inwards, so as to involve the internal capsule or corpus striatum. We have already cited a case of Hitzig's in which spasms confined to certain muscles were set up by a small abscess limited to a small part of the cortex. Sir William Gull has recorded the case of a gentleman who on many occasions had a sudden convulsive affection of his right arm—so violent that he had to support himself by holding on to the table with his other hand, and yet so devoid of pain that he was amused by it; after death a large abscess was found in the occipital lobe.

Tenderness on percussion of the skull may occasionally be elicited, and may be a valuable localising symptom.

Huguenin thinks that the rupture of an abscess into the ventricle may sometimes be recognised by the supervention of spasms in both sides of the face, or in both legs, without loss of consciousness; and he gives cases in which these symptoms were present.

It seems to be very doubtful whether there are any localising symptoms peculiar to abscess of the cerebellum. Huguenin says that the pain is not only referred to the occiput, but often extends down the back of the neck. He also states that the pupils of both eyes are commonly dilated, that vomiting is peculiarly severe, and that the gait is sometimes unsteady, like that of a person affected with locomotor ataxy. In one case which occurred at Guy's Hospital the patient was so feeble as to be hardly able to sit up in bed; but in that instance (as in many other cases of cerebellar abscess) the lateral ventricles of the cerebrum contained a large excess of fluid, a result, in all probability, of pressure upon the veins of Galen.

Huguenin finds that about one fifth of the total cases of cerebral abscess are situated in the frontal lobe, one eighth in the occipital, one fifth in the temporo-sphenoidal, a very few in the parietal, fewer still in the pons or bulb, and most of the remainder in the cerebellum.

In Dr Pitt's series from Guy's Hospital, abscesses from all causes were found 18 times in the frontal lobes, 14 in the temporo-sphenoidal, 6 in the occipital, 5 in the parietal, 8 in the cerebellum, and once in the pons.

Mode of death.—None of the symptoms which have been described are such as would suggest to an unskilled observer that the patient's life is in imminent danger. But after they have been present for several days or even weeks—or, sometimes, without any warning symptoms whatever—he falls into a state which is obviously most alarming. Violent delirium may set in, followed by stupor and coma; or a severe epileptiform fit may occur, after which he may never regain his consciousness. In 1876 a girl, aged eighteen, was admitted into the Clinical Ward one afternoon at five o'clock. She had for six days been suffering from sickness and diarrhœa, with severe headache, so that she was said by the medical man who attended her to have typhoid fever. She then spoke rationally and answered the questions that were put to her, but seemed rather strange in her manner. At eight o'clock the same evening she suddenly made a great noise; the house physician was hastily summoned, and found her partially

insensible, but capable of being roused so far as to say that she was going to die. She seemed to have loss of power in her left arm and leg. An hour later she all at once ceased to breathe. Artificial respiration was kept up, and the heart continued for some little time to beat rapidly, but it soon slackened, and in ten minutes she was dead. We found four or five abscesses in the posterior and middle lobes of the right hemisphere.

The final stage of a cerebral abscess is seldom of long duration; but it may last for a week, and in exceptional cases longer still, during which time the patient is alternately delirious and in a state of stupor, and passes his evacuations under him. Sometimes there is a transient recovery from such symptoms, followed by their return, and by speedy death.

Diagnosis.—That the diagnosis of abscess of the brain is often beset with difficulties must be sufficiently apparent from what has been said of its symptoms and of the conditions under which it occurs. In cases of supposed ague, of hysteria, of enteric fever, and of neuralgia, it is needful to bear in mind cerebral abscess as possible. Between abscess and other organic cerebral diseases, particularly tumour and meningitis, there is no positive criterion.

In practice we recognise abscess of the brain not so much by its symptoms as by our pathological knowledge of the causes to which it is commonly due. Since it seldom, if ever, arises spontaneously, this would give us a good opportunity of diagnosis, but for the fact that both the patient and his friends too often deny the existence of these several causes, otorrhœa, deafness, bronchial and pleuritic disorders, and other sources of pyæmia. Moreover, all the affections that may set up suppuration in the brain are also liable to give rise to other forms of cerebral disease. Thus injury is not infrequently followed by meningeal apoplexy. Again, we can seldom speak with certainty of cerebral abscess as being consecutive to any pulmonary affection, unless we are in a position to exclude the possibility of its being phthisis; for that disease is sometimes accompanied by solitary tubercle of the brain, and very often sets up tubercular meningitis, which is itself a very insidious malady, and attended with the most varied symptoms. Lastly, disease of the ear may either be the starting-point of general meningitis, or may cause thrombosis of the lateral sinus (cf. *infra*, pp. 648, 676).

The *prognosis* of cerebral abscess is fatal, and its only possible *treatment* is surgical.

Mr Hilton used to quote in his lectures a case of Dupuytren's, in which he trephined the skull, expecting to find pus under the bone; he then incised the dura mater, and finally thrust a bistoury into the brain, gave exit to a quantity of pus, and cured the patient. Similar cases have occasionally been recorded since. In 'Ziemssen's Cyclopædia' Huguenin mentions twenty-six cases of supposed success in opening abscesses of the brain. The operation was a much more desperate one before the methods of antiseptic surgery were adopted.

When the abscess results from otorrhœa the mastoid bone has been trephined and the pus successfully evacuated.

In a patient under the writer's care in Guy's Hospital in July, 1879, the symptoms and previous history pointed strongly to an abscess in the anterior parietal region of the left side. Mr Lucas trephined, opened the dura mater, and incised the brain, but without result. No harm was done, and the symptoms of pressure were somewhat relieved; but when death occurred a few days later we found a focus of red softening with infiltrated pus in the corona radiata, close to the part operated on.

In March, 1890, a girl of about twenty was admitted into Mary Ward with otorrhœa, severe headache, vomiting, low temperature, and acute optic neuritis, particularly on the side of the diseased ear. Mr Jacobson trephined the skull and opened an abscess in the temporo-sphenoidal lobe, which, after a second operation, ceased to discharge. The symptoms have disappeared and the patient is convalescent.

With increased means of accurate localisation and advancing surgical experience, we may confidently expect that the number of cases of cerebral abscess successfully treated by operation will increase.

There are now many recent cases of successful surgical treatment of this otherwise fatal disease: among them may be mentioned those of Dr Truckenbrodt in 1886, of Mr Barker (1886 and 1888), Mr MacEwen (1887, who reports twelve cases in all), Mr Horsley (1888, who reports eleven), Prof. Bergmann (who reports four cases from Berlin), Sir Wm. Stokes (a successful case from Dublin), and several from the United States.

RED SOFTENING.—*Ramollissement rouge.* — The morbid condition so named is not very common, and is one of the most curious to which the brain is liable. It is attended with great swelling; convolutions so affected are broader than natural, and the sulci between them deeper; and the corpus striatum or thalamus is rounded, prominent, and generally increased in size. As is implied by the name, there is a marked diminution of the natural firm consistence, and the colour is altered; the cortex assumes a deep purple tint, and the white substance becomes pink or red, with numerous minute ecchymoses. Under the microscope the capillary and other vessels are found dilated and engorged; blood-corpuscles are seen among the nervous tissues, and if not very recently extravasated they are found fused together into shapeless masses. According to Rindfleisch, pus-cells are collected about the smaller blood-vessels. Moxon, however, says that he has often been surprised to find how slight were the histological changes in some cases of this kind: the elements of the tissue were softened and granular, but they still retained their form, and no characteristic inflammatory products could be recognised.

Most pathologists speak of suppuration of the brain as a further stage of red softening. It is true that in cases of pyæmia one sometimes finds reddened patches apparently antecedent to the formation of the pus; indeed, a similar condition is now and then seen in the neighbourhood of an abscess. In the latter case, however, it might fairly be regarded as the result of a distinct morbid process affecting the brain-tissue round the wall of the abscess, just as it may affect that which surrounds a tumour or an apoplectic clot. Moreover, when several suppurating cavities are found in the same hemisphere—however small and recent they may be—the intervening cerebral substance is commonly pale and of firm consistence. Lastly, the causes of red softening are different from those of abscess.

One of these causes is injury. Some years ago a woman died in Guy's Hospital, who had fallen three or four months previously, and struck her head against the wall. Three weeks before her death she had a fit, which was followed by partial left hemiplegia, affecting the side of the face and tongue, and accompanied with ptosis. At the autopsy, all the parts at the base of the brain were found by Dr Wilks to be in a state of softening, "partly red and partly white;" one third of the thickness of the pons was so affected, and nearly the whole of the crus cerebri.

Again, red softening may accompany an inflammation of the membranes, as tubercular meningitis. We have already spoken of it as sometimes arising in the neighbourhood of an apoplectic clot (p. 557), round a tumour (p. 559), or in a part of the brain deprived of its blood-supply by thrombosis or embolism (p. 554).

It is a question still undecided whether red softening of the brain ever occurs as a primary and independent morbid process. In the records of examinations at Guy's Hospital there are very few cases that could be so interpreted. In two instances the occipital lobe of the left hemisphere of the brain, and a considerable part of the cerebellum on the same side, presented the change in question; but in each of them the cardiac valves showed recent vegetations, so that it seems probable that the affection was dependent upon embolism of some of the smaller arteries.

In 1876 the author made an autopsy in which parts of the right superior and middle frontal convolutions were swollen, soft, and of a grey-pinkish colour. There was no caseation, and no definite edge to suggest the presence of a new growth; so that the lesion would have been regarded as primary acute inflammation had not the microscope revealed the presence of a large number of oval and round cells infiltrated between the nerve-fibres. It was a diffused glioma. We must therefore suspect any case of supposed primary red softening, unless a careful examination of every part of the affected structures has been made.

Some years ago a woman, aged twenty-six, died in the hospital, after an illness of three months' duration. The falx cerebri was found adherent to the anterior parts of both hemispheres by granulation-tissue. On section the frontal lobes appeared of a brick-red colour; their cineritious substance was swollen, and the boundary-line between it and the white matter was ill defined. On the left side this red colour extended down to the lateral ventricle. The affected parts were rather harder than natural; but in all other respects the disease corresponded perfectly with the descriptions which writers have given of local inflammation of the brain; and unless there was a new growth which was overlooked, it must have been of that nature. The microscope only showed corpuscular infiltration, with compound granule-masses.

Huguenin, who doubts the existence of "spontaneous encephalitis," nevertheless describes various retrograde changes as consecutive to red softening. He says that the patches may either subside entirely, or pass into a condition of yellow softening with cavities full of serum, or undergo cicatrisation, or become converted into tough, dirty-white, indurated masses. It is difficult to tell on what evidence we could conclude that the conditions in question had followed red softening rather than an effusion of blood or obstruction of an artery.

DISEASES OF THE MENINGES AND VENTRICLES OF THE BRAIN

> Continuo auditæ voces, vagitus et ingens,
> Infantumque animæ flentes in limine primo
> Quos dulcis vitæ exsortes . . .
> Abstulit atra dies et funere mersit acerbo.—VIRGIL.

TUBERCULAR MENINGITIS—*History—Morbid anatomy—Relation of the tubercles to the inflammation—Their occasional absence in basal meningitis—Ætiology—Sex and age—Onset—Course—Later and final stages—Tubercular meningitis in adults—Diagnosis—Prognosis.*

Acute non-tubercular Meningitis—Anatomy—Varied causes—Secondary and idiopathic—Symptoms and course—Diagnosis—Prognosis.

Epidemic Meningitis—History—Morbid Anatomy—Symptoms and course—Varieties, complications, and sequelæ—Ætiology—Diagnosis.

Treatment of meningitis generally.

HYDROCEPHALUS—*In children—Origin—Pathology and anatomy—Effect on the skull—Symptoms, course, and event—Treatment—Adult Hydrocephalus.*

Adhesive thrombosis of the cerebral sinuses—Infective thrombosis of the sinuses.

Pachymeningitis hæmorrhagica.

AMONG the most frequent and the most fatal of all cerebral diseases are those which depend on inflammation of the meninges.

It was once usual to describe separately "inflammation of the dura mater" (*pachymeningitis*), "arachnitis," and inflammation of the pia mater (*leptomeningitis*). It is true that various diseases of the bones of the skull—such as, for instance, caries of the petrosal—may give rise to ulceration or sloughing of the corresponding part of the dura mater. But that local affection requires no special notice; it is only preliminary to diffused inflammation of the pia mater, or to a cerebral abscess; and upon these conditions all the clinical and pathological interest of the case is centred. Again, there is not any disease that can be said to be strictly "arachnitis." Pus or purulent lymph is sometimes found in large quantity within the arachnoid cavity; and its presence or absence constitutes one of the distinctions between two different forms of meningitis. But the limitation of suppuration to a supposed serous sac without implication of the pia mater is unknown to the surgeon as a result of any kind of injury to the skull; and certainly the physician never meets with it. If the arachnoid were perfectly analogous to the pleura or the peritoneum, we should expect it to be liable to serous inflammation attended with the exudation of lymph and serum, like the other great divisions of the pleuro-peritoneal space. But no such anatomical analogy exists: the "parietal layer of the arachnoid" is the endothelial lining of the dura mater; and the "visceral layer" is nothing but an outermost condensed stratum of the pia mater. The arachnoid cavity is now sometimes called the subdural space.

The following diseases are all forms of *meningitis*, *i. e.* inflammation of

the pia mater, or *leptomeningitis*. They all are seated mainly in the loose tissue of the pia mater, and are all diffuse, not focal nor disseminated; but they differ much in intensity, and still more in their causes.

TUBERCULAR MENINGITIS.*—This is the most frequent of all diseases of the nervous centres, but notwithstanding its frequency and the fact that it has been known for more than a century, its real pathology has been recognised only during the last sixty years. The first detailed account of its symptoms was published in 1768 by Dr Robert Whytt (a man of eminence in his day at Edinburgh) under the name of dropsy of the brain; and in the early part of the present century it became generally known as "acute hydrocephalus"—a designation which was retained by Sir Thomas Watson, as recently as 1857, as being "so established, both among medical men and with the public, that he could not venture to propose any change." He was, however, well aware of the fact, first pointed out by Papavoine in 1830, that the essential morbid change is the presence of tubercles in the membranes, and that the fluid within the ventricles is of quite subordinate importance. Since inflammatory products are also present, the name of tubercular meningitis, suggested by Bricheteau, in 1814, is obviously appropriate, and has now met with universal acceptance.

Morbid anatomy.—The appearances found after death vary considerably in different cases, and in different parts of the brain. As a rule, the most marked lesions are found at the base. The diamond-shaped space bounded by the optic tracts and the crura cerebri is filled with gelatinous or puriform lymph, which also envelops the great arteries arising from the circle of Willis and their branches. The inflammatory process may extend along the Sylvian fissures until it occupies a great part of the convex surface of the brain. Generally, however, the surface of the hemispheres shows no obvious changes; the sulci may be reduced in size as the effect of pressure. A large quantity of lymph is often spread out over the pons and bulb, and it may be traceable in the subarachnoid space as far as the lower end of the spinal cord. Almost invariably there is a yellowish patch upon the upper surface of the cerebellum, close to the opening of the veins of Galen; and sometimes the velum interpositum and choroid plexuses are infiltrated and thickened. The fluid within the ventricles is increased in quantity and is generally rather turbid. Its specific gravity may be raised to 1010; it yields a more or less distinct coagulum on boiling, and exhibits leucocytes under the microscope. The ependyma is often granular. The adjacent parts of the brain are usually much softened. The fornix and septum lucidum may be almost diffluent, and even the great basal ganglia may have their consistence so much reduced that they fall into a shapeless pulp as soon as they are removed from the skull. This "white softening" appears to be due to a mere disintegration of the tissue, for no exudation cells are discoverable. It is certainly not a mere result of *post-mortem* maceration.

In addition to acute basal meningitis, more or less numerous tubercles are to be found. If they are but few, they are most easily recognised in the Sylvian fissures or in the folds of pia mater dipping into the sulci; they appear as minute grey dots, adherent to the smaller arteries or to delicate filaments of connective tissue. When they are abundant, they may become fused together so as to ensheath an artery in a granular mass. Some are

* *Synonyms.*—Acute Hydrocephalus—Basal meningitis—Idiopathic infantile cerebro-meningitis—Leptomeningitis tuberculosa.

generally found caseating, and their opaque yellowish colour enables them to be more easily recognised. Cheesy aggregations of considerable size may be found embedded in puriform lymph. If the tubercles are present in very large numbers, they may be seen thickly scattered as milk-white spots beneath the visceral arachnoid, especially on the under surfaces of the cerebral lobes. They may also be developed in the form of very minute transparent granules upon the inner aspect of the dura mater lining the fossæ of the skull, and (according to Huguenin) between the two layers of that membrane, close to small twigs of the middle meningeal artery. In the vertebral canal they are found both on the smooth side of the spinal dura mater and on the visceral arachnoid. Within the ventricles they occur only in the choroid plexuses. They sometimes grow from the processes of the pia mater which pass down between the convolutions, appearing as whitish-yellow streaks along the small arterial branches in the cortex. Dr Gee mentions a case in which the capillary vessels throughout one hemisphere were studded with miliary tubercles, which remained after the softened cerebral substance had been all washed away.

In one instance the author found one hemisphere affected on its convexity with tubercular meningitis, while the left side of the brain and its base seemed to be entirely free. Huguenin describes two similar cases in which the affection was limited to the territory of a Sylvian artery; and Dr Pitt records others ('Brit. Med. Journ.,' 1890, i, p. 772).

The meninges covering the bulb and cord are often affected as well as those of the brain.

Pathology.—Tubercular meningitis very seldom occurs as an independent malady. In almost every instance it is either secondary to some chronic tubercular disease, for which the patient is already under treatment, or else it is only part of a general acute tuberculosis. Often it is both consecutive to a local chronic lesion, and also associated with the simultaneous development of recent tubercles throughout the body.

Of one hundred and twenty-four cases of tubercular meningitis at Guy's Hospital, sixty-five occurred under the age of twenty; fifty-nine between twenty-one and sixty. Among the former there were thirteen in which the cerebral affection was secondary to disease of the hip-joint or spine, or some other malady capable of clinical recognition; among the latter there were twenty-eight such cases, mostly pulmonary phthisis. In eighty-six of the one hundred and twenty-four cases there were miliary tubercles in the viscera or in the serous membranes. In only nine or ten cases is it distinctly stated that recent tubercles were present in the pia mater and nowhere else.

An interesting question concerns the relation of the tubercles to the lymph and other associated products of inflammation. The opinion expressed by Huguenin is that the miliary granulations are first developed, and that the pia mater tolerates their presence for a time, but that they afterwards excite inflammatory reaction. Rilliet had before put forth this doctrine. But, as Wilks and Moxon argued, if such a view were correct, one ought sometimes to discover meningeal tubercles in small quantities, unmixed with inflammatory products, in those cases in which acute tuberculosis destroys life by invading the lungs or other organs, and to find it unattended with the characteristic cerebral symptoms.

The author met with two instances of this kind. One occurred in a woman, aged thirty-two, who was admitted with an abdominal tumour that turned out to be the omentum indurated by tubercle. She was attacked

by hemiplegia, coma, ptosis, and delirium, and died in seven days. At the autopsy the brain and its membranes looked healthy, except for a single minute granule on a fold dipping into one of the sulci, and a little filmy material round one Sylvian artery. So slight was this change that if it had been observable on both sides it would have been passed by. But the microscope showed that even the apparently healthy artery of the opposite side, which had been set apart for the sake of comparison, had a distinct growth of tubercle about it; and the one white granule in the pia mater was already caseating in the centre.

The other instance is that of a man, aged fifty, who became suddenly unconscious on May 2nd, 1876, and was brought to the hospital in a state of coma, with stertor and right hemiplegia. He afterwards partially recovered, and on May 5th he was sensible enough to answer questions that were put to him; but in the following night he was attacked with another fit, of which he died in twelve hours. We found tubercles thickly scattered about the Sylvian arteries and in the adjacent parts of the pia mater, but without any lymph being present; both lungs contained many grey clusters. In that case it is probable that the tubercles had been formed more slowly than usual, for it was stated that a fit had occurred twenty-two days before the man's death, and that afterwards he was always drowsy and stupid.*

About a year later this very question was raised in a trial for murder. Harriet Staunton, the wife of one of the prisoners, had died in a state of neglect and emaciation, which led to suspicions that she had been starved or poisoned. The medical men who made the autopsy gave their opinion that death had been due to deprivation of food, and yet stated that they had discovered in the membranes of the brain bodies which they believed to be tubercles, but to which they attached no importance. What were the exact appearances must remain uncertain, for one report spoke of "small patches of rough, millet-seed-like deposit in the meshes of the pia mater," and another of "a small recent patch of tubercular deposit upon the arachnoid membrane on the upper part of one hemisphere, about the size of a fourpenny piece." The husband and three other persons were convicted of murder. But before the time fixed for their execution the leading pathologists of London addressed a memorial to the Home Secretary, and the lives of the prisoners were spared. If there really were tubercles in the meninges, the *immediate* cause of the woman's death was disease, and not starvation or chronic poison.

The reader may perhaps be disposed to doubt whether tubercles that are only discovered by the aid of the microscope are really so important. But just as in a case of concussion of the brain the presence of obvious ecchymoses of one or two convolutions (which cannot be regarded as themselves the cause of death) is nevertheless of the highest significance, so it would seem that the development of even the smallest tubercles in the pia mater is a sign of invisible changes in the cerebral tissues that are incompatible with the maintenance of life. For obviously meningitis cannot produce cerebral symptoms except by affecting the supply of blood to the brain.

Occasionally we can see obvious lesions in the brain as well as in the pia. Rindfleisch speaks of the superficial layer of the cortex—patches of which often remain sticking to the pia mater when it is stripped off—as infiltrated with leucocytes, and Huguenin says that inflammatory products may even be

* It ought to be added that he was a gouty subject, and had Bright's disease of the kidneys, so that one was obliged to take into consideration the question of uræmia.

found in the white substance of the hemisphere. Indeed, some one part of the brain is now and then found in a state of well-marked yellow or red softening. Five instances of this have occurred at Guy's Hospital, and others are mentioned by Rindfleisch and Huguenin. Sometimes the softened part has been the island of Reil on one side, sometimes the frontal or temporo-sphenoidal lobe. In one case Dr Goodhart could detect no granule-masses in the softened parts, but "the tissue was very fatty and granular, and the nerve-fibres seemed to have undergone destruction, scarcely any of them being visible."

Basal meningitis without tubercles.—In cases in which the symptoms and course have been altogether like those of tubercular meningitis, the membranes at the base of the brain and in the Sylvian fissures are sometimes found after death with well-marked signs of inflammation, but no evident tubercles. Six or seven cases of this kind are to be found in the records of Guy's Hospital, but in all of them, with one exception, the lungs (and often other viscera as well) contained miliary tubercles; in the one exceptional case the bronchial glands were caseous. A sufficiently careful microscopic examination might perhaps have cleared up the difficulty, but the pulmonary lesion was conclusive as to the real nature of the disease.

There is, however, a form of acute basal meningitis in which neither tubercles nor tubercular changes are to be found in any part of the body. This disease is described by Huguenin as *Leptomeningitis infantum*. He relates as a typical case that of a female child, aged eleven months, who died on the fourth day of an attack of measles, attended with convulsions and other cerebral symptoms. Flattening of the convolutions, injection of the choroid plexuses, distension of the ventricles, and softening of the central parts were the only obvious morbid appearances; but the microscope showed that leucocytes were abundantly present in the pia mater.

A striking instance occurred at Guy's Hospital in 1859, in the practice of the late Dr G. H. Barlow. A boy, aged nine and a half years, died after an illness of twelve days' duration, which began with intense headache and ran its course with convulsions, grinding of the teeth, strabismus, and coma. That the disease was tubercular meningitis was doubted by no one who saw the child, and when the skull was opened the brain looked flattened, as if by effusion; but, except that its tissue was soft and that there was a slight increase of fluid in the ventricles, no morbid changes were discovered. There were no tubercles in other organs.

Several similar cases have since been recorded at Guy's Hospital and elsewhere. Clinically, they are indistinguishable from tubercular meningitis of the base; but the prognosis is better, and their real pathology doubtful.

Ætiology.—The causes of tubercular meningitis are those of tubercular affections in general. Impure air, want of exercise, scanty food, and inherited predisposition, are its most important predisposing causes; it often attacks in succession several children of the same parents at about the same ages. When this is the case one is apt to suppose that there must be a special morbid susceptibility of the nervous centres, but it is to be borne in mind that, although clinically the cerebral symptoms mask all others, yet the disease is almost always a general tuberculosis. The exciting cause is the entrance of Koch's bacilli into the circulation.

Over-study, mental shocks, intoxication, or blows upon the head have each been supposed to give rise to tubercular meningitis. A policeman in one of our wards attributed his illness to over-fatigue in attending a review

in Hyde Park in June, 1860, but afterwards admitted that he had before complained of pain in the head. Three children have died in Guy's Hospital in whom the exciting cause of the same disease was attributed to a blow or fall upon the head.

All these "causes" are, there is little doubt, as imaginary as the pretended causes of syphilitic eruptions; but they are worth recording in order to warn us against accepting coincidences for causes in other diseases.

Sex and age.—Tubercular meningitis is much more frequent in males than in females. At Guy's Hospital the proportion has been as eighty to thirty-seven, and it does not seem to have varied very much at different periods of life, although Huguenin says that in children the preponderance of males is much more marked than in adults.

The relative frequency with which persons of different ages are attacked is not yet accurately known. There are no hospitals to which children and adults are brought in numbers corresponding with their ratios to the population as a whole; and until *post-mortem* examinations become universal, the Registrar-General's 'Reports' will fail to show the liability of grown persons to tubercular meningitis. It has always been known to be much less common in infants under two years than in older children, and we now see that this is due to their less chance of tubercular infection. Guersant met with one case in an infant only six weeks old.

Three cases have occurred at Guy's Hospital since 1854 in infants aged six months, ten months, and one year respectively. Of children between two and four years there have been fourteen cases; between five and seven and a half, nine cases; between eight and ten, twelve cases; between eleven and fifteen, eleven cases; between sixteen and twenty, sixteen cases —altogether sixty-five patients under twenty years old. Of adults between one-and-twenty and thirty there have been thirty-one cases; between thirty-one and forty, fourteen cases; between forty-one and fifty, eleven cases; between fifty-one and fifty-six, three cases—altogether fifty-nine patients above twenty years of age.

Clinical course.—The symptoms of tubercular meningitis are not essentially different in patients at different ages, but as the best writers have based their descriptions mainly upon observations of cases occurring at an early period of life, and as such cases present certain minor clinical peculiarities, we will begin with the disease as it is seen in *children*.

Premonitory stage.—In the first place, before any definite indications of cerebral mischief develop themselves, a general failure of health is often observed, which may last for several weeks or even for two or three months. The symptoms which are manifested during this period are termed "premonitory" or "prodromal." In the well-known work of Rilliet and Barthez on the diseases of children an admirable sketch is given, which has afforded materials for all subsequent writers. Probably these symptoms may be due to two different causes, sometimes to a scanty early formation of tubercles in the lungs or other organs, which is afterwards followed by a more abundant crop; sometimes to the slow progress of chronic caseation of the mesenteric or bronchial glands. Foremost among them is emaciation; the limbs waste and lose their roundness, the ribs and the muscles feel soft, are too easily felt and even seen, and the skin is lax and flabby. The cheeks often retain their plumpness of outline, so that the loss of flesh is first observed by the nurse who dresses and undresses the child; but the face becomes pale, the eyes are dull, and there is a want of animation in the

countenance. The appetite is diminished or capricious. The bowels are disordered, being generally constipated, but with intervals of diarrhœa; the evacuations may be pale and offensive. The disposition and the temper often become changed. The child is dull, apathetic, and slow in his movements; he is easily fatigued by lessons, and quickly tired of toys. Headache is sometimes present, or the child complains of being sleepy and tired and wanting to lie down. At night he is restless, lying with the eyes half closed, rousing at the slightest noise, and unable to sleep with a candle in the room; he often grinds his teeth, and starts or cries in his dreams. Whether fever is commonly present at this stage is doubtful, and slight pyrexia in a child might be caused by trifling gastric disorder.

Premonitory symptoms do not always occur, but Dr Gee says that among twenty-six cases collected by him there were only two in which they were not noticed. Their duration is very variable, sometimes not more than a fortnight but generally longer, and now and then even as much as four or six months. They may subside, and the child's health appear to improve, before the disease breaks out in a characteristic form.

Onset and early symptoms.—In marked contrast to the uncertain length of the prodroma, the subsequent course of the disease is seldom prolonged beyond twenty-one days from the occurrence of the invasion. An apparent exception to this rule is in cases of one or more tubercular tumours developing in the brain before the meningeal affection had begun.

The symptom that first excites alarm is most often repeated *vomiting*. Sometimes the child is sick only when it takes food, sometimes it brings up bilious matters even though it may have swallowed nothing. The sickness generally lasts for two or three days only, but it may go on for a week. If it once ceases for twenty-four hours it seldom returns. In some cases there is no vomiting for the first day or two, and occasionally it is altogether absent.

The initial symptom next in frequency is a *convulsion*, more or less completely epileptiform in character. In one instance mentioned by Dr Gee there was general rigidity which recurred several times; in another the attack took the form of temporary unconsciousness. In a single one of his twenty-five cases the invasion was marked neither by vomiting nor by convulsions, but by increase of the headache, drowsiness, and wasting which had existed during the premonitory stage.

Severe *headache* is now almost invariably present; the child keeps its hands pressed against the forehead, or may go on rubbing the scalp, first in one place, then in another. Trousseau lays stress on a peculiar inarticulate "hydrocephalic cry," which is sometimes heard from the very beginning of the disease, sometimes only towards the end; he describes it as a single, sharp, loud sound, like that of a person exposed to some sudden danger; it may be repeated every hour, or every five minutes, for several days together. We may often hear the hydrocephalic cry without hydrocephalus, but the symptom is marked and characteristic enough to deserve mention. For several days there may be no marked impairment of the intelligence; the child perhaps continues to talk rationally, but his answers may be rather slow, and his memory and power of perception somewhat impaired. He is apt to lie in a drowsy state, half asleep, with his eyes staring vacantly, or he may go on talking to himself, or repeating some phrase over and over again, or singing, whistling, and shouting.

The *pupils* are often sluggish, and very commonly one is larger than the

other. A tendency to squint is a frequent early symptom. Trousseau relates a case in which there was transient hemiopia; the child was sitting near a window when he called out, "Oh, mamma, look at that little boy, he has only half a blouse and half a face!"

Such are, as a rule, the symptoms of tubercular meningitis for perhaps eight days after its invasion. During this period there is more or less fever, the evening temperature rising perhaps to 102° or 103°, while in the mornings it may be 101° or 100°, or not above normal. The pulse may be a little quickened, or natural, or slower than natural; sometimes easily made more rapid by the slightest excitement. The frequency of the respirations is but little altered. Constipation of the bowels is almost always present, and the tongue is often but not always furred.

Later stage.—When about eight days have passed, the condition of the little patient undergoes a change, now and then almost sudden, but more often gradual. Its most striking feature is loss of consciousness; and the period which follows has been called the stage of cerebral pressure, in distinction from the preceding stage of cerebral irritation. The child now ceases to take notice of anything that goes on in the room. He often lies on one side, curled up with the knees drawn close to the abdomen, and the hands folded over the pudenda. Sometimes the head is drawn backwards, and the muscles of the nape of the neck may then be felt to be hard and rigid. He may keep grinding his teeth every few minutes, making a disagreeable jarring noise. The pupils now become dilated and insensible. One or more of the cranial nerves may be paralysed; the third, for instance, so that the eyelid drops; or more rarely the facial nerve. There may be loss of power in the limbs. Tickling the soles of both feet may cause only one leg to be drawn up. The evacuations are often passed into the bed.

The order in which the various symptoms make their appearance is uncertain; some of them may begin during the earlier period of the disease, before coma sets in. This is particularly the case with certain characteristic changes in the patient's aspect: a frown upon the brows, and deep lines around the nose and mouth; injection of one cheek, or of both. If the countenance is pale, it is liable to flush when the child is disturbed, or when anything is given it to drink. So, also, any part of the body which has been pressed upon shows a marked injection of its capillary blood-vessels. A particular instance of this was made into a leading symptom of tubercular meningitis by Trousseau, under the name of the *tache cérébrale*. He pointed out that if one draws one's finger-nail gently over the patient's thigh, or abdomen, or face, a bright red line is produced, and that this differs from the effect of an equally slight scratch in a healthy person by appearing earlier (within thirty seconds), by lasting longer (eight, ten, or fifteen minutes), and by being broader and of a deeper colour.

The *ophthalmoscope* may show ischæmia of the optic discs, or descending neuro-retinitis, or tubercles in the choroid. Dr Allbutt found some affection of the retina in twenty-nine out of thirty-eight cases.

Another symptom which may be observed in most cases at this period is retraction of the abdominal walls. Vomiting is generally absent, and the bowels may still remain obstinately constipated; but for some unknown reason the intestines no longer contain the usual quantity of gas; and the belly therefore becomes deeply hollowed, the rib-cartilages, the iliac crests, and the pubic symphysis appearing unduly prominent.

The *tongue* may now be red and dry, but it often still remains moist.

The *temperature* seldom rises above 101°; Dr Gee remarks that it may for days together remain at between 96° and 98°. Thus, if the case is advanced when the child is first brought to one, the disease may appear altogether non-febrile. The *pulse* during this period is generally infrequent, 60 or even 50 per minute; it is still apt to be irregular and unequal in force. A similar irregularity and inequality of the *respiration* are very commonly present; the child perhaps breathes rapidly three or four times in succession, and then the chest may remain motionless for some little time. Trousseau laid great stress on this symptom, and has recorded a case in which the breath was held for as long as fifty-seven seconds. Typical Cheyne-Stokes respiration is sometimes observed.

Final stage.—The "stage of pressure" may continue with but little alteration until it terminates in the patient's death, which is often preceded by a convulsive seizure. But in certain cases the symptoms during the last twenty-four or forty-eight hours are to some extent peculiar. One remarkable change is that there may be a brief return of consciousness for a short time before the end. Dr West relates how a girl, aged seven, who had been in a state of stupor for six days, and profoundly comatose for two days, became conscious, swallowed some drink, spoke sensibly, and said she knew her father; in the course of an hour and a half, however, she became worse again, and a little later she died. The pulse often becomes rapid during the last two or three days; and, as Dr Gee points out, the temperature may steadily rise until it is above 107°. But in some cases the beats of the heart remain infrequent up to the time of death; and the temperature even in the rectum may fall, until it is very low indeed. In one case, three days before the fatal termination, the thermometer registered 97·8° and 96°; next day the highest temperature was 96·2°, the lowest 93°; the day after they were 82·8° and 82·1° respectively; and on the day of death 80·5° and 79·4°. In other cases the face and limbs are livid and cold, and covered with a clammy sweat, while the thermometer shows that fever is still present. Towards the last a peculiar fœtid earthy smell is often perceptible; and it is strange that Rilliet and Barthez, after alluding to Whytt having mentioned this symptom, go on to say that they have not themselves recognised it.

Symptoms in adults.—The onset of tubercular meningitis in adults is seldom preceded by marked prodromal symptoms. It is often secondary to phthisis, and when this is the case the pulmonary affection is rightly regarded as the cause of any general failure of health that may have been noticed. Then, again, few grown-up persons are watched as carefully as children are by their parents and nurses; and in adult life loss of flesh may be due to so many other causes that tubercular meningitis is very unlikely to be thought of.*

The invasion is less often marked by vomiting in adults than in children; and an epileptiform seizure is still less frequent.

But although in adults the disease generally begins gradually and insidiously, yet we have had at Guy's Hospital more than one case in which the sudden occurrence of convulsions has been the precursor of a fatal

* I must state it as my impression that most of the patients whom I have seen have been fairly well nourished at the time of death. I have no recollection of having ever observed a very marked degree of wasting, unless when it was obviously referable to some co-existing visceral disease; and in the case of Harriet Staunton, already referred to at p. 637, I should have been reluctant to regard the extreme emaciation, which must have been going on for several weeks, as dependent upon the presence of meningeal tubercles.—C. H. F.

termination within a day or two, and in which an autopsy has shown that the disease was tubercular meningitis. It is not at all uncommon for death to take place after an illness of twenty-four or forty-eight hours only. In 1868 a man, aged thirty-two, a patient of Dr Wilks, died within two days of having been about his business as a draper; on admission he was so restless that he had to be held down in bed, but he quickly became comatose. In another case there was violent delirium two days before death.

In some cases of tubercular meningitis in adults the first symptom is well-marked *local paralysis*, which is extremely rare in children. In 1871 a woman, discharged from a surgical ward with advanced phthisis, came the same morning to seek readmission as a medical patient. No symptoms of cerebral disease had been observed, but she had paralysis of the left facial nerve, which must have developed within a few hours. She was admitted into the Clinical Ward; and was intelligent enough to be able to say that she could not hear the ticking of a watch with the left ear. But she soon became drowsy, then comatose, and at the end of six days she died. At the autopsy no special affection of the facial nerve could be found either in the petrous bone or elsewhere.*

Lastly, tubercular meningitis in adults may have *hemiplegia* for its earliest, and even for its principal, symptom. Three instances of this have been observed at Guy's Hospital within the last few years. One patient was a man aged thirty-three, who was admitted on account of an abdominal tumour which proved afterwards to be a tuberculous omentum. Some days before his death he was attacked with loss of power in the left arm and leg; he was sensible to the last. In the other two patients the right limbs were affected, and, as the superficial seat of the lesion might have led one to anticipate, the paralysis was accompanied by well-marked aphasia. One of them was a woman, aged twenty-six, who came under Dr Wilks's care in 1867; until the real nature of the affection was revealed at the autopsy there was not the slightest suspicion of its being due to anything but disease of the Sylvian artery. The other, also a woman, aged forty-one, had been attending as an out-patient for phthisis, when she was seized with right hemiplegia and loss of speech; afterwards she became semi-delirious and her paralysis changed sides. In none of these three cases was any well-marked change found in Broca's convolution or in the adjacent parts of the brain; but in a boy aged nine, in whom right hemiplegia and aphasia were combined with the more ordinary symptoms of tubercular meningitis, there was reported red softening of the left third frontal, and of the inner ends of the two left ascending (or central) gyri. Huguenin relates three instances in which paralysis of the right limbs and loss of speech were the chief symptoms; in two of them many tubercles were present in the left Sylvian fissure, but none in the opposite one, and in the third case the pia mater in the left fissure was more thickened than anywhere else. In one of the cases in which there was red softening of the left island of Reil and Broca's

* Huguenin has attempted to trace the affections of special cranial nerves occurring in tubercular meningitis to definite local changes. In one instance, in which the face was paralysed, he found the portio dura thinned, of a yellow colour, and covered with a large quantity of pus; cellular elements were present in numbers in the nerve-sheath, but the fibres appeared to be intact. In another similar case, however, no change could be found in the nerve. In one instance, in which there had been convergent strabismus, the abducens was greatly diseased, diffuse suppuration having occurred between its fibres. Similar appearances were twice discovered in the third nerve, when the muscles supplied by it had been paralysed during life; but in some other cases, in which the same symptoms had been incompletely developed, the nerve seemed to be normal.

convolution, the first symptom after headache was speechlessness, but this lasted only about seven minutes, and two days afterwards the patient (a man aged fifty) was able to go to business as usual. One can only suppose that the aphasia was due to functional disturbance of Broca's region.

The observations made at Guy's Hospital seem to show that, when there are no inflammatory changes in addition to the tubercles, there is usually no persistent coma. Violent delirium is sometimes associated with the presence of numerous tubercles between the convolutions of the upper surface of the brain. Choked discs are probably an indication of a general increase of pressure within the cranial cavity, whereas neuro-retinitis points more definitely to inflammation at the base of the brain, in the neighbourhood of the optic tracts or nerves. The extension of meningitis to the spinal canal probably gives rise to rigidity of the muscles of the neck, which in children at any rate is very common; for this is one of the chief symptoms of an epidemic form of meningitis which affects the spinal as well as the cerebral membranes; and sometimes the occurrence of painful cramps or spasms in the limbs may be attributed to the same cause.

Diagnosis.—This may be very easy or very difficult, either in children or adults. During the first few days of a case which is to last two or three weeks one is often unable to speak positively, but there is seldom much uncertainty when its fatal character is once fully developed.

All writers lay stress on the importance of distinguishing it from *enteric fever*, and the fact is indisputable that many cases, which for a week or ten days are supposed to be examples of that disease, afterwards become attended with such well-marked cerebral symptoms that the diagnosis is forthwith altered to that of tubercular meningitis. Thus Rilliet and Barthez describe a special form as having a *début typhoide*. But although to the clinical observer the meningeal affection masks all the other features of the case, yet the pathologist almost always finds it to be only a part of general miliary tuberculosis, which in reality constituted the patient's illness from its commencement, and to which the early febrile symptoms were due. In our reports at Guy's Hospital two instances are recorded in which the first diagnosis was that of fever, and in which tubercles were found in the meninges and nowhere else. Sometimes the opposite error is committed, enteric fever being attended with strabismus and irregularity of pupils, vomiting, and constipation, as well as with headache, delirium, and coma, so as to be taken for meningitis. The *tache cérébrale* of Trousseau affords little help in doubtful cases.

The clinical value of ophthalmoscopic changes in the optic discs is still uncertain. A normal state of the retinæ is no proof of the absence of tubercular meningitis, and the positive significance of ischæmia, or even of retinitis, is not yet established. One appearance, indeed, is conclusive, namely, the presence of tubercles in the choroid. It is true that they belong, not to the meningeal affection itself, but rather to general acute tuberculosis, but this fact in no degree diminishes their diagnostic importance; for whenever there are tubercles in the membranes the case always assumes clinically the aspect of a cerebral disease, even though they may be far more numerous elsewhere. In fact, in all cases of suspected tubercular meningitis one should carefully search the lungs for the very slight indications of acute tuberculosis which they sometimes yield, and examine the testes, the lymphatic glands, and other organs, for chronic lesions of the same nature.

In a patient fifty years of age who was brought comatose into John

Ward in April, 1875, the writer found evidence of phthisis, and this led to a correct diagnosis.

A condition that formerly was often mistaken for tubercular meningitis, is one, not infrequent among young children, which has been rendered classical by the descriptions given of it by Marshall Hall (1825), Abercrombie (1828), and Gooch (1829). The first of these writers called it "*the hydrocephaloid disease*;" Sir Thomas Watson gave it the name of "spurious hydrocephalus;" and Dr Gooch stated that he had "invariably found it attributed to and treated as congestion or inflammation of the brain." At the present day a skilled practitioner is not likely to fall into the error of diagnosis which sixty years ago was so frequent; and at any rate the mistake is not now likely to be attended with any serious results. Among the chief causes of the affection was one to which children are no longer liable, namely, venesection. A case related by Marshall Hall is that of a girl under three years old, to whom sixteen leeches had been applied for an attack of influenza; and he states that all his patients were in a state of exhaustion before they were attacked by the cerebral symptoms; many of them had had protracted diarrhœa after weaning. On the other hand, Dr Gooch says that in most of his cases there had been no previous illness; but perhaps he scarcely appreciated the gradual effects of insufficient or improper food.

The child's aspect is characteristic; it lies on its nurse's lap, unwilling to raise its head, drowsy or even comatose, with sunken, half-closed eyes, dilated insensible pupils, irregular and sighing respiration. The face is pale, and the skin cold. Dr Gooch, indeed, speaks of a slight and transient flush as being sometimes present, and Dr Hall of an early stage in which the little patient is irritable, restless, feverish; but such symptoms very seldom, if ever, last long enough to mislead a careful observer into the diagnosis of meningitis. Depression of the fontanelle is an important symptom of exhaustion in children, although it seems formerly to have escaped notice. The proper treatment is to give ammonia and brandy, but, above all, to take care that suitable food is supplied. For an infant recently weaned a wet-nurse should generally be procured; and a return to the breast is often advisable, even when the child is some months old, and has long been fed with the bottle. At least it ought to have asses' milk, or goats' milk, if cows' milk should appear not to be readily digested. To prescribe leeches and calomel would probably be fatal.

At Guy's Hospital we have had cases, supposed during life to be examples of *mania*, *delirium tremens*, or *epilepsy*, in which the disease has turned out to be tubercular meningitis. Huguenin remarks upon the possibility of mistaking this disease for *uræmia*, when a chronic renal affection, in reality tuberculous, is attributed to morbus Brightii. A curious case occurred some years ago at Guy's Hospital, that of a painter, who came saying that he had "lead colic," and that his bowels had not been open for a fortnight. In the evening after his admission he had convulsions and became insensible, and on the next day he died. There was caseous disease of two of the lower dorsal vertebræ, with a large abscess in front of the spine; and the membranes of the brain and cord showed the characteristic appearance of tubercular meningitis. The same disease has been found on *post-mortem* examination in two cases of boys admitted into the surgical wards for symptoms supposed due to vesical calculi; no cause could be discovered for the irritability of the bladder from which they had been suffering before any obvious signs of cerebral mischief appeared. A very similar case

occurred in a woman who was taken into a medical ward for vesical symptoms; but in addition to tuberculosis of the pia mater there was a tumour in the spinal cord.

The diagnosis between tubercular meningitis and other *organic affections of the brain*, particularly abscess and tumour, is often difficult and sometimes impossible. Probably not a few cases in England have been recorded as examples of sporadic *cerebro-spinal fever*, which have really been tubercular. It is of practical importance to remember the possibility that syphilis may be the cause of the most varied cerebral symptoms.

In adults, however, the most serious error of all is to mistake tubercular meningitis for *hysteria*. Many such mistakes have occurred, and sometimes with lamentable results. A woman, aged twenty-five, had long been "odd in her mind and scarcely to be trusted;" she was attending as an out-patient, with hysterical symptoms, when she sought admission to Guy's Hospital, and died three days later. There were several yellow tubercles in the brain, as well as the usual appearances of tubercular meningitis.

Prognosis.—Tubercular meningitis is one of the most fatal of all diseases. Rilliet has recorded a single instance of recovery, in which the child died five and a half years afterwards of a second attack, and at the autopsy the remains of the former disease at the base of the brain were clearly recognised; and one similar case is mentioned by Trousseau. The late Dr Carrington recorded a third and a most conclusive case. But such instances are almost without parallel.

It is, however, by no means very rare for recovery to take place in cases in which tubercular meningitis had been diagnosed more or less positively, and for the nature of the disease to remain uncertain, as it must necessarily remain in such cases, unless an accident should lead to an autopsy being made at a future time. How closely a case which terminates in recovery may resemble tubercular meningitis is strikingly illustrated by a clinical history which we owe to Dr West. A child, aged three years and a half, a member of a phthisical family, was attacked by a disease which ran the ordinary course of acute hydrocephalus, unchecked by the customary treatment. Convulsions took place, coma succeeded them, deglutition was very difficult, the pupils were dilated and almost motionless, the pulse was very feeble and very frequent, and everything portended a speedy death. A younger brother had died a year before of the same disease. Food was still given, as the power of swallowing was not entirely lost, and ammonia and ether were administered, and after a time quinine. For days the child remained unconscious, but at length she began to raise her hands to steady the cup that was put to her lips. Next she recovered her sight, after some weeks she became able to speak, and after many months she began to walk with a tottering step. Three years afterwards, although her intellect was not defective, she still had a vacant smile, and had never regained flesh, nor recovered the look of health; her gait also remained unsteady. Whatever the real nature of this case may have been, one cannot be wrong in learning from it not to make an absolutely fatal prognosis in what appears to be a case of tubercular meningitis. However confident we may be in our diagnosis, and however threatening may be the symptoms, our opinion should be given with some reserve.

The *treatment* of tubercular meningitis will be discussed when the other forms of inflammation of the pia mater have been described.

ACUTE SIMPLE MENINGITIS.—Some modern writers describe several forms of acute non-tuberculous inflammation of the membranes of the brain under distinct names, according as the convexity or the base is affected. Undoubtedly a case in which the whole surface of the brain is covered with a thick layer of pus is attended with much more violent symptoms and destroys life far more quickly than one which is limited to a particular region, and in which only a small quantity of lymph is effused. But no complete division into separate categories is possible.

Anatomy.—When the most intense degree of inflammation has been present, one finds, on turning back the dura mater, that the cerebral convolutions are completely hidden by a green purulent material. This looks as if it were spread out over the hemisphere in the *subdural* space; but by scraping the arachnoid surface one generally discovers that little, if any, of it is really free. Moxon says that even when some of the exudation had appeared to come off upon the edge of the scalpel, he often found only epithelium and detritus on examining it with the microscope. Sometimes, however, a considerable quantity of pus is subdural, but only when the disease was due to extension from without. The converse, however, is very far from being true; namely, that meningitis is always attended with exudation into the "arachnoid cavity," when it is set up by such causes as caries of the petrous bone or necrosis of the calvaria. For in the great majority of these cases no subdural pus is found; and whenever it is widely diffused over the hemispheres it is also abundant in the meshes of the pia mater.

The pia mater may be swollen to many times its normal thickness, appearing either soft, green, and gelatinous, or firm yellow and felt-like, according to the consistence of the exudation infiltrated into its substance. And from such a condition there may be every gradation, down to a point at which only slight traces of pus can be discovered along some of the principal vessels, or at the base, or in one Sylvian fissure, or on a single lobe of the cerebellum.

In some of the more severe cases the convolutions themselves are softened. When the pia mater is stripped off it carries with it portions of the cerebral tissue, leaving a ragged surface behind. Huguenin says that numerous leucocytes may be found throughout all the layers of the cortex, and that suppuration of the brain-substance may actually reach such a point as to give rise to a diffused yellow-grey maceration visible to the naked eye. The vessels often contain but little blood in consequence of the increased pressure which precedes death. The ventricles may either be empty or contain a turbid liquid, or even pus. The choroid plexuses are sometimes infiltrated with inflammatory products.

Some of the sinuses in the dura mater are now and then found plugged with *ante-mortem* clots, even when the meningitis is not secondary to any disease of the bones which could itself have set up the thrombosis by extension. In a little girl aged three, for example, who died in Guy's Hospital, and in whom both hemispheres were covered with a thick layer of yellow lymph, each lateral sinus, but especially the left, was filled with a greenish softening material.

How often a primary acute meningitis of the brain spreads to the membranes of the spinal cord it is impossible to say. In the majority of recorded cases the vertebral canal has not been opened, but we have had at Guy's Hospital at least eleven cases in which inflammatory products have been found in greater or less quantity beneath the spinal arachnoid. In

none of them was there any reason to believe that the disease was of epidemic origin; in four it was directly caused by fracture of the skull or severe injury to the brain, or arose by extension of mischief from the cranial bones.

Ætiology.—The causes of simple acute meningitis are various. Most frequent among them are *injuries to the head.* These generally come under the notice of the surgeon, but the physician must remember that the disease sometimes follows a blow or fall which may not have produced any external bruise, and about which the patient may say nothing, particularly if he should have been intoxicated at the time. Some years ago a man was admitted into Guy's Hospital for a fractured thigh, caused by his having fallen into a cellar while drunk. He died at the end of five weeks, having been delirious at the time. He was believed to have delirium tremens; nothing was known of any injury to the head; he had been able to get out of bed and stand upright. At the autopsy it was found that there was general acute meningitis affecting the base as well as the surface; a small part of one parietal bone, over an area an inch in diameter, was of a greenish colour, and its diploë was reddened. Both the periosteum and the dura mater seemed quite healthy, but it was thought probable that the bone had been injured at the time of the accident. Another common cause of meningitis is the extension of inflammation from *chronic disease of the skull.* Syphilitic caries or necrosis of the calvaria sometimes kills in that way, and such a result may be altogether unexpected until a very short time before his death.

Six cases of meningitis due to *affections of the ear* have been observed at Guy's Hospital within the last few years (1880), a smaller number than that of the cases in which cerebral abscesses were due to the same cause.* One patient, a man aged twenty-two, was said to have had sunstroke seven days before being attacked, and another man, aged twenty, had been struck on the head with a bolster immediately before dangerous cerebral symptoms set in. In each instance it was clear that the inflammation started from a diseased temporal bone, but there was a question whether an independent "exciting" cause was not in operation (see a case of the writer's, 'Path. Trans.,' vol. xxxix, p. 228). In the case of a woman, aged twenty-seven, no history could be obtained; and no cause for the meningitis of which she died was discoverable; so the ears were specially examined by the late Mr Hinton, and he found pus in the labyrinth on one side. To this accordingly the disease was attributed, but it must be remembered that in epidemic cerebro-spinal fever inflammation often spreads to the ears from the membranes, and there seems to be no reason why the same thing should not occur in the simple form of the disease.

Numerous other local affections may be mentioned as occasionally giving rise to meningitis. *Local pyæmia* from carbuncle of the face may cause it, as in a case which the writer saw many years ago in a girl about seventeen. In two cases an epithelioma, commencing in the lip, extended with ulceration along the pterygoid region until it passed through the foramen ovale into the interior of the skull. In another, a little girl had necrosis of the upper jaw after measles, and the cavernous sinus was full of a dirty-brown fluid. Or the disease may be set up by suppuration of the eyeball, travelling, in all probability, along the sheath of the optic nerve. Trousseau relates a case

* Dr Pitt also has since found that meningitis without abscess or thrombosis of a sinus is the least frequent of the results of disease of the ear (1890).

in which its starting-point was a chronic affection of the first two cervical vertebræ.

Next in frequency to the cases of meningitis that are traceable to local causes come those which are secondary to some other acute disease. More careful research may hereafter show that these also have a *local* starting-point in some lesion affecting the cerebral vessels.

At Guy's Hospital we have had two cases in which inflammation of the pia mater accompanied an attack of *erysipelas* of the scalp; in one it is noted that the calvaria was discoloured yellow. In four instances it appeared to be part of general *pyæmia;* once there was a grey patch of cerebritis reaching the surface; once pericarditis also was present, so that one might regard the disease as specially attacking the serous membranes if the arachnoid could still be called by that name; once the meningitis was the only evidence of blood-poisoning, but such seemed to be the most probable explanation of its occurrence, as it came on six days after an operation for imperforate anus in a child a year old; once it was associated with an abscess in the lung, and with suppuration in the mediastinal connective tissue and in the substance of one leg, there being, however, no obvious primary lesion. In a single case inflammation of the cerebral membranes was believed to be a complication of *typhus;* no affection of the vessels in the pia mater was discovered, but the spleen contained infarcts, and there was thrombosis of the renal veins. Four times it seemed to be secondary to acute *pneumonia* or pleurisy: all but one of these cases, however, presented peculiar features. One occurred in a man, aged twenty-two, who had been in the hospital six weeks for pleuro-pneumonia, and was convalescent and able to go into the grounds when he was again attacked with dyspnœa and high fever, and died in three days; the spinal membranes, as well as those of the brain, were inflamed. Another was in a woman, aged twenty-seven, who had been ill for a week with pleurisy and then was attacked with an acute diphtheritic affection of the fauces, and died a day later, having been very restless towards the last and having one pupil wider than the other; in her, also, the affection was cerebro-spinal. The third case was that of a man, aged twenty, who had acute meningitis involving both hemispheres of the brain, and pleuro-pneumonia of the lower lobe of the left lung; he was suffering from gonorrhœa, and Dr Wilks was inclined to regard this as the cause of his fatal illness, but it was further said that he had had a blow on the head a few days before he was attacked. In the remaining case the patient was a temperate man, aged fifty-seven, who died, after sixteen days' illness, of acute pleuro-pneumonia affecting the left lower lobe; no cerebral symptoms appear to have been observed beyond delirium before death. There was much recent lymph, both at the base of the brain and on the vertex. The only other morbid conditions that were discovered were chronic renal disease (apparently in moderate degree) and hypertrophy of the heart.

Huguenin says that at Zürich inflammation of the cerebral membranes is an exceedingly frequent complication of acute *pneumonia.* He quotes Chvostek as having found it four times in 220 cases in Vienna, and adds that in Zürich the percentage is higher.

Dr Moxon laid special stress on the fact that *syphilis* sometimes seems to be the cause of an acute simple meningitis, and the author found five cases of this kind in our records, in addition to those in which inflammation of the membranes was set up by disease of the calvaria. Once a gumma was discovered in the brain-substance. In no instance was any disease noticed

in the cerebral arteries, but it seems very probable that such a lesion may hereafter be discovered in similar cases. In 1871 a man, already in the hospital for disease of the aortic valves with regurgitation, died after two days' illness of cerebro-spinal meningitis; no embolism was detected, but the spleen contained infarcts. In 1874 a woman, aged forty-eight, who had been admitted for chronic jaundice caused by biliary calculi, was attacked by a fatal inflammation of the cerebral membranes; this, however, was doubtless consequent upon ulcerative endocarditis of the aortic valves, which was discovered at the autopsy. It is remarkable that our records do not show evidence that Bright's disease is a cause of meningitis.

There remain a few cases in which meningitis can be traced to none of the causes mentioned, and must be called *idiopathic*. In our records at Guy's Hospital there are at least nine cases of this kind. In three of them pus or lymph was spread out in large quantities over the whole surface of the brain, equally over the hemispheres and at the base; in three the amount at the base was much less than upon the convexity: in one the latter region was alone affected, the material being in this instance lymph. Once the under surface of the brain and the subarachnoid space of the spinal cord were all bathed in pus, there being but little upon the hemispheres. In the two remaining cases the distribution of the inflammatory products was the same as in ordinary examples of tubercular meningitis. In every instance the cause of the disease remained a mystery, but sometimes meningitis of the convexity is set up by the action of the sun's rays upon the head. Huguenin speaks of labourers who have been attacked while working bare-headed in the fields, and he gives full details of the case of a sailor who fell ill the day after he had been rowing without anything upon his head in the hottest weather of July, and who died in five days. Another case related by the same writer is that of a student who was foolish enough to spend three hours, from 1 to 4 p.m., during a hot summer's afternoon, paddling about the Lake of Zürich on a floating board. All the exposed parts of his body were severely sunburnt. At 6 p.m. he was seized with headache, and an hour later with shivering, and all the symptoms of meningitis rapidly developed themselves; his illness, however, terminated in recovery. Guersant met with a similar instance, which ended fatally, and was verified by an autopsy, in an infant aged six months, whose cradle had been left in the sunshine in the middle of a garden. Rilliet and Barthez mention the case of a child who was attacked after reading a book in strong sunlight.

Course.—The symptoms of acute simple meningitis bear a general resemblance to those of the tubercular affection; in the typical forms of both there is a stage of "irritation," which is succeeded by a stage of "pressure." But the more severe cases of the former present the peculiarity that each of the two periods, although marked by the most characteristic symptoms, may yet terminate within two or three days, or even less.

Primary meningitis commonly begins quite suddenly; it has no prodroma. The patient, if an adult, may be seized with a rigor; in children this is less common. Headache is generally present from the first; it may either be referred to the forehead, or affect all parts alike. There may be exacerbations from time to time, in which piercing cries are uttered; the agony appears to be altogether intolerable. Giddiness is frequently an early symptom; the ground seems to give way beneath the feet, and the legs may feel so weak that to stand upright is almost impossible. Vomiting, perhaps,

occurs once or oftener. There is an extreme irritability to light and sound. The eyes are brilliant and injected, the face is flushed, the head is hot, the carotids are felt to throb violently. The temperature rises, and may reach 104° by the third day; there may be great complaint of thirst. The pulse is quickened as a rule, but this is not always the case. Epileptiform convulsions are not uncommon, and sometimes mark the commencement of the disease. The back of the neck is often rigid, so that the patient seems to bore his head into the pillow. Sometimes his limbs are stiff. He generally lies coiled up in bed, anxiously avoiding notice, and most unwilling to be disturbed in any way. His mind may for a time be perfectly clear, but afterwards he becomes delirious. In some cases there is violent maniacal excitement from the very first. Sleep is altogether wanting, or very broken and disturbed. The pupils are, as a rule, contracted; the ophthalmoscope may show either ischæmia or neuro-retinitis.

The second stage of the disease is characterised by stupor, which more or less quickly passes into coma. The pupils are sluggish, or even dilated and insensible; they are often unequal in size. There may be loss of power in the limbs of one side with or without a similar affection of the corresponding half of the face. Sometimes the patient squints; but even when the base is involved one can seldom make out a definite paralysis of any of the cranial nerves; they of course remain unaffected when the inflammation is limited to the convexity of the brain. Epileptiform convulsions may return again and again until one of them proves fatal, or they may be altogether absent, or, again, there may be attacks of spasm confined to certain muscles or to one arm and one leg. The temperature generally remains high, ranging from 102° upwards, but the face is now pale, and the extremities may be cold to the touch and bathed in a profuse sweat. Towards the last the evacuations are passed involuntarily.

The duration of non-tuberculous meningitis varies with its intensity. Those cases in which the whole surface of the brain, including the convexity, is inflamed scarcely ever last more than a week, and generally terminate within three or four days. When the base alone is affected the patient sometimes lives as long as in the tubercular form of the disease. But this is by no means always the case; he may die in forty-eight hours, and the only discoverable morbid change may be on the under surface of the brain. In such cases, however, there is no successive development of the symptoms in regular order. Sometimes the patient is attacked with epileptiform convulsions and becomes quickly insensible, or he may be violently delirious. In certain cases loss of spirits is the earliest symptom, as was noticed long ago by Abercrombie. There may be little or no pain in the head, and thus the real nature of the disease may be altogether overlooked.

Diagnosis.—Non-tubercular inflammation of the meninges may be as difficult to recognise, as we have seen tubercular meningitis to be; but the diseases most likely to lead one into error are not quite the same. When it is impossible to make out the presence of any of the recognised causes of the affection, and when the symptoms are obscure, one may be unable to distinguish it from alcoholism, mania, or epilepsy. On the other hand, when the patient is known to be suffering from acute pneumonia or erysipelas it may be very hard to determine whether there is more than the ordinary symptomatic delirium. Cases in which alarming indications of cerebral disturbance are traceable to otorrhœa seem to be peculiarly liable to be wrongly interpreted. In 1877 the

author made an autopsy in the case of a patient of Dr Frederick Taylor's, who had died with what appeared to be clear symptoms of meningitis secondary to disease of the ear, including convergent strabismus, a swollen œdematous condition of both optic discs, delirium, and coma. The only sign of any disease of the encephalon was a blackened state of the arachnoid over a small part of one lobe of the cerebellum, but there was a putrid thrombosis of the lateral sinus and jugular vein, and this had set up numerous pyæmic abscesses in the lungs. In the 'Med. Times and Gazette' for 1877 will be found a precisely similar case, under Dr Wilson Fox, in which the patient had all her limbs flexed, and suffered from headache, photophobia, and hyperæsthesia of the surface, but in which the brain and its membranes were perfectly healthy. It would therefore appear that pyæmia dependent upon thrombosis of a lateral sinus is capable of simulating meningitis. The point is of the more importance in that the former disease seems not to be always fatal; at least this appears to be the most probable interpretation of two cases recorded by Dr Andrew in the 'Med. Times and Gazette' for 1875. One was that of a youth, aged sixteen, who had had a discharge from the ear, and who became drowsy and heavy, with frontal headache, giddiness, sickness, and blurring and œdema of the optic discs. The other occurred in a girl, aged nine, who had also had ear disease, and who was attacked with pain in the head, vomiting, and delirium, so that she screamed and started in her sleep. Meningitis was diagnosed, but each patient recovered after an illness of some weeks' duration, in the course of which there was distinct evidence of pyæmia, one of them having pneumonia, the other having an abscess in the thigh.

Lastly, there is always an element of uncertainty in regard to the diagnosis of those cases in which the typical symptoms of meningitis develop themselves in regular order, and with such rapidity as to threaten a fatal termination within three or four days, or even still earlier. The author recorded more than one instance of this kind, in which the gravest prognosis was given, but in which complete recovery took place. One case was that of a young lady, the daughter of a medical man. Her education had been rather neglected during childhood, and she had been for some weeks straining all her powers to keep pace with her schoolfellows in her studies. Her symptoms appeared to point clearly to the presence of meningitis; yet the attack passed off, and she recovered her previous good health.

Prognosis.—The question as to the possibility of recovery from acute simple meningitis is still more definitely raised by certain cases which come under the notice of ophthalmic or of aural surgeons. In 1866 Mr Hutchinson recorded in the 'Ophthalmic Hospital Reports' a series of cases of children who were brought to him for blindness, which he found to be due to optic neuritis, and nearly all of whom had had a severe illness, attended with delirium and other cerebral symptoms, and supposed to be fever. Dr Allbutt suggests that the disease was really meningitis. This observer also states that he has seen several instances in which a condition of defective mental development or idiocy, associated with atrophy of the optic discs, has in all probability been referable to a long past inflammation of the cerebral membranes.

Certain cases in which permanent deafness has resulted from an acute illness have been interpreted in a very different way by Voltolini (who first drew attention to them in 1867) as well as by some other writers. According

to Brunner (of Zürich) the affection in question commonly sets in suddenly; a child suffers from fever; he may vomit; his head is hot; he becomes excited and delirious, tossing about in bed and screaming violently; within the first twenty-four hours he becomes partially unconscious, and after two to four days passes into a state of coma. At the end of another period of two to four days, however, consciousness returns; but when the child first tries to walk he is found to stagger. He quickly becomes deaf and, as a consequence, remains dumb for the rest of his life. Voltolini's theory is that the disease in such cases is an acute inflammation of the labyrinth; the cerebral symptoms (as in Ménière's disease) being caused in some way by the aural affection. Direct evidence from dissection after death is as yet wanting; but, as Brunner remarks, the fact that the deafness is constantly bilateral is opposed to Voltolini's interpretation. He thinks that there may be some morbid change on the floor of the fourth ventricle, implicating the *striæ acusticæ*.

Voltolini's cases and those of Mr Hutchinson and Dr Allbutt must be taken together with other cases in which recovery takes place after an illness resembling meningitis, but without either deafness or blindness or idiocy resulting.

One difficulty in deciding on the presence of meningitis in such cases is the circumstance that one scarcely ever finds in the deadhouse adhesions of the membranes at the base of the brain or other clear evidences of a former meningitis. It has, however, been shown that in epidemic meningitis recovery is not infrequent, and that the disease often leaves behind it deafness or blindness; in several cases the remains of inflammatory exudation have been discovered when the disease has run into chronic hydrocephalus, which has proved fatal a few months later.

The important point, the practical one, is that cases which appear to be well-marked examples of inflammation of the membranes of the brain do not always terminate fatally.

Thus in a case of meningitis one should never quite lose heart; scarcely any is so hopeless as to justify one's abandoning it altogether, particularly if the patient is a child.

EPIDEMIC MENINGITIS.*—From the earliest years of the present century there have been recorded from time to time, in various parts of the Northern Hemisphere, epidemics of a disease characterised anatomically by inflammation of the membranes of the brain and cord, and clinically by fever, various eruptions, and a number of cerebral and spinal symptoms, especially rigidity of the neck, or of the whole vertebral column. So striking is the symptom last mentioned that in Germany it has given to the affection the popular names of "Genickkrampf" and "Nackenstarre." In medical works it has been called "epidemic cerebro-spinal meningitis." But the epithet "cerebro-spinal" suggests the incorrect notion that an extension of the inflammation to the membranes of the cord distinguishes it from tubercular meningitis.

History.—The first well-ascertained epidemic of this disease seems to have been in 1805 at Geneva. In 1806 it appeared in the United States and continued to prevail there for ten years. During this time, and indeed throughout the past half of the century, it was observed in several towns

* *Synonyms.*—Cerebro-spinal meningitis—Cerebro-spinal fever—Meningitis cerebro-spinalis epidemica.

of France and Italy, in Algeria, Spain, Denmark, &c. In 1854 and for seven years afterwards it raged in Sweden, and destroyed more than 4000 persons. From 1861 to 1864 it showed itself in various parts of the United States. In 1863 it broke out in Germany; the north-eastern provinces of Prussia were the first to suffer; but within the next year or two it appeared in Erlangen, in Nuremberg, and in the country districts of Franconia. From that time it has never ceased to show itself at intervals of a few months or longer, now in one part of the German Empire, now in another.

The British islands have hitherto been remarkably free from this disease. In 1846 it appeared in many of the workhouses of Ireland; and in 1866—1868 a very fatal type of it prevailed in Dublin, and to some extent in other parts of the country. Scotland has not been visited by this disorder; and in England only a few isolated epidemics have been observed in certain provincial towns and villages.

A few cases have from time to time been recorded as sporadic examples by writers who seem to have thought that the fact that a meningitis was cerebro-spinal was of itself sufficient to justify a presumption that it was related to the epidemic disease. But all forms of inflammation of the base of the brain are apt to extend to the cord, and the symptoms observed in the cases in question are far from conclusive.

Anatomy.—The morbid anatomy of epidemic meningitis has little to distinguish it from sporadic basal meningitis. Pus and lymph are found both at the base and on the convexity of the brain, especially between the pons and the chiasma, along the large vessels, and in the various depressions and furrows on its surface. It rarely happens that the hemispheres are uniformly covered by it. In the spinal canal the exudation is generally most abundant at the lower part, and on the posterior surface of the cord, having perhaps accumulated there by gravitation. The ventricles of the brain mostly contain turbid serum or pus; and the choroid plexuses and ependyma may be coated with puriform lymph.

Punctiform hæmorrhage or small spots of softening may be seen in the cerebral substance; or it may be œdematous. The cord presents similar changes but less marked. In one case Fronmüller is said to have found the central canal dilated and full of pus.

Among the appearances presented by other organs are congestion and œdema of the lungs, engorgement of the liver and spleen without much enlargement of the latter, a relaxed state of the heart, congestion of the kidneys with fatty epithelium and fibrinous casts in the tubes, and a granular degeneration of the fibres of the voluntary muscles, especially of those which lie along the spine. Rigor mortis is said to be of long duration. The cadaveric stainings of the surface appear early, and are not always limited to the dependent parts of the body.

The pericardium and the pleuræ are sometimes found ecchymosed, and occasionally lined with puriform lymph. Von Ziemssen once saw the large intestine inflamed as in dysentery. The joints often contain pus, and sometimes there are scattered abscesses among the muscles. All these complications must be regarded as dependent on secondary pyæmia.

Course and symptoms.—As a rule cerebro-spinal fever sets in suddenly; the patient may be at work, or (if a child) at play or at school, when he is seized with shivering and violent pain in the head, and feels so ill that he is obliged to go to bed at once. But sometimes—among Ziemssen's cases in

five out of forty-three—there are slight premonitory symptoms, consisting of headache, malaise, nausea, loss of appetite, and wandering pains. In several instances these passed off, and there was an interval, in which the patient felt perfectly well, before the disease began with its usual violence.

Vomiting is almost always an early symptom, being repeated whenever an attempt is made to sit up. After a day or two it generally ceases, but the headache mostly continues throughout the whole course of the disease, although it may sometimes subside for a time. It varies in character and in seat, being sometimes frontal, sometimes occipital, sometimes diffused over the whole of the head. Giddiness is often present with it. In cases of moderate severity the patient lies in a state of stupor, tossing restlessly about, but rousing when spoken to and trying to answer. He is often very irritable to light and sound. The pupils may at first be normal or contracted; ultimately they become dilated. In the more dangerous cases he quickly becomes delirious, with or without convulsions, or passes into a condition of insensibility. Even when he is deeply comatose he often still feels the pain in the head, and groans or cries out, or grasps the temples between his hands. Aphasia, hemiplegia, and other paralytic symptoms of this cephalic group are sometimes observed; but in the most severe cases of all their presence can hardly be determined.

The *muscular rigidity* of the neck already referred to is scarcely ever entirely absent, but is not commonly a marked symptom during the first day or two. It varies in degree from a slight stiffness, noticed only when an attempt is made to bend the head forwards up to a forcible retraction, bringing the occiput almost to a right angle with the spine. Dr Burdon Sanderson has suggested that it is due to a half-voluntary effort on the part of the patient for the relief of pain in the muscles; but it may be present when there is not pain at all, either in the neck or in the back. It would seem, however, that the muscles below the occipital bone do not feel as hard as might be expected; for Sanderson could not detect any tightness of them so long as the head was thrown back, and Ziemssen probably means little more when he says that the tetanic spasm is "limited to the deeper muscles, the trapezii almost invariably escaping." In about half the cases this symptom is accompanied by a contraction of the extensor muscles of the dorsal and the lumbar vertebræ. Sometimes the back is arched so as to be in a state of opisthotonos, but more often it is straightened into what Ziemssen calls *orthotonus*. If an attempt is made to raise the patient, he either slips down to the foot of the bed without bending his back at all, or allows his body to be lifted a very little way, and at the cost of so much pain that he is very soon put back into the recumbent posture. Almost always he lies on one side with his knees drawn up. He may then be nearly free from pain in the back, but sometimes its intensity is hardly at all affected by the position of the body; it is apt to be particularly severe in the sacrum. Pains in the limbs, and especially in the legs, are often complained of; some writers lay stress on the frequency of an acute pain in the knee. The joints may become hot, red, and painful, as is the case in other spinal affections. An extreme cutaneous hyperæsthesia is another common symptom; the patient, though he may be comatose, will perhaps scream out at the slightest touch, or even if his bed is shaken. Tetanic rigidity of the limbs is seldom present, and so also trismus is of infrequent occurrence.

The degree of *fever* in epidemic meningitis is exceedingly variable, and its course is very irregular, so that even those observers who lay most stress

on the typical fever-courses of many other specific diseases admit that nothing of the kind can be traced here. The temperature usually ranges from 100° to 103°, but it may fall and remain normal or nearly so for a day or two at a time; sometimes it rises to 105° or 107°, especially towards the last. The rate of the pulse may be natural or slightly increased; it is liable to frequent fluctuations; in bad cases it may be very rapid. The face is generally pale. The spleen is sometimes, but rarely, found enlarged. As a rule, the abdomen is retracted, but it may be greatly distended. Ziemssen observed several cases in which there was oppression at the epigastrium, constriction of the chest, or even paroxysms of dyspnœa. An abundant secretion of urine has been noticed by several German physicians, notwithstanding high fever; in exceptional cases a small quantity of albumen or of sugar has been present.

An important symptom in this, as compared with other forms of meningitis, is the occurrence of certain *cutaneous eruptions*. Chief among them is herpes of the face. This generally begins on the lips and spreads to the cheek, nose, ear, eyelids; it is often bilateral, and may cover the whole side of the face, as it does not in any other acute disease. It first appears between the third and the sixth day, but fresh outbreaks may take place as late as the sixth or the seventh week. It is sometimes seen on the trunk or on the limbs; but those parts are more often the seat of a roseola, erythema, urticaria, or purpura. There appears to be nothing specific in the characters of any of these eruptions; they are often mixed together in the same case. Ziemssen lays stress on the symmetry with which they are distributed on opposite sides of the median line—herpes on each wrist, urticaria on each leg, or petechiæ on each shoulder. Dr Collins, of Dublin, saw purpuric spots suppurate and scab over, so that they finally left pitted cicatrices. Sometimes large patches of hæmorrhage coalesce and give a uniform purple hue to a considerable part of the body.

The organs of *sight and hearing* are affected in many cases of cerebro-spinal fever. There is often intense conjunctivitis, attended with chemosis. Ulceration of the cornea, irido-choroiditis, or optic neuritis, may develop. Vision may be suddenly lost at an early period of the disease; and is often not regained if the patient recover.

The hearing was found by von Ziemssen to suffer in eight cases out of forty-two. Pain, tinnitus, impairment of hearing were generally experienced soon after the patient fell ill; they either passed off or ended in a partial or total deafness. Such symptoms sometimes depend upon suppuration of the tympanum, leading to perforation and discharge through the meatus. It has been suggested that in other instances they may be direct results of inflammation of the floor of the fourth ventricle, involving the *striæ acusticæ*, or that they may be due to suppuration around the seventh pair of nerves. In certain cases in which deafness had been present, Heller discovered after death a suppurative process in the labyrinth, besides an infiltration of the portio mollis with pus. It then became a question whether these morbid changes were caused by an extension of mischief from the pia mater, or whether they began simultaneously with the meningitis. Heller was disposed to adopt the former opinion. It is curious that in cases of this kind the portio dura constantly escapes, so that facial paralysis is not observed. Severe inflammation of the labyrinth usually leads to an absolute loss of hearing; and in most cases both ears are affected. The consequence is that the patient, if very young, never learns to speak. Even children two or three years old

who were able to talk before they fell ill with the meningitis, and whose articulation after their recovery was at first tolerably distinct, soon begin to lose the power of speech, and ultimately become unintelligible. How important a part epidemic meningitis sometimes takes in the production of deaf-mutism is shown by the fact that in 1874 every one of the inmates of an asylum at Bamberg owed the defect to this disease.

Varieties.—Cerebro-spinal fever has many degrees of severity, so that several distinct forms have been described. Some cases are called *foudroyant* or fulminant, the patient dying within a few hours from the commencement of his illness. Thus Dr Gordon recorded one instance in Ireland in which the disease ran a fatal course in less than five hours. There was a dark purplish eruption of spots of various sizes and shapes, especially upon the lower limbs. Ziemssen says that among forty-three cases he met with four in which the duration was from twelve to thirty hours. He relates a curious instance of a girl who was attacked one afternoon with headache and vomiting, but who got up in the middle of the following day feeling perfectly well, and went out of doors to fetch some beer; at about 2 o'clock she was again seized with violent headache and fell into convulsions, and at 6.30 she died. This form of the disease is seen chiefly at the beginning of an epidemic. According to Ziemssen, the presence of meningitis is discoverable only with the aid of a microscope, which reveals exuded leucocytes, principally along the blood-vessels. But in Dr Gordon's case greenish lymph had already been poured out both on the brain and at intervals along the cord.

On the other hand, there are cases which are termed *abortive*, in which the patient is only confined to bed for a day or two, or may even go on with his work as usual from the beginning to the end of his illness. Ziemssen has recorded in detail three examples of this form of the disease; in each of them headache, a painful stiffness of the neck, and vomiting (or at least nausea) were present; in one there was also rigidity of the upper dorsal vertebræ, and in another herpes and partial deafness were observed. In every instance recovery took place within four or five days. Abortive cases are said to be especially numerous when an epidemic is declining. Their proportionate frequency is very variable. According to Hirsch it sometimes happens that the greater part of the population of a district in which cerebro-spinal fever is prevalent are affected by this mild variety of the disease. Or it may be seen in the adults and the old people, while the severe form is raging among the children. To what extent the morbid changes in the membranes are developed in cases of this kind is as yet altogether unknown.

As a rule, however, the severity of epidemic meningitis is intermediate between these two extremes. If it terminates in recovery, it does not begin to subside before the end of a week or a fortnight.

There are certain other modifications of the disease to which brief reference must be made.

One is an *intermittent* form, in which there are regular paroxysms of fever, recurring after a quotidian or tertian type, with aggravation of all the other symptoms, the intervals being more or less completely apyretic. There are two ways in which cases of this kind have been explained; one supposition has been that they depend on the combined action of the marsh poison and of the specific poison of epidemic meningitis; the other, that the last-named disease is itself really of malarial origin.

But it is evident that neither of these opinions is correct, for the intermittent variety has been observed in districts where ague does not occur. Moreover, Ziemssen has shown that when measured by the thermometer the fever is far from exhibiting the regular course of that disease: the remissions or intermissions often last over several days, the temperature is irregular, and it is unaffected by quinine.

Ziemssen further remarks that the districts in which this disease has prevailed have often been dry, sandy, elevated plateaus; and Hirsch, that it differs from ague in being especially apt to occur in the winter and spring. Yet another point of distinction is its tendency to attack children rather than adults.

Another so-called variety is the *typhoid*. This arises in protracted cases, and is marked by muttering delirium, a dry, brown tongue, sordes on the lips, involuntary evacuations, and bedsores.

Several observers have found that during or just after an epidemic of cerebro-spinal fever, meningitis has presented itself with unusual frequency as a complication of other acute diseases. This is especially apt to be the case with acute lobar pneumonia; no less than fourteen instances of it are recorded as having occurred in Erlangen between 1866 and 1872. It has also been noticed with pleurisy, acute tonsillitis, and scarlatinal nephritis. Many of the patients recover perfectly well, the headache and stiffness of the neck passing off as the symptoms of the primary malady subside; it may then be said that the meningitis is of the "abortive" variety. But not a few such cases terminate fatally; and the usual morbid changes in the membranes are then found at the autopsy.

Relapses are not very uncommon, even in cases of moderate severity, after a week or two of illness; and even when the patient's recovery is uninterrupted, it is often very slow. The headache sometimes continues throughout his convalescence, and may persist for years afterwards, undergoing aggravation when he stoops or makes any mental or bodily effort.

Sequelæ.—These symptoms may probably be referred to cicatricial thickening of the pia mater, and this has been actually found present when there has been an opportunity of making an autopsy, at an interval after recovery from epidemic meningitis, in consequence of *chronic hydrocephalus* having developed itself as a sequela. Recent observations seem to show that this is not an infrequent occurrence. Ziemssen has recorded three instances in which death happened ten weeks, twenty-seven weeks, and thirty weeks respectively from the commencement of the patient's illness; and one in which it took place as late as seven years after. In all of them the membranes were thickened and opaque in different places; and twice cheesy masses were found upon the convexity as well as at the base of the brain. The quantity of fluid in the ventricles was often very great; in the case which proved fatal at the end of thirty weeks—that of a boy, two years old—the thickness of the hemisphere (white and grey matter together) was only about an inch. A sufficient explanation of the occurrence of hydrocephalus under such circumstances may perhaps be found in the persistence of the inflammation of the ependyma which exists during the acute stage of the disease. But, as Ziemssen remarks, the fact that between the meningitis itself and the commencement of the symptoms of the ventricular effusion there is often a clear interval, during which convalescence appears to be going on favourably, tends to support the opinion that the secondary

affection is in some way dependent on the cicatricial changes in the membranes. Niemeyer has suggested that the outflow of blood through the veins of Galen is interfered with by pressure. Dr Collins, of Dublin, in a case which he examined on the sixty-sixth day, found the cerebro-spinal opening between the cerebellum and the bulb occluded, and attributed to this the hydrocephalus, in accordance with the well-known views of Mr Hilton. But in a series of cases recorded by Dr Merkel, of Nuremberg, in vol. i of the 'Deutsches Archiv,' it was noticed that there was always also a large quantity of fluid beneath the arachnoid around the *cauda equina*. This, it is obvious, is altogether inexplicable on the mechanical theories just referred to. And on the whole it seems best to fall back upon the idea of a chronic ependymal inflammation, which indeed is directly supported by the histological investigations of Merkel, who found the ventricular lining and its vessels thickly set with nuclei.

The symptoms which indicate the supervention of hydrocephalus are said by Ziemssen to be chiefly severe headache, and pains in the back and limbs, occurring only in paroxysms, and attended with vomiting, loss of consciousness, convulsions, and the involuntary discharge of fæces and urine. During the intervals, which may last for weeks at a time, the patient may appear to be in good health, mentally and bodily; but very often he is dull and stupid, or he is affected with a general cutaneous hyperæsthesia, or with paralysis or contraction of one or more of the limbs. Progressive emaciation appears to be another marked symptom. Whether it is possible for recovery to take place from the hydrocephalus is as yet uncertain. It is to be noted that failure of memory and weakness of intelligence, when they immediately follow an attack of epidemic meningitis, do not point to the presence of ventricular effusion, and are not of evil omen, since they generally pass off in the course of a few months; and the same thing may be said of various paralytic affections which are now and then observed during convalescence from the disease.

Ætiology.—The causes of epidemic meningitis and its relations to other specific diseases are still very obscure. Those who are attacked by it are comparatively seldom over forty, and generally less than twenty, years of age; in some epidemics almost all the cases have been in children under fifteen. But two of Ziemssen's patients were old people, aged seventy and seventy-seven respectively. Males appear to be more often affected than females. Ziemssen says that it chiefly seizes upon strong, healthy subjects; but he and other observers are agreed that it is apt to be especially prevalent among the poor, who are ill-fed, and who live crowded together in dirty, damp, ill-ventilated dwellings. When it occurred in France at the beginning of the century it was often entirely limited to the soldiers in barracks, the civil population in the same towns escaping entirely. In Ireland, in 1846, it affected principally the inmates of the workhouses; in the United States it fell with especial severity upon the negroes.

Some of the facts just mentioned would seem to indicate that epidemic meningitis resembles typhus in its distribution, and may therefore be infectious. But of its being contagious in a narrow sense there is no evidence whatever. All observers are agreed that it does not pass from the sick to the healthy directly under ordinary circumstances. Hirsch, however, has collected a series of cases which seem to show that a man going from an infected to a healthy place may sometimes carry with him

the germs of the disease, so that not only he himself afterwards falls ill with it, but others are attacked in their turn. One suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being. It is stated, on the authority of Mr Ferguson, Veterinary Officer to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country there has been an epizootic of the same kind among pigs and dogs.

The transmission of a contagion, if there be one, is unusually rapid and mysterious. Dr Sanderson reported that in 1865 meningitis broke out on or about January 15th in two districts of the department of Dantzic, distant at least thirty miles from each other. So, again, Stillé insists on its having repeatedly prevailed in Europe and in America in the same years, and on the way in which it has made its appearance within the United States at places hundreds of miles apart. In this respect he compares it with influenza, and defines it as a pandemic disease.

Diagnosis.—This is seldom difficult. At the commencement of an outbreak, however, it would seem that enteric fever may sometimes be mistaken for it. Leyden says that among the German troops before Paris in 1870 a series of cases occurred in which marked rigidity of the neck, severe headache, and hyperæsthesia were present, while the abdomen was flat, the temperature was low, and the bowels were confined. At first it was doubtful whether the disease was not meningitis, but the autopsies showed that it was really typhoid fever, with but slight implication of the intestine. Epidemics of the two diseases may also prevail simultaneously. Leyden has seen this several times; he insists on the facial herpes as distinctive, since it is never observed in enteric fever.

When occurring as a complication of acute pneumonia, epidemic meningitis may be far from easy of diagnosis. Ziemssen remarks that rigidity of the neck is often entirely absent in such cases. In very young children, too, this symptom possesses very little significance even when it is present; and convulsions, coma, and other signs of cerebral disturbance are equally without value. Maurer maintains that a tense projecting fontanelle affords great help, since it proves that the intra-cranial pressure is excessive, and this is not likely to be the result of simple pneumonia.

Again, if an isolated case of meningitis should occur, it is often impossible to say whether it belongs to the epidemic, the simple, or to the tubercular form. It would seem that the spinal symptoms—the painful stiffness of the neck, the rigidity of the vertebræ, the hyperæsthesia, and pains in the limbs—are generally more marked in the epidemic disease. Tubercular meningitis may often be distinguised by its prodromata, by its gradual onset, by its slow and interrupted course. The presence of a roseolous or purpuric eruption would probably be a conclusive proof that the case was one of cerebro-spinal fever.

In giving a *prognosis* it is important to bear in mind the treacherous character of the disease. A case which at first appears to be of but little severity may afterwards develop dangerous symptoms and prove rapidly fatal; while, on the other hand, patients whose condition had seemed hopeless sometimes recover.

The *mortality* appears to vary in different epidemics from 30 to 70 per cent.; the mean mortality is estimated at 40 per cent.

Treatment of meningitis generally.—At an early stage one important object is to relieve the headache and to diminish the cerebral excitement. The patient should be placed in a cool, dark, well-ventilated room, and should be kept perfectly quiet. The hair should be cut short or shaved. Cold should be applied to the head, and for this purpose a large bladder containing small pieces of ice and a little water is more serviceable than anything else; evaporating lotions are far less effective, and require very frequent renewal.*

In epidemic meningitis Ziemssen and other German physicians keep ice-bags to the head, back, and neck for weeks together, and find that they relieve the patient's sufferings, and enable him to sleep.

Most physicians abstain from administering anodynes in cases of meningitis, as being likely to mask the symptoms; but at an early period of the disease this objection seems unfounded, and Dr Bristowe says that he has frequently given opium with manifest relief. Huguenin recommends the subcutaneous injection of morphia in small doses, and the use of enemata containing fifteen to forty-five grains of chloral. Ziemssen says that in epidemic meningitis remedies of this class are indispensable.

To check vomiting the patient may have little pieces of ice to suck; or bismuth, hydrocyanic acid, and similar remedies may be prescribed.

There is perhaps no reason to suppose that leeches, or venesection, or the most active purging could cut short a meningitis which is going on to the effusion of lymph and pus. But is it equally certain that such measures are useless in those cases which are less severe? In epidemic meningitis German authorities now recommend all those "antiphlogistic" measures which were in vogue in this country thirty years ago. They not only apply leeches behind the ears and cupping-glasses to the spine; in the most acute

* In 1878 I saw a case which appeared clearly to demonstrate the usefulness of the local application of ice. A man aged forty-three, a baker, was taken with a kind of fit on the evening of the 26th of June, and was seen the same night by Dr Churchward with a temperature of 104·6°, a pulse of 160, a very flushed, hot face, vomiting, and great pain in the head and neck, as well as in the back and in the limbs. He had been driving about all day in a hot sun; three days previously he had stood for some hours in water up to his knees in consequence of his cellars having been flooded by a heavy rainfall; for more than a month he had been unusually irritable in his temper. For a day or two he seemed to improve, but during the night of the 28th he became collapsed and almost pulseless, with a cold clammy sweat. After this he remained sleepless and was very delirious, with contracted pupils and constant twitching of the hands. On the 30th I saw him and found the optic discs normal; his symptoms were then of the most alarming character. We prescribed a mixture containing the iodide and the bromide of potassium, and a draught of chloral and morphia in the hope of inducing sleep. Next day (July 1st) he appeared to be rather worse than better; he had not slept for more than five minutes; he had been sick again; he was constantly talking and picking the bedclothes. Dr Churchward therefore ordered a towel to be wrung out of iced water, and to be kept applied all over his head and neck with lumps of ice between the folds. The effect seemed to be magical; in a few hours he became quiet and fell asleep; the sickness and the clammy perspirations ceased. He liked the cold to his head, and during the night he slept for six hours. On the following day (July 2nd) he was rational, and told Dr Churchward that he remembered nothing since the 29th June, except that another doctor had examined his eyes. The ice was continued until July 4th. His recovery was uninterrupted; on the 7th he got upon the sofa, and by the 11th he was able to be drawn out in a carriage. This patient died in the spring of 1880 of acute pneumonia. Dr Churchward obtained for me permission to examine his brain. There was no conspicuous change,—nothing, I think, that would have attracted my notice if I had not known of his former illness. But the arachnoid and pia mater at the base of the brain appeared to me to be thicker and crisper than natural, especially over the right Sylvian fissure; the olfactory lobes were more firmly bound down; and there was more connective tissue on the under surface of the pons. The velum interpositum also seemed to be increased in density, although the lateral ventricles were not dilated.—C. H. F.

cases, or when the patient is very restless, they employ venæsection; and they administer calomel, or rub in blue ointment, at the same time washing out the mouth with solution of chlorate of potass, to prevent the gums from being affected by the mercury.

In simple meningitis the older English physicians believed that they had seen successful results from energetic treatment.*

It is, no doubt, possible that the favourable issue of such cases was spontaneous, and would have occurred independently of all active treatment. But, on the other hand, it may be that under the negative practice of the present day they would have terminated fatally.

Of course it is essential that our treatment should do no harm. When there is any doubt of the diagnosis between enteric fever and tubercular meningitis, it would be very wrong to purge. Other cases in which uncertainty of diagnosis ought to influence our practice, are those of probable meningitis secondary to disease of the ear. This form of the disease is almost certainly hopeless, but we have already seen that pyæmia may simulate it very closely.

Again, in adult patients it is of the utmost importance that we should not overlook the possibility that syphilis may be the cause of symptoms resembling those of meningitis. We have seen that it may give rise to inflammation of the membranes of the brain, but it does not follow that syphilitic meningitis would be especially amenable to treatment. The point is rather that one may be called in to see a man who is insensible, and that the history of the case may seem to point to meningitis, but that the lesion may really be one of the more direct results of syphilis. It would then be cruel to ask of a wife, or of a mother, questions which might leave the most painful impressions, and which, if answered negatively, could never decide the matter. But the most careful search should be made for

* Some of Abercrombie's cases will perhaps still bear quotation. (Case 69), "A girl, aged eleven, had violent headache and vomiting, with great obstinacy of the bowels, and these symptoms were followed by dilated pupils and a degree of stupor bordering on perfect coma; pulse 130. She had been ill five or six days; purgatives, blistering, and mercury to salivation had been employed without benefit. One bleeding from the arm gave an immediate turn to this case; the headache was relieved; the pulse came down; the vomiting ceased; the bowels were freely acted upon by the medicines which they had previously resisted; and in a few days she was quite well." (Case 72), "A gentleman, aged twenty-one, was first affected with confusion of thought and very considerable loss of recollection. He then complained of headache, and after a day or two had double vision. At this time he was out of bed the greater part of the day, but was restless and confused, and at times incoherent. He was then confined to bed, and had constant headache, much incoherence and oppression, the double vision continuing. The pulse was at first frequent, but fell gradually and sank below the natural standard; and the symptoms went through a course exactly similar to that which has been described in many of the fatal cases. As the pulse fell in frequency he became more and more oppressed, until he sank into a state of stupor, from which he could scarcely be roused to answer a question of the most simple kind. The case went on in this manner for eight or ten days, during which time he was treated by repeated general and topical bleeding, cold applications, blistering, &c. The bowels were very obstinate, and large doses of the most active purgatives were given with little effect. The case was considered desperate, when he began to take croton oil in full doses, repeated every two or three hours. In a few hours he was purged very actively nine or ten times; the same evening he was relieved from every alarming symptom, and in a few days he was free from complaint." (Case 75), "A girl, aged seven, had severe headache, impatience of light, fever and slight delirium, followed by stupor, squinting, and great obstinacy of the bowels. The tongue was at first foul, but became clean after a day or two. . . . She was considered as being in a hopeless state of hydrocephalus. At the end of a week, strong purging being produced, she recovered rapidly, and in a few days was free from complaint."—'Diseases of Brain and Spinal Cord,' 3rd ed., 1834, pp. 155, 157, 158.

evidence of syphilis, such as the presence of nodes or gummata, and the recurrence of miscarriages or sterility on the part of the wife. If there is any reason to suppose that this cause may be in operation, full doses of the bichloride of mercury or of the iodide of potassium should be given. If this treatment is unsuccessful it can do no harm, and its success is sometimes very great. Whether it is advisable to prescribe iodide of potassium or mercurials (except in purgative doses), in cases which are believed to be idiopathic meningitis, is very doubtful.

As to the stage when coma sets in, Sir Thomas Watson says that in his experience the patient has sometimes recovered consciousness after a cap of blistering plaster has been put upon his head. He does not tell us whether the improvement was permanent. Huguenin speaks in high terms of the value of energetic cold affusion for the same purpose. But it seems possible that to excite a merely temporary return of consciousness might be injurious instead of beneficial. The application of mustard plasters to the calves of the legs must surely be altogether futile. Our endeavour should rather be to sustain life from hour to hour, in the hope that the tide may turn. Sir Thomas Waston remarks that "patients apparently moribund are occasionally saved by the judicious administration of stimulants and restoratives, of ammonia, Hoffmann's anodyne, beef-tea, wine, and (it may be) of well-timed opiates."

HYDROCEPHALUS.*—Among the diseases to which infants and young children are liable is distension of the cerebral ventricles with fluid.

Origin.—Hydrocephalus is sometimes *congenital.* At birth the fœtal head may be so large as to prevent its passage until it has burst or has been perforated by the instruments of the accoucheur. If the enlargement is less considerable, expulsion may at length take place, and the child may live for a shorter or a longer time. In slighter cases the head of the newly born infant is noticed to be softer and more pulpy than natural, but there is no obvious increase of size until a few weeks have passed. Among twenty-six cases collected by Dr Dickinson ('Lancet,' ii, 1870) there were four in which the disease was said to have been present from birth, sixteen in which it was discovered within the first six months, six in which it was not noticed until between the sixth and the twenty-sixth month. One must bear in mind that these figures express, not the relative frequency of the congenital and acquired forms of the disease (for the list probably includes no infants dying within the first week or two), but the number of cases of hydrocephalus as seen later on in childhood which are congenital. It is evidently impossible to determine in how many of them the hydrocephalus really began *in utero;* but probably in most the brain was healthy at the time of birth. We shall afterwards see that hydrocephalus sometimes arises at a more advanced period of life than in any of Dr Dickinson's cases.

Practical convenience prevents our describing separately the congenital and the acquired forms of hydrocephalus, as is done by Huguenin in Ziemssen's 'Handbuch,' nor does there appear to be any real difference in their pathology.

One supposed cause of "water on the brain" during early infancy is want of resistance in the parietes of the head, allowing the normal ventricular fluid to accumulate in excessive quantity, and thus to distend the

* *Synonyms.*—Chronic Hydrocephalus—Hydrocephalus internus—Water on the brain—Dropsy of the head.

ventricles. Huguenin says it is very common for rachitic children, if they are attacked with whooping-cough and bronchitis, to acquire a form of hydrocephalus due to this passive distension. Dr Dickinson and other observers, who have insisted on the frequency with which water on the brain occurs in cases of *rickets*, would include these cases as rachitic. The occasional association of hydrocephalus with congenital *syphilis* is supposed by Dr Dickinson to depend on a defective growth of the cranial bones, owing to the constitutional taint.

What little beside is known as to the ætiology of hydrocephalus tends, for the most part, to support the view that it is of an *inflammatory* origin.

A few striking instances of its occurrence in several children of the same father and mother have been recorded. Frank saw in one family six, in another seven cases. Gölis reported the case of a woman who aborted six times in succession with dead hydrocephalic fœtuses at the sixth month, and bore three living children, two of whom died of the same disease when eighteen months and three years old respectively. He adduced some facts to show that drunkenness on the part of either of the parents during procreation may dispose to hydrocephalus.

When it begins after birth, hydrocephalus appears sometimes to be directly traceable to an injury. West records the case of a little girl who, some months before her head began to enlarge, had fallen out of the arms of her nurse, and had subsequently been convulsed and comatose for some hours. In that instance the only structure that is said to have been thickened was the ependyma; but in many cases the membranes at the base of the brain are found opaque and adherent. There has been much difference of opinion as to whether hydrocephalus often arises out of acute meningitis. In the epidemic form of that affection such a termination is not infrequent (p. 658); but there is no evidence of its following tuberculous meningitis.*

Pathology.—It has been supposed that the physical and chemical properties of the hydrocephalic fluid enable one easily to determine whether the morbid process was of a passive or of an inflammatory origin. The normal cerebro-spinal fluid is well known to have a very low specific gravity, and to contain scarcely a trace of albumen. In some cases of hydrocephalus the fluid has possessed similar properties, and, according to C. Schmidt, the proportion in it of potass salts to soda salts, and that of phosphates to chlorides, have differed altogether from those which are met with in ordinary serum, and even in fluid derived from the membranes on the surface of the brain—a fact which is cited by Vogel as proving that the hydrocephalic fluid is a specific secretion of the choroid plexuses. But there are other cases in which the fluid has been of higher specific gravity, and has contained from 3 to 11·5 parts of albumen in 1000; and this is supposed to be a proof of the inflammatory nature of the exudation. The presence of leucocytes or of minute flakes of lymph is probably safer evidence. The author observed no less than four instances of the fluid being almost free from

* Huguenin, however, speaks most positively of having seen hydrocephalus begin with acute symptoms exactly like those of (non-tubercular) infantile lepto-meningitis, but which instead of terminating fatally, have subsided and passed into those of a chronic cerebral affection, attended with great enlargement of the head. He says that in such instances he has found the pia mater at the base of the brain thickened and opaque, and the choroid plexuses also showing traces of a former inflammatory change. In a case of this kind related in full detail by Rilliet and Barthez, in which the early active symptoms lasted six days, it is expressly stated that the ventricular fluid contained only a trace of albumen, that the ependyma was normal, and that there were no adhesions of the pia mater at the base.

albumen in cases which were of inflammatory origin. The patients were all adults, and there was no reason to believe that they had had hydrocephalus for more than a few months. Their cerebral symptoms were of recent development, and in one instance were distinctly traceable to an injury.

Several pathologists have shown that there is sometimes closure by adhesions, either of the foramen* at the lower angle of the fourth ventricle, or of the aqueduct of Sylvius; and Hilton propounded the doctrine that the obliteration of these channels is often the cause of hydrocephalus, by preventing the outflow of the normal intra-ventricular fluid into the sub-arachnoid space of the cord, which ought to occur whenever increase in the physiological activity of the brain leads to an increase of its supply of blood. But it is difficult to see why the fluid should continue to be poured out under the increased pressure which must necessarily result, and which certainly is present in most cases of hydrocephalus. Moreover, since the adhesions themselves are the results of a more or less widely diffused meningitis, such as is often attended with an inflammatory change in the ependyma, it would seem more reasonable to regard this as the cause of the effusion than to adopt Hilton's theory. It is well known that in one and the same case the various serous membranes may pour out fluids of very different specific gravity; and it is not improbable that the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when the process is inflammatory. But the question can only be settled by careful observations as to the state of the openings into the fourth ventricle in a series of cases of hydrocephalus in which the physical and chemical properties of the fluid are also accurately determined.

The quantity of water on the brain in cases of long standing is sometimes very great; six, eight, twenty, even twenty-seven pints have been measured after death in different cases. The hemispheres are transformed into a thin shell, perhaps not more than a line or two in thickness, so that it is difficult to understand how they retained any of their functions. The distinction between the white and the grey matter is lost; and the sulci, if visible at all, appear only as shallow grooves beneath the pia mater. The nerve-cells are said to be more or less completely atrophied and destroyed; but accurate histological details seem to be still needed. The corpora striata and the thalami are flattened and broad, as are the crura cerebri, the pons, and other structures at the base. The weight of the brain is in most cases much reduced. Some of the more delicate structures, such as the septum lucidum and the soft commissure, are defective or absent, probably in consequence of the stretching to which they have been subjected. The foramen of Monro and the aqueduct between the third and fourth ventricles are generally widely dilated. The ependyma is generally thick, tougher than natural, and of an opaque white or grey colour. It often contains a large number of amyloid bodies. Its free surface is covered with granulations, or with a number of translucent beads which give it an appearance aptly compared by Dr Moxon to the leaf of an ice-plant.

Under the name of *Hydrocephalus externus* a separate variety of the disease has been described, in which the seat of the effusion is said to be the subdural (arachnoid) space. No doubt this may be the case where there is an extreme degree of malformation of the brain, as in some microcephalic or anencephalous fœtuses, but Dr Wilks has always disbelieved in

* See Cruveilhier, tom. iii, p. 385; and Hilton, on 'Rest and Pain,' 3rd ed., p. 23, and figs. 1, 2, 8, and 9, with Mr Jacobson's note, p. 39.

the occurrence of external hydrocephalus under other conditions. The classical example is that of James Cardinal, recorded by Dr Bright, who died in 1824, at the age of twenty-nine, in Guy's Hospital. The total quantity of fluid in this case was seven or eight pints, and all of it, with the exception of one pint, lay beneath the dura mater at the time of the autopsy. There was, however, a hole in the corpus callosum, and Bright himself supposed that the ventricles were the original seat of the effusion. Dr Wilks believes that the rupture through the corpus callosum did not take place until just before death; and it is evident that the case cannot be fairly cited as an instance of a special form of hydrocephalus (see Catalogue of Museum, No. 1000).

Congenital hydrocephalus is often associated with malformation of other parts as well as of the brain itself. It is seen in combination with spina bifida, and in one of these cases at Guy's Hospital the central canal of the cord was greatly dilated.

A certain degree of secondary dilatation of the ventricles is common in cases of cerebral tumour. This is seldom a matter of clinical interest; but sometimes, when the cranium becomes enlarged, the disease is mistaken for primary hydrocephalus, until a new growth is discovered at the autopsy.

Anatomy.—Enlargement of the head is usually the first, as well as the principal symptom of the hydrocephalus of early childhood. Among forty-five cases collected by West there were twelve in which the frequent repetition of fits first drew attention to the disease, four in which it began with some other indication of cerebral disturbance, and six in which it arose out of an acute attack: in the remaining twenty-three cases no definite cerebral symptom preceded the discovery that the head was increasing in size.

The effect of the disease upon the cranial bones is to separate them more and more widely except at the base. Trousseau compared the change to the falling back of the petals of an opening flower. The frontal bone rises vertically above the eyebrows, or even overhangs them; the temporal and parietal bones arch outwards, so as to hide the ears when the scalp is viewed from above; the occipital bone extends backwards almost horizontally. Thus the head may acquire an enormous circumference. Dr Dickinson had under his care a child nine months old, in whom it measured thirty-one inches, and instances in which it ultimately reached forty and even fifty-two inches are cited by Trousseau. A strange contrast is afforded by the shape of the face, which appears unnaturally small, with angular features and a sharp chin. The base of the skull is generally narrow, with shallow fossæ. The presence of the fluid in the anterior cornua of the ventricles affects the orbital plates, so that they become convex downwards instead of upwards, and thus arises a very important character of the disease in an abnormal position of each eyeball; a large part of the iris, and even of the pupil, is hidden by the lower lid, while the sclerotic above the cornea is exposed to view.

The vertex of the head forms an open area, which corresponds with the natural fontanelles, expanded so as to meet together between the parietal bones and dividing the two halves of the frontal bone by a cleft that reaches nearly to the root of the nose. There is always, however, a work of ossification going on which tends to cover the brain in, provided that the process of expansion is not the more active of the two. In examining the crania of hydrocephalic children, whose death has occurred while the

disease was in progress, the author has found that the original outlines of the bones were still plainly visible, but that they were surrounded by broad zones of new osseous material, marked by radiating lines which showed the direction of their growth. The closure of the skull is often hastened by the formation of *ossa triquetra*, which may be felt loose in the membrane. The date at which it is completed varies widely in different individuals. In Dr Bright's patient, Cardinal, the anterior fontanelle is said to have been finally ossified at about the twenty-seventh year. Sometimes irregularities seem to occur in the union of the several bones. At least this has been assumed to be the cause of a want of symmetry in the cranium which has sometimes been noticed. As a rule the shape of a hydrocephalic skull approaches that of a sphere. It often, however, appears somewhat quadrilateral from the projection of the frontal and parietal eminences. The bones have no diploë; they are generally very thin, and are sometimes translucent; but where life had been prolonged past middle age they have been found greatly thickened. The museum of Guy's Hospital contains a specimen of extensive ossification of the dura mater, which was taken from a case of this kind.

The separation of the bones of the head by an accumulation of fluid within the cranial cavity is not altogether confined to infants. There is a classical case, recorded by Matthew Baillie, of a boy, aged seven, whose skull had appeared to be firmly united, but in whom at the time of death there was an interval of three quarters of an inch at the sagittal, and one of half an inch at the coronal suture. Dr Dickinson says that the same thing has been known to occur in adults; but even in children it is exceedingly infrequent when the bones have once become interlocked.

Physical signs.—In well-marked cases of hydrocephalus it is easy to transmit a wave of fluctuation from one hand to the other across the distended scalp. The skin and the subcutaneous tissues are exceedingly thin, and the ramifications of large veins are often plainly visible through the scanty hair. The head may appear obviously translucent when a candle is held on the opposite side of it. In Cardinal's case this was observed, if the sun was shining behind him, until he was fourteen years old.

Some German observers state they have elicited a *bruit de pôt fêlé* on percussing a hydrocephalic head, especially when the mouth was opened; it is supposed to have been due to resonance of the air in the vault of the pharynx. Auscultation has also been brought to bear upon the diagnosis of the disease. It is well known that on listening over the fontanelle of a healthy child one hears a systolic murmur; this has been supposed to arise in the veins that open into the longitudinal sinus, as a consequence of their being compressed at the moment when the cerebral arteries become distended with blood. Now Rilliet and Barthez maintained that in hydrocephalus this murmur is not to be discovered; but more recent observers have shown that it may often be plainly heard, although they admit that it is absent in acute meningitis, or where effusion is going on very rapidly. The contradictory statements which have been made may be in part due to the fact that even under normal conditions this cranial murmur is only audible between the eighteenth week and the fourth year.

Symptoms.—One consequence of hydrocephalus is a difficulty in keeping the head supported. An infant is often unable to raise it from the pillow, and, if placed in a sitting posture, lets it roll backwards and forwards, as if it had no power in its cervical muscles. An older child perhaps sits with

the head resting on the table, or walks with it carried between his hands, "just as a milkmaid steadies her pail."

In those rare instances in which a skull already fully developed expands under the influence of the disease (as in Baillie's patient already referred to), headache, stupor, and paralysis of all the limbs may be present for several months before any enlargement is noticed. Such cases are transitional between the hydrocephalus of early childhood and that of adult life. But in young children, in whom the cranial cavity readily yields, it is often surprising how few symptoms of cerebral disturbance can be made out, even when the quantity of fluid is already large. Headache is often present; even very young children show that they are in pain by restlessness and a sad whining cry. Vomiting is of frequent occurrence, and, like the headache, it is apt to be excited by movements of the head, especially when the child assumes the erect posture.

The *sight* sometimes remains good throughout the whole course of the disease; but in many instances there is blindness almost from the commencement, due to changes in the optic discs, which are commonly white and atrophied. It is probable that this sometimes occurs as the direct result of the pressure of the ventricular effusion upon the optic tracts; but in many instances it is secondary to "choking" or to neuritis. Huguenin examined three infants with congenital hydrocephalus between the twentieth and the thirty-fifth day after birth, and in each case found the discs reddened and swollen.

The *hearing* is very seldom impaired; as Dr Dickinson remarks, a young child who is perfectly blind may at once recognise its mother by her voice. The smell is said to be sometimes absent, and the olfactory lobes atrophied. Probably in such cases there would be other malformations of the nervous centres as well. The *taste* seems often to be perverted; for the appetite is voracious and indiscriminate.

Impairment of the muscular power of the limbs may be present in all degrees from a slight failure of co-ordination in standing or walking, up to a total paralysis. There is sometimes more or less complete hemiplegia or paraplegia, but such symptoms probably depend upon some additional local lesion of the brain or cord. Spasmodic affections of various kinds occur; nystagmus especially, but also partial clonic or tonic spasms of the limbs, and even general epileptiform convulsions, the latter being very apt to be brought on by any accidental circumstance which shakes the head or gives it a mechanical shock. The legs and arms are often stunted in their growth, and their muscles very small. Huguenin mentions that in one case, in which the lower limbs were for a time contracted but afterwards became relaxed, the muscles were found to have lost their contractility to faradic currents; indicating that there was some secondary or concomitant affection of the grey matter of the cord.

In some cases of congenital hydrocephalus there is no advance of intelligence from the time of birth onwards; and they generally terminate fatally at an early age. In other instances the brain develops to some extent, but very slowly, taking perhaps ten times as long as usual. During childhood such patients would generally be classed as imbeciles.

Cardinal, however, went to school at the age of six, and he soon learnt to read well and to write a little; but he was obliged to give up the latter accomplishment, as stooping caused a pain in the head. When he was twenty-nine years old his mental faculties are said to have been "very fair.

His memory was tolerable, but it did not retain dates and periods of time; and it was stated of him that he had never been known to dream. There was something childish and irritable in his manner, and he was easily provoked. He was stated not to have sexual desire, but he was fond of society and affectionate to his mother. His voice was not manly, but feeble and somewhat hoarse." He died about three months later, having become exceedingly feeble, with a protracted cold, febrile symptoms, diarrhœa, and loss of appetite. Probably, if the lungs had been examined, it would have been found that phthisis was the cause of death.

In the Fulbourn Asylum the late Dr Bacon once showed the author a woman, aged fifty-three, whose head measured twenty-seven inches in circumference; yet she could sing and talk well, and had tolerably good memory and intelligence.

In some rare cases, in which hydrocephalus is thus arrested, the intellectual faculties are said to reach a normal or more than normal standard. The name of the author of 'Vanity Fair' has often been cited as an illustrious example of the fact. But it is doubtful whether the difficulty of distinguishing a rachitic enlargement of the head from "water on the brain" has been sufficiently taken into account in regard to such cases.

For, strange as it must appear, it is a fact that errors have not infrequently been committed, even by skilled observers, as to the presence of fluid within the cranial cavity; and not only has hypertrophy of the brain been mistaken for it, but it has sometimes turned out that the disease has been nothing more than rickets. However, one can for the future avoid this particular blunder by measuring the circumference of the skull, and comparing it with what it should be, according to the age of the child, in a table of standard measurements, such as one which is given by Huguenin. And that writer says that even where the yielding of the softened bones leads to an accumulation of ventricular fluid in a rachitic infant, the ophthalmoscope enables the true character of the affection to be easily recognised, since the optic discs retain their normal appearance. But it is very doubtful whether such tests are applicable to the cases in which there is really most danger of diagnosing hydrocephalus wrongly. These are probably examples of the disease which will be described as hypertrophy of the brain (p. 691) in which the skull is much enlarged.*

Course.—The course and termination of hydrocephalus vary widely in different cases. Sometimes the disease undergoes a rapid advance, and destroys life in a few months by coma or by a succession of epileptiform seizures. Very often the child dies through some intercurrent malady, such as measles or whooping-cough; or, if he is rachitic, he is cut off by laryngismus stridulus or by some other complication. In certain very rare cases the fluid makes its way through the skull, and may even be discharged externally. Rokitansky once saw it poured out through the open sutures and diffused beneath the tissues outside the cranium. Several writers have recorded instances in which it has escaped through the nose, or even through the upper eyelid; and some of them have ended in the recovery of the patient. When the discharge takes place into the nasal fossæ it is supposed

* I suppose that it was a case of this kind which many years ago occurred to me at the Evelina Hospital, but of which I have unfortunately preserved no notes; during the child's life the presence of hydrocephalus was never doubted by me, nor by anyone else who saw it, although the necessity of caution in the diagnosis of that disease had already before that time been impressed upon my mind; but when the skull was opened it was found to contain nothing but a large solid brain.—C. H. F.

that the ethmoid bone must have been loosened from its attachments by the pressure to which it is subjected.

In 1884–85 the writer had a child under his care in Guy's Hospital with extreme hydrocephalus, whose case is worthy of note from its having been under continuous observation from the beginning of the disease till its end. The patient was a remarkably fine boy, six years old, and free from family proclivity to diseases of the brain. He first, while living in the country, began to lose power over his bladder, then he had occasional fits, vomiting, headache and staggering, with slight loss of power in the limbs, and then dimness of sight. We found optic neuritis. It was at first thought that there was a tumour of the superior vermiform process pressing on the veins of Galen and causing secondary hydrocephalus; but the subsequent course of the disease did not confirm this supposition. The child went through a slight attack of diphtheria. The head gradually enlarged, the gait became feeble, complete blindness with double optic atrophy followed, and at last he lay passive and almost motionless in bed with incontinence of fæces and urine, but almost always free from pain and with no convulsions or paralysis. During the last few weeks there was tonic contraction of the arms with ankle-clonus, and for the first time rapid loss of flesh. He died at the age of eight, having lived nearly two years in the hospital (Dec. 18th, 1885). *Post mortem* all the organs were healthy, except the brain, which was distended by serous effusion in both ventricles. The central canal of the cord was wider than usual in the cervical region, and there was early sclerosis of one lateral column.

Prognosis.—If the quantity of fluid is not large, and if the tendency for it to go on accumulating has never been very active, it often happens that the morbid action undergoes arrest after a time, and that as the child grows older the increased size of the head ceases to be conspicuous. Some years ago, in examining the body of a man, aged forty-three, who had been killed by an accident, the author found well-marked hydrocephalus, probably a residue of an attack of the disease in childhood; no information as to the man's mental capacity or attainments could be obtained. Persons have been known to live under such circumstances to advanced age.

It is, however, believed that cases of this kind have a marked tendency to relapse, and that excitement or overwork is apt to light up morbid changes afresh. It is supposed that there is special risk of this occurrence at the period when the ossification of the skull is finally completed.

Treatment.—The treatment of hydrocephalus is unsatisfactory. As might be expected, the drugs which are chiefly prescribed are those which increase the secretions of the kidneys, and thus promote the absorption of the fluid products of inflammation. Small quantities of grey powder or of calomel, pills containing mercury and squill in doses adapted to the age of the patient, the liquor hydrargyri perchloridi, and iodide of potassium are most frequently prescribed. It is doubtful whether benefit is obtained by such remedies, notwithstanding the two remarkable cases of Dr Gower's cited by Sir Thomas Watson.*

The frequent presence of rickets in hydrocephalic children would suggest

* In Lord Herbert's Life (c. 1625) he relates a very similar case. "My cosen, Athelstan Owen, having an hydrocephale in that extremity that his eyes began to start out of his head, and his tongue to come out of his mouth, . . . I prescribed for him the decoction of two diuretic roots, which after he had drank four or five days, he urined in that abundance that his head by degrees returned to its ancient figure, and all other signs of health appeared."

the administration of cod-liver oil and of the preparations of iron; with which, however, one may combine digitalis, or the acetate of potass, or the perchloride of mercury, if it should be deemed advisable.

The mode of treatment which has the largest amount of testimony in its favour consists in the application of pressure to the head. At one time it was usual to cover the whole of the scalp with strips of adhesive plaster. But of late it has been found sufficient to surround the head with a fillet of elastic webbing, two or three inches wide, and carefully adjusted, so as not to cause redness, or to impress the pattern of the material upon the skin. Care must also be taken to shift it from time to time, so that it may not irritate the frontal eminences. The necessity for such precautions is shown by the fact that sloughing of the integuments, leading to the death of the patient, has been known to occur when they have been neglected. Dr Dickinson speaks in high terms of this procedure, and says that it may be expected to succeed in arresting the disease in the majority of cases, provided that the child is young and that the enlargement of the head is of recent development. One instance which he relates in detail is that of a boy, aged thirteen months, whose skull had been growing out of proportion to the rest of the body for nine months, and measured twenty-two inches round. The eyes were depressed. There were occasional convulsive attacks. An elastic bandage was put on, and it was worn for three years, being renewed as often as was necessary. Diuretics and cod-liver oil were given during the same period. Within four months the circumference of the head became reduced to $20\frac{5}{8}$ inches. At the end of the treatment the bones were fully ossified; and although the head, which was then completely covered with hair, measured $21\frac{1}{2}$ or $21\frac{3}{4}$ inches, its disproportionate size was much less conspicuous. The position of the eyes was natural. There had been no convulsions for eighteen months. The child, at this time more than four years old, was sensible, and could make use of simple words for talking; but his memory was deficient.

Paracentesis of the head with a small trocar, at the outer angle of the great fontanelle, has been recommended by some physicians, and it has been performed on several occasions at Guy's Hospital. Perhaps the best thing that can be said in its favour is still the fact that in a case recorded many years ago by Mr Greatwood, a hydrocephalic child, having accidentally fallen upon a nail which penetrated its skull, recovered after three pints of fluid had slowly escaped through the wound. Cases are also on record of recovery after evacuation of the fluid through the nose. Scarcely less successful results have been reported after paracentesis; but the cases in question do not stand criticism.* Only a small quantity of fluid (not more than two or three ounces) should be withdrawn at a time, on account of the danger of setting up convulsions. If the result appears to be good, one may have recourse to the same procedure again and again, a bandage being applied in the intervals. But such repeated operations are very likely to be followed by acute meningitis, or to set up suppuration in the ventricles. It is doubtful whether this mode of treatment should be adopted, except in the hope of warding off death for the time, when it is threatened by epileptiform seizures or by coma. The injection of iodine into the distended ventricles has occasionally been practised, and has not

* See, for example, Dr West's remarks in 1842 on fifty-six cases of hydrocephalus treated by tapping the skull ('Lectures on Diseases of Children,' p. 130); and those of Dr Wilks ('Diseases of the Nervous System,' p. 175).

been followed by death; but there is no ground for supposing that it does good. After having several times practised paracentesis, Dr Eustace Smith has seen no decided benefit result, and sometimes decided evil, and Dr Goodhart speaks but little more favourably of the operation.

HYDROCEPHALUS OF ADULT LIFE; CHRONIC MENINGO-EPENDYMITIS.—Many writers state that adult patients are sometimes affected with a chronic disease of the brain, in which the most conspicuous anatomical change is distension of the ventricles with fluid. Dean Swift is said to have died of this malady, after an illness of three years' duration, in 1745; but one cannot speak confidently of the accuracy of an observation made so long ago, when scarcely anything was known of pathology. Sir Thomas Watson records the case of a young man, who, after one or two sudden attacks of unconsciousness, became dull, stupid, and insensible, and at length died; the only alteration that could be detected in the brain was the presence of a large quantity of serous fluid in the ventricles.

Pathologically there is a close correspondence between cases of this kind and those of hydrocephalus occurring in childhood. Similar changes are found in the ependyma of the ventricles; it is tough and thick, and may be granular and feel rough to the touch, or may have an areolated appearance, like that of the capsule of the liver in some cases of perihepatitis. The floor of the third ventricle often forms a protrusion like a bladder of fluid. The membranes at the base are found greatly thickened, opaque, and matted together; even more so in the affection of adults than in that of children. There is, of course, a great difference in the quantity of the fluid, which seems not to have exceeded fourteen ounces in any of the cases observed at Guy's Hospital within the last few years. Whenever its characters have been recorded, it was clear, and contained only very little albumen. The cranial bones are generally thin, and there is deficiency of the diploë; the interior of the skull is marked by sharp ridges, with sulci between them, which are obviously caused by the outward pressure of the brain. There is a marked flattening of the hemispheres.

Clinically, the relation between the hydrocephalus of adult life and that of childhood is one of contrast rather than of resemblance. The enlargement of the head, which renders the diagnosis of the disease so easy, is wanting; and in the symptoms which are present there is scarcely anything to distinguish it from other chronic affections of the brain.*

* The following are the more striking among fifteen cases recorded at Guy's Hospital. In all of them the ventricles were dilated with effusion: additional facts are added in brackets.

1. A man, aged twenty-three, a patient of Dr Wilks, said that he had been well until a year before, when he noticed a numbness in his feet and legs, which gradually extended up to his face. He had kept his bed for three months. A fortnight back he once found himself unable to micturate, so that a catheter had to be used. Within the last weeks he had had two fits, in which he was insensible. On admission there was a slight convergent squint; the pupils were dilated; the optic discs were ill-defined and red, with some plugging of the veins and retinal hæmorrhages. He had numbness of the feet and legs, and of the face about the mouth. He did not chew his food properly; portions of it would fall out of his mouth while he was eating. His mind seemed not to be clear; and his statements varied. He passed his urine into the bed. After a few days he died suddenly.

2. A man, aged thirty-two, under the care of Dr Pavy, had been obliged to give up work eighteen months before his admission into the hospital, on account of a pain in the head. Nine months ago he lost his speech, had right hemiplegia, and was insensible for three weeks. From that time his memory was imperfect. Eight days before his death he again became affected with partial hemiplegia and inability to speak. He passed his urine and fæces under him. During the last few hours of his life he had a series of fits, which began in the left side of the face, and in which the eyes were turned to the right.

It is clear, from the cases recorded below, that in the adult diagnosis between hydrocephalus and other chronic cerebral diseases is exceedingly difficult, if not impossible. Bilateral symptoms might suggest the nature of the disease, but hemiplegia is sometimes present, and seems to be indistinguishable from that which a tumour might cause.

Huguenin has attempted to give a systematic account of the hydrocephalus of adults; but his description of the symptoms in 'Ziemssen's Handbuch' by no means corresponds with our experience. He speaks of the affection as bearing the closest resemblance to general paralysis of the insane, and he gives full reports of two cases in which such a resemblance undoubtedly existed, although there was no *délire des grandeurs*. Neither of Huguenin's patients died in a general hospital; one was an inmate of the lunatic asylum at Zürich, the other was nursed at home. His cases

3. A man, aged fifty-seven, a cooper, was taken in with partial right hemiplegia, which had come on suddenly four days previously, while he was in the act of striking a piece of iron; he managed, however, to get home afterwards, the distance being half a mile. He did not seem very ill on admission, and looked a strong, healthy man; but he died rather suddenly, nine days after the attack. [In addition to immense distension of the ventricles, with roughening of the ependyma, there was in this case softening of the superficial parts of the corpora striata, and granule-masses were discovered with the microscope.]

4. A man, aged fifty-five, was admitted for chronic dilatation of the stomach, in order that the treatment with the stomach-pump might be carried out. But he became light-headed, and two days later he was convulsed and fell into a semi-comatose condition, in which he lay groaning and muttering until at the end of a few hours he died. He had complained of severe pains in the head three months before.

5. A boy, aged fifteen, who had two sisters in an asylum, was taken in with very obscure symptoms. He was anæmic; he vomited occasionally; there was slight fever; some of the superficial glands were swollen; he complained of pains in the head and in the back of the neck. After a time his mind began to wander; he lay on his side, with his legs drawn up; he answered questions slowly and unwillingly; his eyes were half closed and pupils dilated; the temperature was now below normal, sometimes not above 97°. He died very gradually.

6. A man, aged twenty-two, was admitted under Dr Habershon in 1871 for severe cerebral symptoms. He had once fallen from a scaffold, striking the left side of his head; and was insensible for a fortnight, with bleeding from the mouth, the nose, and the left ear. At the end of three months he resumed his work, but was noticed to be strange in manner. Seven months before his death he was attacked with violent headache and shivering. He gradually became unable to stand, and passed his urine and fæces under him. While in the hospital, he lay all day in a drowsy condition. He would answer questions, but soon began to ramble in his talk. He was occasionally sick, his pupils were dilated, and his head was drawn back. Two months before his death he had a fit; and a month later a second one. After this he lay perfectly still, saying nothing, and eating no solid food; towards the last he became extremely emaciated. [Besides well-marked indications of chronic meningitis, and a greatly dilated state of the ventricles, the anterior and middle lobes of the brain were adherent to the dura mater at the base, especially on the left side. There was a little ochrey-yellow discoloration, extending into the brain-substance, no doubt the result of effusion of blood at the time of the injury. The foramen of Magendie at the apex of the fourth ventricle appeared to be closed, so that the case might be cited in support of Hilton's theory of hydrocephalus (p. 665). The ventricular fluid contained scarcely any albumen.]

7. A man, aged thirty, a patient of Dr Wilks, died in the hospital in 1876, from disease of the aortic valves, and bronchitis. Towards the last he seems to have had no marked cerebral symptoms, but when admitted he was comatose, passing urine and fæces under him, and remained so for several days before he gradually recovered consciousness. His coma began with a succession of fits, and, while in the ward, he had one or two attacks affecting the left side.. He seemed intelligent, and answered questions readily. Eight years before, when he was a healthy young man, he had fallen from a ladder, and cut his head. He was brought home insensible, was delirious for two days, and was laid up for eleven weeks. Ever after he was unable to do any hard work. His memory failed him, he had headache, and feeling on the left side of the body gradually became defective. His first fit occurred five years before his admission. [Besides distension of the ventricles and the aqueduct, which held fourteen ounces of fluid, there was a repaired fracture of the base of the skull, and the brain on its under surface was discoloured.]

only afford additional proof of the variety of aspects that the disease may assume, and of the difficulty of its diagnosis.

In the last two of our cases hydrocephalus was attributable to a severe fall on the head. In one of Huguenin's patients a similar origin was no less directly traced, although the accident was a comparatively slight railway collision. It is an interesting and important question whether the occurrence of serious cerebral symptoms under such circumstances may be taken to indicate that chronic meningo-ependymitis with effusion into the ventricles is developing itself. There are, indeed, other possibilities to be taken into account. A tumour has sometimes been found where during life the symptoms were supposed to be of traumatic origin; and it may be that new growths are, in fact, sometimes set up by injuries. It has been commonly believed that an abscess is not unlikely to be met with in such cases, but this seems to be very doubtful. When cerebral disturbance has lasted for a long time, the only lesion discoverable may be an ochrey-yellow discoloration from bruising of the surface of the brain; or there may be none whatever. The author once examined the body of a man who died of some other disease, but who was said never to have recovered from the effects of an injury to the head, so that he was unable to take stimulants even in moderation; and in that instance the brain, the membranes, and the bones all appeared to be thoroughly healthy.

Huguenin refers to cases in which persons who had experienced blows or falls on the head, after suffering for several months, or even for years, have at length recovered; and he believes that they depend on chronic meningitis. He remarks that the chief symptoms are headache, giddiness, and other subjective sensations of a distressing character, which often cause the patient to withdraw himself from society. The pupils are sometimes unequal or sluggish. The author once had a case of this kind under observation. A bank porter, seven years before, had received a blow on the head from the heavy door of an iron safe; ever since he had been liable to pain in the occiput, vertigo, and a peculiar light feeling in the head. Sometimes he was free from these symptoms for weeks, but the least thing affected his head, so that he could take scarcely any stimulant. Reading often made him feel giddy; and he had been obliged to go out of church on account of indescribable discomfort. Once he complained of numbness in the left side of his head, and again of feeling as if his collar were too tight. The optic discs were normal.

The perchloride of mercury was more useful than any other medicine to this patient; on one occasion it entirely kept off his symptoms for about a year. He also took bromide of potassium and the ammoniated tincture of valerian; and a blister was once applied to the back of the neck.

In more severe cases of hydrocephalus (if that was the nature of these cases) similar measures appear to be the best. Huguenin advocates the continued application to the head of a bladder of ice or a stream of cold water; he also recommends periodical leeching, saline purgatives, and small doses of chloral for the relief of pain. The bromides he has found useful, but not the iodides nor mercurial preparations.

Plastic thrombosis of the sinuses of the dura mater.—This rare primary affection consists in plugging of one or more of the sinuses with a firm clot, which becomes closely adherent, sometimes laminated, and tends to undergo

organisation into permanent tissue. To von Dusch and Gerhardt we are indebted for the best account of it.

In the first edition of this work the author could only refer to a single case from the records at Guy's Hospital. Dr Pitt, in his 'Gulstonian Lectures' for 1890, quotes eight additional cases from the same source. Three were in infants, two in patients between 20 and 30 years of age, and three between 40 and 46; two of the adult patients were men, and three women.

The most usual seat of the thrombus is the longitudinal sinus; sometimes it extends into the tributary veins, so that the hemispheres appear to be covered with coiling worm-like bodies; sometimes it is prolonged into one of the lateral sinuses. In some instances hæmorrhage in the pia seems to have occurred as a consequence of this affection, in others the substance of the brain has been found ecchymosed or softened; but it appears doubtful whether those cases have been correctly interpreted.

There are certain conditions under which this form of thrombosis is especially apt to occur, and which, therefore, may suggest a suspicion of its presence. It has been observed in ill-nourished infants, six months or a year old, who have suffered severely from diarrhœa for some weeks before their death. Such cases generally resemble those of the "spurious hydrocephalus" described at p. 644, but with the addition of some more definite cerebral symptoms, such as nystagmus, squinting, ptosis, facial paralysis, and especially rigidity of the neck, back, or limbs. The fontanelle is generally sunken, but in one of Gerhardt's cases it became tense towards the last.

In adults, adhesive thrombosis of the sinuses seems sometimes to occur spontaneously; sometimes it is consecutive to enteric fever, or to parturition, especially when much blood has been lost. Accordingly this affection has been described as "thrombosis from marasmus," to distinguish it from infective or pyæmic thrombosis. It also occurs in cases of phthisis and chronic Bright's disease; in conditions of feeble cardiac power and liability to venous stagnation.

This rare disease is liable to be mistaken for several others which are comparatively common,—for the less acute forms of meningitis, for tumours, or for other lesions of the upper parts of the hemispheres.

Indeed, the clinical recognition of plastic or adhesive thrombosis of a cerebral sinus is rarely possible. Some writers have described engorgement of veins running from the anterior fontanelle of an infant to the neighbourhood of the temples and ears, and the occurrence of epistaxis, as signs of plugging of the longitudinal sinus; and œdema over the mastoid process as pointing to obstruction in the corresponding lateral sinus.

A curious case is reported by Dr Hyslop from the West Riding Asylum, of a woman suffering from acute mania, who lost all her symptoms for a period coincident with the appearance of hæmatoma of the ears and œdema of the mastoid region and of the orbits. Temporary plugging of the sinuses was diagnosed ('Brain,' April, 1886, p. 90).

The duration of the thrombosis is variable; it may last several weeks, or death may occur a few days after its commencement, so far as this can be determined from the clinical history of the case.

Its *treatment* would probably consist in keeping the patient in a state of perfect quietude, and perhaps in administering ammonia. In one remarkable case, the late Dr Moxon believed that salicylate of soda was of the utmost benefit.

Infective thrombosis of the sinuses.—Far more common than the last-described condition is one of purulent septic thrombosis of the sinuses.

This is always secondary, the thrombus is soft and readily detached, and the endothelium is rough and swollen. Anatomically it has the same relation to the last lesion as septic phlebitis has to adhesive inflammation of a vein, and it is as dangerous as the same process elsewhere. Putrefactive bacteria are always present.

It is most commonly the result of otorrhœa, with caries of the petrosal bone, but sometimes follows compound fractures of the skull, or caries of the bones of the nose or other parts.

It may lead to cerebral or cerebellar abscess, or to meningitis; or the septic phlebitis may spread from the lateral sinus down the internal jugular vein and produce purulent lobular pneumonia on that side, or general pyæmia.

Dr Pitt found 36 cases recorded at Guy's Hospital in the twenty years, 1870 to 1889. They were all secondary: 22 were due to septic suppuration of the ear; 7 followed fracture of the skull, and 3 carbuncle of the face; only one (a case of pyæmia) was caused by a distant lesion.

In 15 of these cases there was also lobular pneumonia; in 4 general pyæmia; in 3 abscess of cerebrum (1) or cerebellum (2); and in 5 meningitis.

He found the most frequent seat of intra-cranial thrombosis (whether simple or septic) to be the lateral sinus (36 cases out of 44), next the longitudinal (12), and next the cerebral veins (7). The other sinuses affected were the cavernous (4), circular, and superior and inferior petrosal.

This condition can only be recognised when local symptoms of the primary disorder, such as otorrhœa or tenderness over the mastoid process, give a local interpretation to the general signs of intra-cranial disease—severe headache, vomiting, and optic neuritis. The temperature is irregular and often very high, seldom normal as in cases of cerebral abscess. Rigors are usually present.

Treatment.—In such cases the mastoid bone should be trephined, and a suppurating lateral sinus may thus be reached and opened. With the happy audacity of modern surgery, this has been repeatedly performed, and a patient's life has been sometimes saved. Moreover, to prevent extension of the septic phlebitis down the internal jugular vein, that vessel has next been ligatured, opened, and washed out; and, lastly, the upper segment has been fastened to the surface of the neck, so as to give free external exit to the products of inflammation in the skull. Mr Lane had a successful case of this kind in a boy ten years old, in August, 1888; and while these sheets are passing through the press, Mr Ballance, of St Thomas's Hospital, has brought before the Medical Society of London, March 31, 1890, four similar cases, two of which were brilliantly successful.

HÆMATOMA OF THE DURA MATER.*—There still remains to be described an affection of the cerebral membranes which, having for many years been a puzzle in pathology, has recently become a subject of clinical interest also. It consists in the presence of one or more membranous layers on the inner surface of the dura mater, within the so-called arachnoid cavity. When recent they are soft and vascular, but in course of time they may become tough, white, and fibrous, so as to resemble in appearance the dura mater itself. Virchow in one instance counted no less than six or seven

* *Synonym.*—Pachymeningitis interna hæmorrhagica.

lying one upon the other. They commonly extend over the greater part of one hemisphere or symmetrically over both. They adhere at their margins so as to form a closed sac, or a series of sacs, which are generally attached much more firmly to the inner face of the dura mater than to the outer surface of the pia. In consequence they were formerly supposed to be formed by a separation of the imaginary parietal layer of the arachnoid from the dura mater; but this notion has long ago been refuted. Indeed, the cyst is sometimes perfectly unattached, so that it falls out as soon as the fibrous covering of the brain is cut through. A considerable quantity of blood, either recent or tawny-brown with age, is commonly found between the layers, which themselves are often deeply stained with hæmatoidin. But sometimes that which fills the adventitious cavity, or cavities, is a thin serous fluid which may contain a quantity of cholesterine; it is believed that such cases are the only examples of what was formerly described as *Hydrocephalus externus*.

In 1845, Sir Prescott Hewett, in a paper read before the Royal Medical and Chirurgical Society, maintained the opinion, which had before been promulgated by Houssard and Baillarger in France, that the starting-point of this disease is an effusion of blood. But a few years afterwards Virchow gave the powerful support of his authority to a very different doctrine (previously upheld by Bayle and by Hodgkin), namely, that the earliest morbid change is an inflammation of the dura mater; this, he supposed, becomes hyperæmic, and exudes upon its under surface a delicate material, richly supplied with wide, thin-walled vessels, which rupture and yield the blood that is so commonly found extravasated. Virchow's view has been adopted by most of those who have since written on the subject; and his name, "pachymeningitis hæmorrhagica," has met with very general acceptance.

Huguenin has, however, reverted to the theory that the disease begins as a hæmorrhage. If his observations are correct, one can hardly escape from the conclusion which he draws; and in any case the best designation seems to be the old one of "hæmatoma of the dura mater," which leaves the question of its origin open. In a large majority of instances the affection is merely a complication of cerebral atrophy, whether senile, or due to chronic alcoholism, or associated with general paralysis of the insane. Now, Huguenin states that in a number of cases of general paralysis he has been able to trace what he believes to have been the earliest stage of a hæmatoma, in the presence of a soft layer of blood-clot, spread out over the convolutions, having its greatest thickness (2 mm.) opposite the parietal eminence, and gradually thinning off towards its margins. This substance comes away from the dura mater in shreds, and has no vascular connection with that membrane. Moreover, the microscope shows that the clots contain at this period nothing but a network of coagulated fibrin, blood-discs, and leucocytes. Afterwards the clot undergoes organisation, vessels are developed in it, and become continuous with those belonging to the dura mater, which now looks more or less injected. Subsequent changes lead to the formation of thick membranous layers, with accumulated blood or serum between the layers.

The most usual seat of hæmatoma of the dura mater is over the upper surface of the hemispheres, corresponding to the dura lining the parietal bones. In about half the recorded cases it has been bilateral.

The original source of the hæmorrhage Huguenin believes to be in the veins which open into the longitudinal sinus; he has noticed that these

vessels are often varicose and thinned, that their coats are affected with fatty degeneration, and that they are sometimes filled with thrombi.

Hæmatoma of the dura mater occurs more often in males than in females. It is met with chiefly in those who are advanced in life, but exceptional cases of it may be seen at all ages; in 1864 Dr Wilks exhibited to the Pathological Society a specimen taken from a young adult. It is scarcely ever observed in the deadhouse of a general hospital; the specimen just mentioned came from the dissecting-room, the subject, a "half-witted" young man, having died in a workhouse.

There are enumerated among the causes of hæmatoma chronic affections of the lungs, heart, or kidneys, anæmia, scurvy, and hæmophilia, typhus, smallpox, and acute rheumatism. Injuries to the head seem occasionally to produce it. In 1855 Dr Quain showed to the Pathological Society a specimen taken from a farmer, aged fifty-eight. He had for three years suffered from various cerebral symptoms, which dated from a fall from his cart, when he received a large scalp wound, and was for a time insensible.

Excessive anæmia, particularly Addison's anæmia gravis, and chronic alcoholism, appear to be the best established predisposing causes. Whooping-cough is an exciting cause, as can be readily understood.

The clinical course of hæmatoma of the dura mater varies widely in different cases. Sometimes the only symptoms are a fatal apoplectic seizure, consequent on the sudden outpouring of a large quantity of fresh blood in or between membranes formed out of a coagulum of old date, which itself had in no way disturbed the patient. Very often the affection is found on *post-mortem* examinations of those who have died of general paralysis of the insane, without any unusual symptoms to suggest the presence of a hæmatoma. It would be a great mistake to suppose that the epileptiform and other attacks which are of frequent occurrence in general paralysis are usually, if ever, traceable to that condition. Indeed, as Huguenin remarks, the wasted brains of these patients allow of the accumulation of a large quantity of blood on their surface, without pressure. Even headache seems to be very generally absent in such cases.

Huguenin relates the case of an intemperate man, aged thirty-one, who about two years before his death began to complain of severe headache, and who one day fell down unconscious, with a slow, full pulse, contracted pupils, transitory convulsive movements of the right side, and partial paralysis of the right side of the face. After twenty-four hours he gradually recovered his senses and got up, but he continued to suffer from pain in the head, and slowly lost his memory and intelligence, and finally he had another seizure which proved fatal in four days. The brain was found atrophied, with dilated ventricles, and with a hæmatoma on each side consisting of a complete membranous sac divided into compartments.

That it is possible for hæmatoma to subside after giving rise to well-marked symptoms appears to be established by the following case recorded by Bouillon Lagrange, and quoted by Huguenin.

A man, aged seventy-five, who was suffering from drowsiness, and failing in memory and bodily activity, had a fall from his horse. Though not injured outwardly, he now became more deeply unconscious, with right hemiplegia. At the end of two months he was comatose and almost totally paralysed; he was unable to speak, and passed his motions under him. After this, however, he gradually began to improve, he regained his senses and his memory, he recovered the use of his limbs, and when two more

months had elapsed he was considered to be cured. He remained well for six months, and then he was murdered. On a *post-mortem* examination, besides a recent injury to the skull, the back part of the right hemisphere was found to be flattened by a cyst, which adhered to the visceral arachnoid and contained three or four spoonfuls of a bloody liquid.

Griesinger ventured to infer the presence of dural hæmatoma in a man, aged fifty-seven, who recovered and was in good health at the time when his case was published. He had been a spirit-drinker, and for about five months had suffered from severe headache, extreme drowsiness, and confusion of ideas. His gait then became unsteady, his pupils were contracted, his pulse was rather irregular, and there seemed to be slight paralysis of the left facial nerve. He would sleep all day long, and passed his urine in his bed; but at the end of a month he began to improve, and in about ten days from that time he was perfectly well. Griesinger confesses not only that he anticipated a fatal issue, but also that he expected to find a rapidly growing cerebral tumour. So little is positively known of recoveries from any organic disease of the brain, that the above observations of Griesinger and Bouillon Lagrange are well worthy of being remembered.

The best treatment for a case supposed to be one of hæmatoma would probably consist in the application of cold to the head, in the administration of purgatives, and perhaps in the abstraction of blood by leeches or by venesection.

Meningeal Hæmorrhage.—When blood is found effused into and under the pia mater the symptoms are those of cerebral hæmorrhage producing apoplexy or hemiplegia or both, and have been described in the chapter on that subject (*supra*, p. 557). The effusion is commonly large, the symptoms severe, and the fatal issue not long delayed. In a woman who was more than once under the writer's care with symptoms of mitral stenosis and embolism of the femoral artery, and subsequently of the radial, death followed about eight hours after a sudden attack of apoplexy, and meningeal as well as cerebral hæmorrhage was found after death (April, 1890).

Congenital meningeal hæmorrhage sometimes occurs during protracted labour from interference with the circulation. It may prove rapidly fatal; but when the infant survives, atrophy of the cortex corresponding with the hæmorrhage follows, and produces a remarkable clinical disease characterised by spasmodic movements, and described as congenital choreiform spasms (*vide infra*, p. 745).

GENERAL PARALYSIS

AND OTHER CHRONIC DISORDERS DUE TO DIFFUSED LESIONS OF THE BRAIN

"Quemne ego servavi in campis Gurgustidoniis,
Ubi Bombomachides Cluninstaridysarchides
Erat imperator summus, Neptuni nepos!"
PLAUTUS, *Miles Gloriosus.*

GENERAL PARALYSIS OF THE INSANE—*History—Ætiology—Symptoms, stages and course—Anatomy—Diagnosis—Prognosis—Treatment.*
Other forms of cerebral atrophy—Senile—Alcoholic—Saturnine.
Chronic diffused inflammation of the brain—Hypertrophy of the brain.

IN 1860 Griesinger, in a paper in the 'Archiv der Heilkunde,' drew attention to the importance of distinguishing among organic affections of the brain those which are limited to some particular region (focal, *herdartig*) from those which are diffused. Of the diseases which he placed in the latter class, some, belonging to the membranes and ependyma rather than to the cerebral substance, have been described in the last chapter. But there remain others, which we will now discuss. As might be expected, they have no localising symptoms. Griesinger briefly sums up their effects as consisting of "giddiness, diffused headache, delirium, and mental failure—from slight dulness of intelligence up to imbecility,—drowsiness and stupor, tremors and involuntary quiverings of the muscles, vomiting, and, lastly, the more positive indications of augmented pressure within the cranial cavity, slowing of the pulse and of the respiration."

In the diagnosis two points have to be borne in mind: (1) that local affections may produce like symptoms if they happen to take up a large space, or if they are seated in a part of the brain of which no special function is known; (2) that diffused organic diseases are very apt to be confounded with certain neuroses, and with the various forms of insanity (*psychoses*), in which no anatomical change is present.

GENERAL PARALYSIS OF THE INSANE.*—With the exception of a slight reference in the works of Thomas Willis, the celebrated anatomist and physician, who died in 1675, the earliest mention of this disease seems to have been made by another Englishman, John Haslam, in 1798; but afterwards its study was taken up in France, and the first complete account was published by Calmeil in 1826. Of late years it has attracted much attention both in England and in Germany, but even Griesinger refuses it an independent position in the nosology, and describes it in a chapter on the complications of insanity. Yet we shall find that its symptoms and course are remarkably definite. It shows little

* *Synonyms.*—Dementia paralytica—Softening of the brain—Cerebral atrophy with delusions—General paresis.—*Fr.* Folie paralytique—Polyparésie—Délire des grandeurs.—*Germ.* Progressive Paralyse der Irren.

or no tendency to shade off into the other forms of mental disorder; it is not apt to come on secondarily in persons who are already the subjects of chronic insanity, nor does a relapse of mania or of melancholia ever assume its characters. It is, in fact, from the first distinguished more or less plainly by features of its own. The impairment of muscular power, too, is of a peculiar kind. The mere association of hemiplegia or paraplegia with unsoundness of mind does not constitute paralytic insanity. In 'Ziemssen's Handbuch' the article on this disease, which is written by Hitzig, is rightly separated from those on the psychoses proper, and appears among the organic affections of the brain. Perhaps the best name for it is that which he uses, "progressive paralysis of the insane," but in this country it is commonly called "General Paralysis of the Insane," or more briefly "General Paralysis." It constitutes a large proportion of the cases which in popular language are called "Softening of the Brain." Its importance may be estimated from the fact that in some lunatic asylums as many as one in four of all the male patients are said to suffer from it.

Ætiology.—The persons most apt to be attacked by general paralysis are married *men* in the prime of life. The proportion of males to females is variously stated by writers; some give it as eight or even ten to one, others as no higher than two to one. The women who fall victims to this disease almost all belong to the lower classes, whereas it is most common in highly educated men, with powerful frames and handsome faces, who have enjoyed life and lived hard. According to Mr Austin, general paralytics are (in England) fair-complexioned and thin-skinned, with blue or grey eyes.

The *age* at which it usually occurs is between thirty and fifty, particularly about the middle of that period. In persons over sixty it is scarcely ever seen, although Mr Austin refers to one instance in a man aged seventy-six. Those who live in towns and cities furnish a far larger proportion of cases than do rural populations. For this reason probably, the disease is rare in Ireland, in the Highlands of Scotland, and in Wales.

General paralysis, it is believed, differs from other forms of insanity in being less frequently due to inheritance or congenital predisposition; and if so, one would expect to be able to trace it to acquired conditions. But there is much difference of opinion with regard to its exciting causes.

Dr Blandford is strongly disposed to attribute it in most cases to sexual *excesses*. He admits, indeed, that he has not always been able to refer it to such an origin, but then, as he says, in married men there may be great difficulty in ascertaining the fact, and an amount of sexual intercourse which to some would be perfectly harmless may be injurious to others. Dr Sankey found that at Hanwell a large proportion of the women affected with general paralysis had led irregular lives. Griesinger attributes the disease to excessive mental excitement, especially to "emotional agitations." Austin says that it commonly follows a painful mental shock, such as would be caused by bankruptcy, or might arise from remorse. Hitzig believes that it results most frequently from the combination of hard work with venereal excesses and indulgence in drink.

It sometimes follows *injuries* to the head, or an acute febrile disease. Dr Savage finds it rare in epileptics, and frequent in those who have suffered from syphilis, as well as in subjects of tabes.

Symptoms.—The course of general paralysis is commonly divided into three stages.

(*a*) During the *premonitory period* an alteration in the character is the most striking symptom. Perhaps the man is extravagant in his expenditure, making presents to persons whom he scarcely knows; or he may be dull and sulky in his demeanour, or depressed and melancholy. So far there is nothing distinctive of this rather than of other forms of insanity; but there is commonly some impairment of memory. Dr Blandford says that such a patient is regardless of appointments, forgetful of the time of meals, of the hour for going to bed, and the like. He comes and goes, scarcely noticing those about him, giving absurd and conflicting orders to his servants, and falling into a passion if they are not instantly executed. He neglects his business, and is careless and indifferent to things which formerly interested him. When he takes up a new scheme his interest in it soon flags. He may commit indecent actions, but if he exposes his person he often seems to be half unconscious of what he is doing; or he perhaps commits assaults upon women, without regard to opportunity, place, or consequences. He sleeps ill, and drinks to excess from inattention, forgetting how much he has taken. He eats hastily, and is apt to spill his food on his dress. He is neglectful of his appearance, and his costume is often unsuitable for the occasion, or its different parts are incongruous. At this time there is no physical change detected by the physician.

Dr Savage notes, among early or premonitory symptoms, change of expression and of temper, perversions of the sense of smell, muscular fatigue, temporary aphasia, *petit mal*, and fits of unconsciousness or of convulsions without the characters of idiopathic epilepsy.

It is of the utmost importance that the distinctive characters of incipient general paralysis should be well known to the general practitioner, since he alone is likely to see the patient at this period of his disease.

(*b*) After a few weeks, or at the end of a month or two, the *second stage* develops itself, and the patient becomes manifestly insane. The *delusions* which he now exhibits are almost always connected with ideas of colossal size, or magnificent wealth, or extravagant numbers. He may say that he can walk 100 leagues in a day, or write 100 tragedies and 1000 poems in the same space of time, or that he is going to make his fortune by buying up all the joint-stock banks, or that he is about to marry the Queen and all the princesses. French writers give to this form of insanity the name of *délire des grandeurs*. These "large delusions" are not maintained from day to day, nor are they consistent with one another. In fact, in spite of his eager excitement, the patient's mental condition continues to be one of progressive decay. He is extraordinarily self-satisfied, full of ideas of greatness, importance, and riches; but he takes no notice of the fact that the palace in which he resides is really a mad-house, and that the great men who surround him are lunatics like himself. Thus he cares much less about being placed under restraint than other insane patients.

In some instances, however, the delusions are not all of such a kind. Dr Blandford mentions that one of his patients thought he was going to be arrested, that people were about to injure him, that they were maligning, and would rob him. Yet, although his symptoms so far resembled those of melancholia, this man was often cheerful and talkative; he was very vain of his appearance, and exceedingly fond of his food.

Though cases of general paralysis usually begin with maniacal excitement and "exalted" delusions, a certain number begin with hypochondriacal

fancies. It is common to meet with patients of the age and with the history common in general paralysis, who imagine that their bowels are obstructed; or that their body has become very small, so that the term "micromania" has been invented to contrast with "megalomania," the delusion of greatness and power. Beside melancholia, any other form of mental disorder may accompany general paralysis. A practical aid to diagnosis is the fact that, however depressed such patients may be in the earlier stage of the disease, they become fat and easy in mind afterwards.

This second stage of general paralysis is further characterised by certain physical symptoms, which must be carefully looked for. The earliest of them is commonly a defect of articulation, an indistinctness or *thickness of speech*, or a hesitation in the middle of a sentence, or a tendency to substitute for the proper word another of which some letters are the same. The tongue, when protruded, is *tremulous;* that is, it not merely shows a fibrillary tremor—which is often observed in persons who are in perfect health, if they are nervous or anxious—but it oscillates irregularly to and fro, and cannot be held quiet. Sometimes the lips are seen to quiver, as they do in persons about to burst into tears. Austin adds that the mouth is generally closed, that the lips are compressed, and that the upper one is straight, its natural curves being obliterated.

The *pupils* are almost always unequal, often small; and they are sluggish in their reaction to light and to accommodation.

In some cases the *gait* becomes tottering, the patient walks stiffly and does not lift his feet; he straddles, or shambles along, and stumbles over any obstacle in his path. The movements of the hands may also be impaired; Griesinger speaks of them as being "stiff;" objects are grasped convulsively, and suddenly allowed to fall. The late Dr Mackenzie Bacon noticed that an important indication of the disease at this stage is an alteration in the character of the *handwriting;* it becomes tremulous and uncertain; sometimes, too, words are omitted, or the same sentence is written over and over again, or the whole becomes an incoherent jumble. All these, however, are indications of mental rather than of bodily failure.

The *knee-jerk* is sometimes found to be absent; sometimes it is exaggerated. Irregularity in this function is often a valuable early symptom.

In almost all cases one or more "fits" occur during this period of the disease. These may be of various kinds; the patient may become comatose, with complete paralysis, anæsthesia, and abolition of reflex movements, and in such an attack he may die. Or he may be less deeply insensible, with loss of power affecting one side only. The paralysis is often transitory, disappearing in a few hours. Austin believed that seizures of this kind were often direct results of the accumulation of scybala in the large intestine, and that the administration of purgatives and enemata was efficacious in removing the hemiplegia. In some cases the fits are epileptiform, and these are often directly fatal, as in the case of a man, aged thirty-seven, who died in Guy's Hospital in 1855. Lastly, the patient may be attacked with convulsions without becoming insensible, or falling.

Persons affected with general paralysis in its second stage are exceedingly liable to *paroxysms* of rage and fury, surpassing in violence those that occur in any other form of insanity except paroxysmal mania (p. 773). Even in an asylum such patients cause unusual anxiety and trouble to those responsible for their safety, and all writers are decided against allowing them to remain

in the hands of friends or relations at this period of the disease. Dr Blandford remarks that these are the persons who sometimes get their ribs broken by attendants before they can be mastered. They make the most desperate efforts to escape; or they tear up their bedding and clothes and go about naked; and they are often filthy in their habits.

After a variable period, a week, or a month, or a longer time, these violent symptoms commonly pass off. The patient's condition may even improve so much that he is able to leave the asylum. The disease is then sometimes said to be cured, but all writers are agreed that permanent complete recovery is the rarest possible event, and almost all deny that it ever occurs. Dr Blandford says that he has known some persons affected with general paralysis who were able to live with their families, to spend their money without extravagance, and to write long letters without mistakes, detailing their travels and amusements; but he adds that he has not met with one case in which the patient was capable of work or business. The friends and relations of such persons notice a childishness and slowness of intelligence in them, and if they attempt to resume their former occupations they break down and have again to be placed in confinement.* In many cases no improvement takes place at any stage of the disease.

(*c*) In the *third stage* the patient's mental condition gradually passes into one of dementia, and his bodily state into one of complete paralysis. His notions of magnificence may continue a little longer, but his understanding and memory soon become altogether destroyed. Sometimes he sits and grinds his teeth for hours together. His speech cannot now be understood; his power of swallowing is greatly impaired. His hands tremble so that he can scarcely hold anything; and it is as much as he can do to shuffle about the garden with the aid of an attendant.

This stage leads up to a fatal termination, which is seldom long delayed. Writers are agreed that the average duration of the disease is short. According to Hitzig the majority of patients die in from fifteen to thirty months after their admission into an asylum. Calmeil and Griesinger speak of the ordinary course of general paralysis as lasting from several months to three years. But the last-named writer alludes to exceptional cases, in which life was sustained for as long as ten years; and Dr Blandford mentions particularly the case of a baronet of large fortune, on whom a commission of lunacy was held in 1858, when he was suffering from general paralysis, and who was alive in 1870. In his experience the average duration of the disease has been considerably longer than that stated by Griesinger. He remarks that persons affected with general paralysis are always more feeble in cold weather; during the heat of summer they may regain strength to a surprising extent, but with the first frosts they fall back, and are apt to sink.

The immediate cause of death is often something which may almost be termed accidental. Thus a frequent mishap is choking; the patient goes on filling his mouth without swallowing the food, until it gets into the larynx and trachea, or at least fills up the pharynx so as to obstruct the entrance of air. Persons suffering under general paralysis should always have their meat minced, and an attendant should be present at meal-times. Another way in which death occurs is by the supervention of bronchitis or congestive pneumonia; if overlooked at its commencement this may destroy

* The only exception to this rule that I have met with in my reading is a case related by Schüle ('Allg. Zeitschrift f. Psychiatrie,' xxxii).—C. H. F.

life in a few hours.* Bedsores sometimes seem to bring about the fatal termination.

Anatomy.—General paralysis differs from all other forms of insanity in being constantly attended with morbid changes in the nervous centres. This, indeed, is admitted by all observers, but unfortunately, when the question is that of defining accurately their nature, and of distinguishing them from those which occur under other conditions, there is by no means the same agreement. Some writers have laid much stress on the fact that the pia mater is often abnormally adherent to the convolutions, so that the cortical substance becomes torn and assumes a ragged appearance when one attempts to strip off the membrane. But in other cases there is an excess of fluid beneath the arachnoid, and the pia mater can then be removed more easily than from the healthy brain. Thickening of the arachnoid has now and then been noted, pachymeningitis and hæmatoma of the dura mater, or increased density and thickness of the calvaria. The absolute weight of the brain is said to be diminished; the ventricles are described as being of unusual size, and their ependyma abnormally thick.†

The ganglionic cells of the brain have been found affected with pigmentary degeneration. Hitzig says that this change is almost always present in a very large number of the cells; but he goes on to quote Westphal as having stated that similar appearances are observed in other cerebral diseases, and even under normal conditions, so that he is obliged to add that it is only the extent of the degeneration that can be regarded as peculiar to general paralysis. Less commonly, he says, cells are seen which are swollen and sclerosed; this seems to be in accordance with an observation of Dr Major's, who in one case ('West Riding Asylum Rep.,' vols. iii and iv) discovered what he describes as an hypertrophy of certain cells, these being abnormally large and furnished with an excessive number of branches. Some have thought that the neuroglia is increased both in the grey and in the white substance. In the latter, patches of degeneration have been observed, of the kind termed miliary sclerosis (Rutherford and Tuke).

In one case Dr Lockhart Clarke found the white substance of the convolutions full of little cavities, of round, oval, fusiform, or crescentic shape, and varying in size from that of a small pea or barleycorn to that of a grain of sand, so that the cut surface looked like Gruyère cheese, or crumb of bread. These vacuoles were doubtless analogous to those which have been described by Dr Dickinson in other diseases. Most of them are empty, but some contained the remains of vessels mixed with granules of hæmatoidin. Probably they all were originally perivascular canals.‡ Changes in the blood-vessels have been described by some observers as features in the morbid histology of general paralysis. The small arteries become dilated and tortuous, and present twists or kinks in their course. Their nuclei and those in the walls of the capillaries are found to have undergone proliferation. Granules of hæmatoidin may be scattered

* I know of one case in which the patient, a nobleman, had been for his usual drive in the afternoon, but in the evening was noticed to be out of sorts; on auscultation it was found that he had pneumonia, and next morning he died.—C. H. F.

† Some of the earlier observers, including Calmeil, described the cortical substance of the brain as of a peculiar violet-red colour; Westphal admits that it sometimes presents this appearance, but he says that in other cases it looks remarkably pale and faded. As he remarks, these variations probably depend on the mode of death.

‡ See some cases of this remarkable condition of the brain described by Drs Savage and Hale White, in the 'Pathological Transactions' (vol. xxxiv, 1883).

upon their exterior, and they may be affected with fatty degeneration or calcification.

Moreover, other parts of the nervous system, as well as the brain, present morbid changes. The state of the spinal cord has been especially investigated by Westphal, and he has found that its posterior and lateral columns are often extensively altered. In the former the appearances are generally those of grey degeneration, exactly like that of locomotor ataxy; in the lateral columns the appearances are those of chronic myelitis. It is not supposed that such changes arise by the extension downwards of a morbid process which had commenced in the encephalon, for Westphal could not trace the affection of the posterior columns above the commencement of the fourth ventricle, nor that of the lateral columns beyond the lower end of the crura cerebri. They must, therefore, be regarded as of independent origin. Indeed, it would appear that even the peripheral nerves fail to retain their normal structure. At least, the sciatic nerves are said by Dr B. Lewis ('West Riding Reports,' vol. v) to be smaller and softer than natural, and less rounded in form. And under the microscope he was able to detect atrophy of the nerve-tubules, with overgrowth of the connective-tissue elements.

Bonnet and Poincarré state that the sympathetic ganglia, especially in the cervical region, present constant morbid changes. Their nerve-cells are sclerosed and pigmented or destroyed. The discoverers of these appearances believe that they constitute the starting-point of the disease, and that its phenomena depend upon the resulting vaso-motor disturbances. But this conclusion is unjustified. The changes in question are probably not morbid in the strict sense of the word, but after childhood are almost constant degenerative changes (see Dr W. H. White's paper, 'Guy's Hosp. Rep.,' 1890).

Diagnosis.—This must be considered from two points of view. In some cases it is a question between general paralysis and the psychoses; in other cases between it and paralysis dependent upon chronic alcoholism, wasting of the brain, or other diffused changes in the nervous centres.

It is not generally difficult to say whether a patient is suffering from general paralysis or from *mania*, even at an early stage. But mistakes have sometimes been committed, from its being supposed that the presence of exalted ideas is characteristic. Thus Dr Blandford mentions the case of a gentleman who wanted to make a tunnel through the earth to the antipodes, and who thought that people might live a thousand years if they would bathe in beef-tea and beer, and that he should be able to pay off all the mortgages on his estate by assembling 10,000 persons in his park, having them photographed, and selling the photographs at five pounds apiece. But it was clear that he was not labouring under general paralysis, for he did not stutter, his memory was perfect, and (above all) he had had a similar attack some years before. The opinion was given that the case was one of mania, and that it might pass off; and the sequel proved the correctness of this judgment.

The exceptional cases of general paralysis in persons at an advanced age may be difficult to distinguish from those of *senile dementia*. The childish nature of the delusions in those who are suffering from senile dementia commonly lends aid to the diagnosis: that complaint is slower and more uniform in its course; it is less apt to be complicated with epileptiform fits, and any apoplectic attacks that may occur are much more likely to be followed by permanent hemiplegia.

Chronic *alcoholism* sometimes gives rise to a chain of symptoms so like those of the less marked forms of general paralysis that it may be impossible to make a diagnosis between the two diseases until one has watched the case for a little time.

Lastly, there may be a difficulty in distinguishing general paralysis from some affections of the spinal cord. There are cases in which an impairment of muscular power shows itself some months before any psychical symptoms develop themselves. It has even been a question whether the disease may not sometimes run its course without being attended at any time with impairment of the mental powers. This question is answered in the negative by alienist physicians; but one may fairly reply that those observers would have no chance of seeing such cases, supposing them to occur. It is in the wards of a general hospital that they must be looked for. Dr Wilks published in the 'Guy's Hospital Reports' (xvi, p. 194) an instance in which the patient's mind was unaffected while paralytic symptoms were well marked. But this patient had only been ill nine or ten months, and in all probability mental symptoms afterwards developed.

There is sometimes great difficulty in distinguishing general paralysis in its early stage from *tabes dorsalis*. Dr Wilks relates that two physicians—one an authority with regard to the former disease, the other with regard to the latter—had the same case shown to them, and that each of them pronounced it to be an example of the malady with which he was the more familiar. The explanation seems to lie in the fact that the two diseases are not infrequently present in the same individual at the same time. In Westphal's cases of grey degeneration of the posterior columns of the cord with general paralysis, he expressly states that the patients had presented the ordinary symptoms of locomotor ataxy. As far back as 1862 Baillarger published clinical reports of five cases of a similar kind. General paralysis is thus a sequel of tabes.

Dr Bristowe has published an important lecture on the occasional difficulties of diagnosis between General Paralysis and *Insular Sclerosis* ('Brit. Med. Journ.,' Jan. 1st, 1887).

The *prognosis* is unfortunately uniformly unfavourable, and is little affected by remedial measures. The course is rather rapid, varying from a few months to two or three years.

Treatment.—In the early stage of general paralysis confinement in an asylum is absolutely necessary. It is, indeed, a question whether it might not sometimes run a more favourable course if the patient were secluded at an earlier period than is usually the case. His friends and relations are but too apt to take him to the sea-side, or even to make him travel from place to place; whereas what his brain really needs is absolute rest. Among medicinal agents Dr Blandford speaks highly of tincture of digitalis in doses of ℥xv to ℥xxx every four hours. He says that it often soothes such patients wonderfully, and restores them from a state of noisy turbulence to one of comparative rationality. According to Hitzig the application of galvanism to the nape of the neck is sometimes of marked temporary utility; he also speaks favourably of iodide of potassium. The bichloride of mercury has been largely used, but without any good result. Dr Blandford lays stress upon the importance of withholding stimulants, such as brandy, while there is excitement; but he says that opium, morphia and chloral are often useful both in the early, and still more in the later stages of the disease. The same writer insists on the

value of tonics when the more acute symptoms have passed off, the tinctura ferri perchloridi and the other preparations of iron being particularly serviceable. Dr Crichton Browne published two cases in which the extract of Calabar bean in doses of a quarter to a third of a grain, continued for nine or twelve months, appeared to cure the disease ('Journ. of Mental Science,' 1875).

Other forms of atrophy of the brain.—In sharp contrast with the definite clinical course of General Paralysis, which disease is chiefly seen in asylums for the insane, there occurs in ordinary medical practice a precisely similar atrophy of the brain, attended with the greatest possible variety of symptoms, or even with no symptoms at all. Clinically, therefore, it is distinct, but anatomically it is identical with the malady just described. This fact has been generally ignored, although Dr Wilks published some observations in reference to it in the 'Journal of Mental Science' for 1864. But that it is not an infrequent condition is evident from notes of no less than fifty cases in which it has been found in the *post-mortem* room of Guy's Hospital within the last twenty years; and that number might be considerably augmented, for only the more striking examples were selected.

The wasting of the cerebral substance is characterised, not only by loss of weight, but also by an obvious shrinking. When the dura mater is turned aside, after the calvaria has been removed, the surface of the hemispheres may appear to be covered with a gelatinous substance, which, however, is really an accumulation of serous fluid in the meshes of the pia mater, and runs out as soon as the arachnoid is punctured, leaving the membranes collapsed and wrinkled. The convolutions are small, and they are separated by deep, broad sulci; the ventricles are often large, the ependyma granular, and the choroid plexuses have undergone more or less cystic degeneration. The cerebral substance itself is in some cases firm and natural-looking. But in other instances it presents patches of softening, either thickly scattered throughout the white matter beneath the convolutions or limited to the basal ganglia. In an old man of seventy-six, whose body was examined in 1876, the cut surface of the hemispheres showed smooth-walled cavities round the vessels, especially in the neighbourhood of the grey matter, so that it had an appearance like that of Gruyère cheese—exactly such as was described by Lockhart Clarke in cases of general paralysis (p. 685). When morbid changes of this kind are present, the minute arteries are, as a rule, diseased. Not unfrequently they are so thickened, and even calcified, that their cut ends look like so many bristles embedded in the brain. The membranes, too, are often thickened and opaque, especially over the hemispheres. It would be difficult to deny that these last appearances are indicative of chronic inflammatory change, rather than of mere atrophy; but they certainly occur where there is no other evidence of inflammation.

The causes of atrophy of the brain vary in different cases. Sometimes it seems to be a *senile change*, and it may then give rise to no obvious symptoms; the old man whose hemispheres resembled Gruyère cheese was said to have been perfectly clear in his mind. In other persons of advanced years, however, there is a more or less marked failure of both mind and body. They are eccentric or odd in manner, or demented. One patient, a woman, aged sixty-eight, who was brought to the hospital for a fractured

thigh, got out of bed, splint and all, and tried to walk about, with the result that the limb became gangrenous. Another, a man, aged seventy-four, who was taken in for bronchitis, showed no special cerebral symptoms, except that he passed his evacuations under him. Others again have been admitted for injuries due to attempts at suicide; a woman had thrown herself out of the window and fractured her spine, and a man had cut his throat. Probably many cases of this kind are indistinguishable during life from those of ordinary insanity, in which the brain remains perfect, so far as our means of investigation enable us to determine.

Cerebral atrophy is not confined to old people; cases occur in persons between forty and fifty years of age. Thus in 1867 there died in Guy's Hospital a man, aged forty-nine, who had been an accountant, but who for some time had been unable to do his work. His memory had failed, his speech had become uncertain, he wrote badly, and he misspelt his entries in a cheque-book. Afterwards his writing improved, but he formed his words very slowly. When speaking he would lose the thread of his discourse, and vainly clasp his head with his hands in search of what he wanted to say. For two or three days before his death he lay in a doze. At the autopsy patches of brown discoloration, breaking down in the centre, were found in each corpus striatum; the cerebral arteries were rigid and calcareous, and the kidneys were granular.

In some cases of this kind the disease advances to a fatal termination within a few months of its commencement; its diagnosis from other organic affections of the brain may then be impossible. There is often more or less complete paralysis of the limbs; sometimes convulsive movements or jactitations occur, and sometimes epileptiform seizures. Headache appears not to be generally a marked symptom; but in one patient it was exceedingly severe for the last six years of his life.

Cerebral atrophy is often the result of chronic *alcoholic poisoning*. A large proportion of those who die of delirium tremens have wasted brains; but there remain cases in which the brain is found apparently healthy.

Another cause of cerebral atrophy is *poisoning by lead*. In 1863 a compositor, aged thirty-four, died in Guy's Hospital, who had been admitted for colic, and who afterwards became almost totally paralysed; the convolutions of the brain were shrunken, the sulci were deep and contained an excess of fluid, the ventricles were unduly large. Chronic plumbism, as is well known, sometimes gives rise to epileptiform convulsions, which may be repeated at intervals, and followed by delirium and by a fatal coma. In cases of this kind the brain is commonly found wasted.

In a patient of the writer's, however, a woman, aged thirty-five, who died after eclampsia, obstinate vomiting, and other symptoms ascribed to plumbism, the brain and membranes after death were found perfectly normal, and weighing 42 ounces. No trace of lead could be found here, although it was abundantly present in the liver, spleen, and intestines. The patient had worked in a white-lead factory, and showed the characteristic blue line; she also suffered from colic and paralysis of the extensors of the hand.

CHRONIC DIFFUSED INFLAMMATION—*General sclerosis of the brain.*—If we consider how important a part in the pathology of the spinal cord is played by diffused chronic inflammation, it is remarkable that similar lesions

are hardly known to occur in the brain.* The author, however, made two autopsies in which cerebral sclerosis was found.

One of them occurred in the practice of Dr Wilks, who recorded it in the 'Guy's Hospital Reports' (Series iii, vol. xxii, p. 22). A girl, aged fourteen, was admitted, in 1876, for general failure of mind and body, which had been coming on for about a year. The earliest symptom was said to have been an attack of weakness and loss of sight, which came on one day while she was out on an errand. Soon afterwards she had two epileptiform seizures, which began with a scream. She generally lay quiet, with a vacant stare, making no complaints, but smiling when spoken to. She could evidently see, and the optic discs were normal, but the pupils were dilated. If asked what her name was, or any other simple question, she answered sensibly, but very slowly. When food was put into her mouth she would cease to chew without swallowing it, so that it lay there until removed by the nurse. She often vomited. She was unable to stand, and had very little power over her legs; even her arms were moved but slowly and feebly. Her skin appeared to be sensitive. A month before her death she tried to strangle herself, and was found with a towel tied round her neck. Towards the last she lay with her eyes open, making no attempt to speak beyond a slight motion of the lips. Her temperature ranged from a little below to a little above normal. Before death her extremities became remarkably livid; and large bullæ made their appearance on the feet, which looked as though gangrene were beginning.

At the autopsy a deep purple discoloration was seen over the feet and ankles, the cuticle was detached from the rete mucosum, and the deeper tissues were all infiltrated with blood. The calvaria was thick and heavy and very unsymmetrical. The membranes were a little opaque, and there was an excess of fluid at the base. Over the convolutions the pia mater was everywhere abnormally adherent, so that in attempting to strip it off one peeled away a thin granular-looking layer of the cortex, leaving a roughened surface. The brain was small, weighing only thirty-four ounces, and its substance generally was tough. The cortex was not obviously wasted, and the boundary line between the grey and the white matter was everywhere well defined. The ventricles were much dilated, but their ependyma was not granular, except in the fourth ventricle.

The other case, one of less clinical interest, occurred in 1878 in a child, eighteen months old, a patient of Dr Pavy's. Its illness was said to have begun with a fit at the age of four months, and from that time it had been subject, especially at night, to seizures, in which it would fight for its breath, and appear to be choking, but would afterwards lie insensible for three days at a time, taking very little food, and twitching at the corners of the mouth. Fifteen days before its death it had a fit more severe than any previous one. Eleven days later it was again attacked, and on the following day it was brought into the hospital. It then lay comatose on its left side with the corners of the mouth drawn down and constantly twitching: when it was moved over, its whole body would become rigid, and the twitching at its mouth was more marked. It appeared to have lost sensation in the left side. The temperature, at first 102·4°, rose to 103·8° before death, and the pulse became scarcely perceptible.

* Acute myelitis is, however, almost as rarely represented by acute idiopathic softening in the brain as chronic sclerosis of the cord by sclerosis of the brain. The various systemic diseases of the cord have also no counterpart in cerebral pathology.

At the autopsy the child was wasted, with a pigeon-shaped chest and rickety enlargement of the ends of the ribs and of other bones. The brain was pale on the surface. The meninges looked healthy; they were not at all thickened or opaque, but very thin and delicate, as is usual at an early age. On attempting to strip the pia mater from the convolutions, however, it was at once found to be abnormally adherent, so that a thin, uniform, superficial layer of the cortex peeled off with it, leaving a rough granular surface behind. The substance of the hemispheres also was indurated, cutting firmly, and giving sections with sharp resistent edges, but the most striking appearance of all was a discoloration of the white matter of the hemispheres, which had a yellowish tint. This was more obvious on the right side than on the left, and in the anterior and lateral parts of the hemispheres than in the posterior parts. It was also more marked in the prolongations of the white matter into the convolutions than towards the centre of the brain, and in some of the convolutions the limit between the white and the grey substances was ill defined. The grey matter itself looked natural. The ventricles were not dilated. The basal ganglia, the pons, the bulb, and the cerebellum were all of their natural soft consistence.

Portions of the affected tissues from each of these cases were examined after the most approved methods by Dr Savage, of Bethlem Hospital, and also by Dr Frederick Taylor, but no histological change could be detected, except possibly a slight excess of corpuscles in the interstitial neuroglia.

Dr Gee has reported two cases of general cerebral sclerosis occurring in children: in one, a girl of eleven, there was spastic paralysis, and after death the brain was found to be small and firm, with excess of leucocytes in the lymph-sheaths of the cerebral arteries; in the other, a girl of ten, there was right hemiplegia with indistinctness of speech, and there was found atrophy of the left hemisphere with general induration of the brain ('St Barth. Hosp. Reports,' vol. xvi).

HYPERTROPHY OF THE BRAIN.—It has long been known that in certain cases in which the head appears unduly large, so that the presence of hydrocephalus is suspected, a solid brain really fills the whole interior of the skull. Instances of this kind were recorded by Morgagni; and others in the early part of the present century by several French observers, among whom were Laennec and Andral. The condition has generally received the name of hypertrophy of the brain; but its nature and its relations to other diseases still remain among the most obscure questions in pathology. Virchow several years ago suggested that it consisted in an overgrowth of the neuroglia rather than of the nervous elements of the hemispheres. Sir William Jenner, not long afterwards, maintained that it was due to an "albuminoid infiltration" of the tissues, like that which he believed to cause enlargement of the liver and spleen in rickets; he regarded it, in fact, as one of the minor effects of that disorder. But it appears to be certain that the subjects of cerebral hypertrophy are not always rachitic.

Magnan is said to have minutely examined the structure of a child's brain so affected in 1874, and to have found it perfectly normal. D'Espine and Picot accordingly still describe the disease as a true overgrowth of all the various elements of the cerebral substance. This conclusion, however, we can scarcely accept, not only because of its inherent improbability, but still more on account of there being other lesions of the brain (*e. g.* "diffused chronic cerebritis" and "yellow softening") which are easily recog-

nised by the naked eye, but in which the microscope equally fails to reveal marked histological changes. Indeed, it is worthy of notice that in each of the two cases of which Andral has given full descriptions, the grey substance of the convolutions is said to have been undistinguishable from the white, while the medulla resembled a hard-boiled egg, and in some parts was harder still. How can such a condition be distinguished from sclerosis?

Dr Fletcher Beach, of the Darenth Asylum, kindly furnished the following notes of two among six cases of this affection that have come under his notice. He found that the white matter showed "a uniform granular appearance under the microscope, with nerve-cells scattered sparsely throughout. There were a number of leucocytes present. The increase in size was evidently due to the large amount of granular matter."

In each instance the size of the head was a conspicuous feature during life. In one, a boy aged sixteen, it measured twenty inches in circumference; in the other, a boy aged ten, the measurement was twenty-two inches. Its form was, in each instance, square, not, as in hydrocephalus, round.

Other points of distinction from hydrocephalus are that the enlargement is more marked just above the superciliary ridges than at the temples, the eyes are not so wide apart, and the fontanelle is often depressed instead of being full and elastic. This last character seems hardly to consist with the accounts which have been given of the dura mater bulging as soon as the skull was opened, and of the convolutions being flattened. Although the brain in the older of Dr Beach's patients weighed no less than sixty-two ounces, the subarachnoid fluid was increased, and the ventricles were rather dilated.

None of Dr Beach's patients presented any signs of rickets, but, as he himself remarks, it is possible that they may have been rachitic at an earlier period, since they were, with one exception, above the age of ten years when they came under his observation.

Clinically it would seem that hypertrophy of the brain is characterised by a more or less marked deficiency of intelligence, drowsiness, headache, and liability to epileptiform convulsions; the gait is sometimes slow and tottering, and the weight of the head may cause it to hang forwards, or may throw the patient down upon his face when he is walking.

Under favourable circumstances life may be prolonged for years. The disease often ends by some intercurrent pulmonary affection, or by the supervention of acute symptoms and coma which, in one of Dr Beach's cases, were due to acute suppurative meningitis.

With regard to treatment nothing is as yet known.

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM

THE SPASMODIC NEUROSES

"Una senum facies, cum voce trementia membra."—JUVENAL.

Arrangement of the Neuroses and particularly of Spasmodic Neuroses.

LOCAL SPASMODIC NEUROSES—*Facial or Histrionic Spasm—Spasmodic Wryneck.*

Reflex and Fatigue Spasms: Saltatorial and Salaam Spasm—Spasmus nictitans—Singultus—Athetosis, etc.—Scriveners' Palsy, Hammer Palsy, and other handicraft-spasms.

Tetanilla—Description—Relation to Trismus neonatorum, Laryngismus, and Carpo-pedal contractions, and of all to Rickets—Ætiology—Treatment.

Thomsen's Disease—Its history, characters, and symptoms.

PARALYSIS AGITANS—*History—Symptoms—Pathology—Diagnosis—Ætiology—Treatment.*

TETANUS—*Symptoms—Varieties—Ætiology—Pathology—Diagnosis—Events—Acute and chronic course—Mortality and Prognosis—Treatment.*

CHOREA — *Nomenclature — Symptoms and course — Chorea gravis — Morbid anatomy—Statistics—Pathology and Ætiology—Relation to Rheumatism and Cardiac disease—Embolic theory—Treatment.*

Congenital choreiform spasms of cerebral origin—Hereditary chorea in adults—Myoclonus multiplex—Dubini's disease.

WE have now completed the survey of those diseases which can be more or less certainly associated with organic lesions of the nerves, the spinal cord, or the brain. We have said nothing of diseases of the "sympathetic system," because, in the first place, the nerves and ganglia so named are not independent, but form part of the spinal system; and because, in the second, there is no evidence of any clinically recognised disorder depending on derangement of the "sympathetic" nerves and ganglia.

There remain the Functional disorders of motion, sensation, and intelligence, for some of which perhaps a special morbid anatomy is yet to be discovered; while others in all likelihood depend, not on lesions which can ever be distinguished by the scalpel or the microscope, but on nutritive disorders which pervert action without perceptibly altering structure.

We found it desirable to have regard to clinical characters even in the arrangement of organic diseases; and, in our ignorance of the causes and nature of almost all functional disorders, we have no choice but provisionally to classify them by their symptoms and course.

The first group of Neuroses may be termed Spasmodic,* for all of them are marked by spasms or cramps in the voluntary muscles.

FACIAL SPASM.†—This is a disorder for which we have no distinctive title in English. Dr Sieveking, in translating Romberg, introduced the term *histrionic spasm* as an equivalent for the German name, but it does not carry its meaning with it, and has not been generally accepted.

Its essential feature is the occurrence, at longer or shorter intervals, of a sudden and rapid involuntary contraction in a single muscle or group of muscles. One eye may be instantaneously closed and opened, or the forehead may be wrinkled on one side, and as quickly made smooth again; or the angle of the mouth may be twitched to one side, so as to cause a meaningless grin. Or, again, the head may be made to nod forwards, or the neck may be distorted to one side, or the shoulder shrugged, or the diaphragm may contract and cause the utterance of a sharp cry.

The person himself is often unaware of the complaint, which is, indeed, scarcely more than a morbid habit or trick. Trousseau says that he was consulted by a lady whose three daughters had muscles in different parts of the body affected with spasmodic tic, for which she found great fault with them, without knowing that she herself had it in her face.

Another point which this writer mentions is that the movements sometimes shift from one part and attack another. Thus a patient came to him for violent spasms of the head and one shoulder: methodical gymnastic exercises were prescribed; and after some time the affection disappeared from the affected shoulder, and presently showed itself in the other. A similar instance is related by Sir Thomas Watson, of a gentleman who when young used to give an involuntary shake of his head: a blister, applied to his throat for some affection of the air-passages, made this movement painful, and the movement ceased; but, to use his expression, it broke out in his nose, so that he was ever afterwards in the habit of wrinkling it. A boy, aged twelve, four years before the author saw him, had acquired a trick of sniffing and coughing, as though he would be choked; this was followed after two years by a habit of "making faces;" and a month before he was examined he began to toss his head over to the right side at intervals of a few minutes, whereupon the sniffing and coughing stopped.

Sometimes, however, an affection of this kind lasts for a whole lifetime unchanged. Trousseau mentions that after the lapse of twenty years he recognised a former schoolfellow, who happened to be walking behind him, by a sort of barking noise that he made.

It does not appear that these histrionic or pantomimic spasms are ever influenced by treatment, but probably they might be corrected when they first appear in children, in the same way as any other bad habit. The application of the continuous current to the portio dura is, however, advised.

SPASMODIC WRYNECK.‡—Beside the form of spasmodic tic just mentioned, which consists in twitching of the patient's head to one side, the

* Ποιεῦσιν δὲ αὐτῶν (sc. Λιβύων) συχνοὶ τοιάδε· τῶν παιδίων τῶν σφετέρων, ἐπὰν τετραέτεα γένηται, οἵσπῃ προβάτων καίουσι τὰς ἐν τῇσι κορυφῇσι φλέβας . . . ἢν δὲ καίωσι τὰ παιδία σπασμὸς ἐπιγένηται, ἐξεύρηταί σφι ἄκος· τράγου οὖρον σπείσαντες ῥύονται σφέα.—HEROD., iv, 187.

† *Synonyms.*—Histrionic spasm—Clonic spasm in the area of the portio dura.—*Fr.* Tic convulsif—Tic non-douloureux faciale.—*Germ.* Mimischer Gesichtskrampf.

‡ *Synonyms.*—Torticollis—Tic rotatoire—Nickkrampf—Clonic spasm in the area of the nervus accessorius.

cervical muscles are liable to another kind of spasm, which is called "wryneck" or "torticollis." This is characterised by a rapid succession of jerking movements, which draw the head with great force towards one shoulder, and give rise to extreme deformity. When one sterno-mastoid is chiefly involved, the corresponding ear is pulled down towards the clavicle, while the chin is pushed upwards, and the whole face is thrown to the opposite side. When it is the trapezius, the head is drawn backwards, without rotation of the chin, the shoulder being at the same time raised.

One or more muscles in a state of powerful contraction can generally be felt, or even seen, through the skin.

Ætiology.—Spasmodic wryneck occurs chiefly in adults. Dr Reynolds says that all but one of the patients whom he has seen have been more than thirty years of age, and the majority more than forty. However, one well-marked case has recently been observed at Guy's Hospital in a girl, aged eleven, who had had it ever since she was a baby. Men and women appear to be affected in about equal numbers.

This disease is often directly traceable to cold. Thus Dr Golding-Bird ('Guy's Hosp. Rep.,' vol. vi) had a patient who was attacked shortly after having been thrown into a state of partial stupor by driving across an open country in a gig one severe winter's night. And Dr Bright relates the case of a woman who was sitting exposed to a draught of cold air, when she suddenly felt what she thought was "a nerve giving way" on the left side of her neck, whereupon her head was drawn to the right. We had two instances in which the complaint appeared clearly to be the result of a fall upon the head. Very often, however, no exciting cause can be discovered. The patient, if a woman, may have been pregnant when the complaint began, or it may have followed a severe mental shock or an attack of fever; but none of these conditions precede it often enough to be regarded as even predisposing causes. Dr John Harley has reported, in vol. lvii of the 'Med.-Chir. Trans.,' the case of an engraver, whose head was habitually kept raised and slightly rotated to the right, so that he might have an oblique view of the plate on which he was engaged. He also mentions the case of a governess, who spent much of her time in ruling the pupils' copy-books, and who used to rotate her head rhythmically and with emphasis from left to right; she, in her declining years, lost her control over this movement; and her face was twisted every second to the right shoulder, unless her attention was strongly engaged in some other matter.

Irritation from disease of the teeth is no doubt a cause of wryneck. In two such instances, however, the spasm seems to have been *tonic*. One is Mr Hancock's case ('Lancet,' 1859, i, p. 80) of a girl, whose head for more than six months had been drawn down nearly to the left shoulder; extraction of a stump and of a decayed tooth in the left side of the lower jaw completely cured her in a few days. The other case, which occurred in 1813, is related by Mr Mitchell in the fourth volume of the 'Med.-Chir. Transactions.' The spasms began in the tongue and side of the face, and only affected the neck after the lapse of some days; but precisely similar changes, in the extent to which different muscles are involved, occur in the ordinary clonic form of wryneck. The patient was a woman, aged fifty; during the attacks her neck was drawn round to the left shoulder, her arm was rigidly extended, her eyelids were widely opened, and the two eyes directed to the left; her mouth was opened and distorted to the left side, showing the clenched teeth; her tongue felt as hard as a board, and was

curved to the left side; her forehead was wrinkled, and all the muscles of her face were thrown into a state of rigid distortion. After three months the tonic spasm was succeeded by a rapid tremulous motion of the affected parts, and the attack passed off, to be repeated at intervals which rapidly grew shorter, so that a fortnight from the commencement of the disease the spasms became almost continuous. Extraction of some carious teeth and fangs in the left upper jaw was quickly followed by a cure.

Course.—In cases not directly traceable to cold, the development of the complaint is generally very gradual. Dr Reynolds describes the patient as at first feeling uneasy in his neck, and as thinking that something is wrong with his cravats or with the pillow, until after some months he discovers, or is told by a friend, that his head is not straight. During the early progress of the case, an ordinary observer might suppose that the man's shirt-collar was uncomfortable, and that he was trying to ease it by twitching his neck; or that he was trying to look over his shoulder. Pain is at first absent, but after a time he complains of a dull, aching sensation, which extends to the shoulder or down the arm. At first, by an exercise of the will, he can overcome the spasm, so as to look straight before him, or towards the affected side; even at an advanced stage he can sometimes by a very powerful effort restore the equilibrium of the two sides for a moment, but at the cost of much distress, and with the risk of aggravating the severity of the spasms afterwards. As the disease progresses, he generally gets into the habit of bringing his head into the proper position with his hands. Thus a patient used to walk about with his arm raised, and ready to seize hold of his nose, which he employed as a kind of lever to control the movements. The spasms are always arrested during sleep, and they often cease when the patient is in the recumbent posture, and when he supports his head with his hands. They are at once brought on by any excitement, or by talking, or by attempting to walk. In the more severe cases the head is jerked about with extreme violence, and this goes on for hours or even for days without any interval. Sleep is almost impossible, and the patient's state is one of the utmost misery.

Spasmodic wryneck may remain stationary for years, neither advancing nor receding. But sometimes it extends from the parts first affected to the side of the face and the eye, or to the shoulder and arm. In two cases at Guy's Hospital the movements began in the upper limb and afterwards attacked the neck. This, and the fact that the deep cervical muscles are sometimes the ones mainly concerned, prove that the disease is not specially associated with the sterno-mastoid muscle nor with the distribution of the spinal accessory nerve, as would appear from the description given by Erb and others. In some cases it passes off within a few months under treatment, and a long while afterwards returns with its characters unaltered. Thus in a case of Dr Golding-Bird's, already referred to, there was an interval of about seven years between the first and second attack; and in another case the patient got well, returned to his work as a gasfitter for thirteen years, and at the end of that time was again seized with the disease, but on the opposite side.

Wryneck does not appear, like spasmodic tic, to desert one part of the body in order to attack another immediately afterwards.

In the advanced stages of the disease, the muscles which are the seat of the spasms sometimes fall into a state of *tonic* contraction, so that for hours together the head remains obliquely drawn down to the shoulder but

perfectly motionless. Even then, however, any kind of emotional excitement, or the slightest attempt at voluntary movement, generally leads to the development of clonic spasms also.

Writers have described primary *tonic wryneck* as a separate form (*caput obstipum spasticum*), for in some cases there is no history of the occurrence of the ordinary jerking movements at any period. Thus a girl's head was drawn down so that the chin rested on the second left costal cartilage, and the right side of the lower jaw was so close to the sternum that it was difficult to introduce a finger between them. Very slight twitching movements were all that had been noticed, but the affection was said to have developed itself while the patient lay paralysed in bed, unable to move a limb, and this perhaps accounts for the absence of clonic spasms. The girl was the subject of well-marked hysteria, and probably the wryneck was of the same nature as hysterical contractions of the limbs.

Results.—One curious effect of wryneck when it occurs in childhood is the permanent deformity in the bones of the face to which it gives rise. In the case just alluded to the left side of the face was considerably larger than the right. When the girl's head was erect, the right eye lay at a higher level than the left one, the median line of the face, instead of being upright, formed an obvious curve, with its concavity towards the left, and the left side of the upper jaw projected beyond that of the lower jaw, while on the right side the relation between them was reversed. The vertebræ of the neck were also distorted, there being a projection on the left side, apparently caused by the transverse process of the atlas. Precisely similar deformities were noted in a little girl of eleven, who had been in the hospital a few years before. They are of importance, because they might be regarded as indications of primary disease of the vertebræ, such as often produces a prominence of the cervical spine on one side, and also causes impairment of the movements of the head.

Treatment.—This is very unsatisfactory: on the one hand many cases are altogether intractable and are not benefited by any curative measures; on the other hand those in which relief is afforded, or which get perfectly well, yield to the most diverse remedies, which must differ altogether in their operation; moreover, each in turn fails more often than it succeeds. Dr Reynolds speaks of galvanism as being very useful in cases which are not already of too long standing. He finds that a continuous current of moderate intensity, passed through the muscles which are the seat of spasm, causes them to relax, at least for a time. But he adds that even when by this means the head has been maintained in equilibrium for many minutes, day after day, he has often observed that, as soon as the poles are withdrawn, the spasms instantly return; and sometimes it has been obvious that the ultimate effect of the treatment has been to increase the movements. In a patient who some years ago came under Dr Habershon's care at Guy's Hospital a considerable amount of relief was afforded by the plan of fastening the sponges upon different parts of the neck, and leaving them for some hours at a time. Faradisation of the muscles on the opposite side of the neck is sometimes useful, by increasing their power of resistance. On the affected side there is an exalted sensibility by interrupted currents; the patient may be altogether unable to bear the application of one which is so weak as to cause no pain whatever on the healthy side.

Rest is of course essential; and it would seem desirable that some mechanical support should be provided which may keep the head in its proper position. Dr Reynolds, however, says that he has not yet seen any apparatus which a patient with confirmed wryneck could habitually wear, but that the appliance suggested by Dr Hearne, of Southampton, is useful in recent cases, by controlling the movements for an hour or two; as, for instance, when a clergyman wants to get through a service.

Another measure of which he speaks highly is the hypodermic injection of morphia. Beginning with a tenth of a grain, he recommends that one should, if possible, increase the dose until two or even three grains are administered twice daily.

In one case recorded by Dr. Radcliffe ('System of Med.,' ii, p. 133) arsenic was injected subcutaneously with very striking results. From five to fourteen minims of Fowler's solution were thrown into the connective tissue over the affected muscles at intervals of three or four days or longer; they caused much local irritation and inflammation; but this was afterwards diminished by diluting the solution with water. After the fortieth injection, at the end of about nine weeks, the patient left the hospital almost well. This method of treatment has, however, been tried at Guy's Hospital by Dr Wilks and also by Dr Moxon without success.

Dr John Harley has recently ('Med.-Chir. Trans.,' lvii) related two cases in which the administration of large doses of succus conii proved very useful. Beginning with an ounce of the liquid, he rapidly pushed the dose in each case until one patient took three and a half ounces once daily, and the other four ounces twice daily. The effect aimed at was the production of a general relaxation of the muscles, so that at the end, the second hour after swallowing the hemlock juice, the patient should be unable to rise from the sitting posture or to walk unassisted, and that there should be ptosis, impairment of power to masticate and swallow food, and slowness of speech. In the first case the spasm became greatly diminished; but the drug had ultimately to be discontinued on account of the mental depression to which it gave rise. In the second case the affection was almost cured, and the patient resumed his occupation, working half-time, with only an occasional tendency to a twist of the head if he became over-tired. Dr Moxon has followed Dr Harley's plan with a decided measure of success.

In a case recently in Philip Ward the constant galvanic current failed to do good, but the patient gradually improved under a mixture of bromide of potassium and arsenic, and went out apparently cured.

A severe plan of treatment, by which Busch succeeded in curing several cases permanently, is the application of the actual cautery to the neck on both sides of the spine, in lines five or six inches long; he kept up suppuration for some weeks after the separation of the eschars.

In one very severe case ('Med.-Chir. Rev.,' 1866) Mr Campbell de Morgan excised an inch of the trunk of the spinal accessory nerve, above the origin of the branches to the sterno-mastoid muscle, with permanent relief to the spasm, but with equally permanent paralysis of the affected muscles. Another operation which has been performed several times with good results is subcutaneous section of the sterno-mastoid muscle.

In those cases in which the spasm is tonic, it is advisable to straighten the head under chloroform, and to endeavour, by mechanical appliances, to prevent the return of the contraction.

FATIGUE-SPASMS.*—Within the last few years several observers have drawn attention to cases in which spasmodic contractions of various kinds are brought about by voluntary movement, the exciting cause in each instance being generally limited to some one particular action, so that the patient remains quiet when sitting or lying down, and is able to use all his muscles for other purposes perfectly well. One of the best papers on the subject is by Dr Weir Mitchell in the 'American Journal' for 1876. His name for this group of affections is that of "functional spasms;" but this might be taken as meaning "spasms which are functional and not organic," in which sense it would include all the diseases described in this chapter. "Function-spasms," "Movement-spasms," and "Fatigue-spasms," are terms which, if less euphonious, are also less misleading.

Many of Dr Mitchell's cases are exceedingly curious. Two are examples of what he terms "lock-spasm." A watchmaker, who often had to pick up and adjust very small screws, would find ten or twelve times a day that his thumb and forefinger suddenly became locked upon one of them, so that he had to release it with a loop of twine, not always without wounding the finger; even then the cramp would last for some considerable time longer. At a later period, when he was turning over the pages of a book, the finger and thumb would sometimes close with violence, so as to tear the leaf. Another man, a sawyer, was liable once or even twice a day to have the arm arrested, and fixed in a state of flexion at the moment when his saw was drawn back to prepare for the downward movement. By no effort that Dr Mitchell could exert was he able to overcome the spasm; on one occasion he made the patient bend over, so as to bring the forearm into a horizontal position, and he then found that for five minutes the biceps supported a weight of eighty pounds suspended from the wrist. Such cases are clearly analogous to the cramps of writers, pianists, telegraph workers and milkmaids (p. 702).

In other cases, the attempt to walk was accompanied by a tonic spasm of certain muscles, so that a kind of stringhalt was produced. Another patient, a journeyman tailor, had an extraordinary seizure whenever he jumped up suddenly from the prone or the sitting posture. The right leg was then bent at the knee, the left was thrown over it in violent flexion, the body and the head were twisted to the right, the right arm was extended and raised, the left was thrown outwards and backwards in extreme pronation. Then there was a general writhing of the whole frame, the facial muscles twitching here and there; and the attack passed off with a groan of relief. This man could make every possible movement, slowly or abruptly as he pleased, so long as he remained seated or lying down: he was even able to get up; and after cautiously standing still for a moment, could walk away as well as anyone else. Such cases resemble the saltatorial spasm to be presently described, and also the hereditary cases described by Thomsen (p. 712).

In other cases, again, spasms accompanied the act of laughing or talking, or, above all, chewing. One patient, as soon as he began to masticate his food, had his mouth jerked open, so that he had to keep shutting it with his hand. When he wanted to swallow, his face assumed an aspect of terror.

* *Synonyms.*—Functional spasms (Weir Mitchell)—Movement-spasms. This group includes most Reflex spasmodic neuroses, Saltatorial spasms and the Beschäftigungsneurosen of German writers, viz. writers' palsy, fiddlers', tailors', cigar-makers' cramp, lock-spasm, milkmaids' cramp, &c.

"He would suddenly muster courage, and swallow the contents of his mouth at a gulp. Then instantly the jaw flew open, the head was drawn back and down upon the left shoulder, the face was convulsed, and sweat ran from the forehead." In another case of the same kind the gastrocnemii were involved in the spasm, so that the patient was jerked into a squatting position. An instance came under the author's observation in which the chewing of food brought on a kind of wryneck, the head being twisted over to one shoulder; and another, in which the effort of speaking was attended with an extraordinary series of grimaces. These cases again are pathologically identical with the spasms brought on by handicrafts.

Saltatorial spasms.—Perhaps of all this group of spasmodic neuroses called forth by voluntary or reflex movements, the strangest are certain cases in which any attempt to stand excites violent convulsions. The patient is thrown off his feet, his legs pass into a state of clonic spasm, and he begins to execute a succession of the most rapid and irregular jumping and skipping movements, even though he may be supported on each side by another person. So great are his efforts that his face flushes, his pulse is quickened, and he breaks out into a profuse sweat. Yet when he is lying or sitting he remains quiet, and can move his legs perfectly well in all directions. For this affection a special name was invented in 1859 by Bamberger, who recorded in the Vienna 'Med. Wochenschrift' two cases of what he called "Saltatorial reflex-spasm, a remarkable form of Spinal Irritation." Dr Gowers described two cases of the affection ('Lancet,' July and August, 1877), with an analysis of the five previously recorded. In their details these cases differ to some extent from one another, and this affords an additional reason for including them under "reflex fatigue-spasms."

Sometimes it has been possible for the patient to excite the movements by pressing with his feet against the foot-board of the bed, even while he is lying down; in one instance, tickling the skin of the soles would elicit them; in another, pressure under a tender part of the spine had the same effect. They have often been much augmented by emotional excitement; and this has sometimes led to doubts as to their genuineness, it being found that they are much less violent when no one is looking on; but the same thing may be said of many other affections belonging to the same class. Erb suggests that saltatorial spasms will ultimately be found to be a kind of exaggerated tendon-reflex; but Dr Gowers found no ankle-clonus in his cases. No doubt the movements are due to the combined and simultaneous action of peripheral and voluntary stimuli upon irritable centres in the cord; but the same stimuli are in action in all healthy persons whenever they stand upright. In Bamberger's first case, that of a youth of nineteen, the affection came on during convalescence from acute pneumonia; recovery took place in about a month under the internal administration of morphia. Other cases have been much more protracted; in one instance the spasm had not subsided when the patient died of fever at the end of five years. There have been two cases in women, both of whom were affected with hysteria; one had several attacks of the spasm, each lasting some months; her recovery was once quite sudden, exactly as so often happens in hysterical contractures.

There is sometimes pain in the contracting muscles, sometimes none; there is no loss of power or of sensation.

The reflex arc does not appear to begin in the skin of the feet, but in deeper structures, probably in the tendon. It would therefore be analogous

to knee-jerk. In future cases the condition of this phenomenon and of ankle-clonus will no doubt be investigated.

The muscles of the trunk and head are sometimes affected, as well as those of the legs, but the arms escape.

This curious affection has been observed in both sexes, and at all periods of life, from childhood to old age. It appears to be sometimes associated with spastic paraplegia as a sequel of some form of myelitis. But most cases seem to be "functional," and occur with hysterical symptoms. What was called Chorea Major, or dancing madness, would probably in a sporadic form approach some of the cases of saltatorial spasm in young women. It has also relations to tetany (p. 707) and to post-hemiplegic chorea (p. 569).

Saltatorial spasms, once begun, may continue for months and years, or may return after disappearing. They do not appear to lead to further symptoms or to threaten life. No special treatment is known.

Other varieties.—Many other rhythmical spasms may be observed, chiefly in patients not suffering from organic disease. One variety is what has been called "Salaam palsy" (*Eclampsia nutans*), a habit of continually bowing the head as if making a profound reverence. It has been frequently observed in children. Similar spasms may affect the arms, as in a boy once in Guy's Hospital, under Sir William Gull, who was continually moving one arm up and down (*Malleatio*). The writer remembers a man who used, apparently without knowing it, to lift up one leg every third or fifth step in walking and strike it with the hand. Such a trick, again, has relations with such scarcely morbid and unconscious motor habits as Dr Johnson's of counting the posts, and the late Prof. De Morgan of touching the railings one by one as he went to college.

Clonic spasms may affect individual muscles of the face or limbs. When the eyelid of one side is the seat of the disorder, it has been named *spasmus nictitans* or *blepharospasmus*. Singultus or hiccough is a clonic spasmodic affection of the diaphragm.

Athetosis is the name given by Dr Hammond of New York to slow rhythmical clonic spasms of the hands. It has been already mentioned (p. 569) as an occasional sequela of hemiplegia.

Stammering and stuttering (*balbuties*) is due to laryngeal or labio-lingual spasm, or to both, and exhibits the same union of irregular contractions, with loss of power, and explosive completion of a co-ordinated function, which we have had occasion to notice in the whole group of which we are here treating. Some of these spasms are clearly reflex. Some resemble histrionic spasm, some saltatorial cramp, some hysterical movements, chorea major, and "ugly tricks" in children. Many are curable by discipline, or a shock to the imagination, or by growing out of them.

In some of his cases of function-spasm Dr Mitchell found benefit from the injection of solution of atropine into the affected muscles.

*Spasmodic affections caused by habitual handicrafts.**—There is a class of cases which evidently belong, pathologically, to the same group as the last, but which differ from the rest in the fact that the involuntary motions that attend them are only just sufficient to disturb the due execution of some

* There is no name which would conveniently include this group of spasmodic affections; that of "co-ordinated-business-neuroses," proposed by Benedikt, is somewhat unwieldy, and "occupation-spasms" does not seem quite right. "Handicraft-spasms," perhaps, might serve.

highly specialised action, involving numerous and complicated muscular movements, and that over-exercise of this action appears to produce the spasms. They are examples of voluntary "movement-spasms," of what Dr Poore calls "fatigue diseases."

A musician, for example, becomes unable to play the piano or the violin, whichever happens to be his instrument; a telegraph clerk cannot word Morse's machine; a tailor or shoemaker no longer has the power to sew; a milkmaid ceases to be able to press the milk from the cow's teats; a smith to bring down his hammer upon the anvil. In one case a man, whose whole occupation was to clean knives and boots and shoes for a large house, became completely incapacitated for his work, although he could still do everything else. Far more numerous are those in which the complaint consists in an inability to write, and this deserves special attention.

*Writers' cramp.**—The exact nature of the impairment in the power of writing varies widely in different cases of this disorder, so that hardly any two are alike. Sometimes the patient's complaint is that after he has been writing for a few minutes, his hand and forearm feel wearied; sometimes that the fingers become suddenly extended, so that the pen drops from his grasp; sometimes that the index finger is straightened and drawn off the pen-handle; sometimes that the fingers are pressed too tightly upon the pen, so as to impede its movements. In many cases there is no visible cramp or spasm; all that a looker-on can see is that the motions of the hand are arrested in the act of writing; but in other cases there is obvious shaking of the pen.

The handwriting is often altered in character; in some instances the change consists in a reduction of the size of the letters, the power to make free strokes failing, and the writing becoming "cramped;" other patients become unable to accomplish a "running hand," and are obliged to confine themselves to a slow "round text." The author had one patient who could write anything but shorthand perfectly well, but unfortunately he was a law reporter. In the great majority of cases, however, the peculiarity consists in an uncertain tremulous formation of the strokes which make up the letters. When the affection is severe the handwriting becomes illegible—a meaningless succession of shaky lines and curves.

Persons affected with writers' cramp adopt all sorts of odd devices. Some will write only with a quill, and, so far as it goes, this is undoubtedly a good plan, for the muscular effort required is much less than with a steel pen. Others employ a large, thick penholder, or fix a nib in a broad piece of cork. Dr Poore ('Practitioner,' xi) mentions the case of a man who used a rounded mass of wood of the shape of a boy's top, which he held tightly with the hollow of the palm of his hand. Others grasp a penholder with the closed fist, or fasten it to one finger by means of a ring.

Sometimes, however, the fault seems to be not so much in the hand as in the wrist and forearm. A patient of Dr Poore's said that the first thing he noticed was a difficulty in bringing down the hand upon the paper, and for three days he forced himself to write by holding down the right wrist with the left hand, but at last that resource failed him. Indeed, one of the most important characteristics of "writers' cramp" is that it is a progressive disease. For a time a person affected with it may succeed

* *Synonyms.*—Scriveners' Palsy.—*Fr.* Crampe des écrivains.—*Germ.* Schreibekrampf.—Penman's spasm—Mogigraphia (μόγις, with difficulty; γράφω, write)—Graphospasmus.

in accomplishing the art of writing by bringing different sets of muscles into exercise. Thus, instead of employing the weak muscles of the thumb and fingers, he may use those of the forearm; or he may place the arm in some extraordinary attitude as soon as he takes up the pen, resting, perhaps, his wrist upon the table, and raising his elbow at an angle; or pressing the limb close to the side of his body; or, again, straightening the elbow, and holding the instrument at arm's length. But if he persists in writing much all these resources soon fail him. The muscles which he has called in to his assistance "give out" in their turn. Even if he should have taken the pains to learn to use the pen with the other hand, that too is after a time attacked. At advanced periods of the disease the muscles are often affected with spasmodic contractions of considerable violence independently of all efforts at writing. Dr Poore's patient complained that his hand would sometimes bounce suddenly out of the side-pocket of his coat while he was walking in the street, and he once broke a jug in consequence of an unexpected spasm while he was pouring out some water.

In some cases the spasm extends to the muscles of the neck and body. Dr Reynolds mentions an instance in which the effort to write caused the head to be drawn downwards to the right shoulder and the trunk to be contorted, so as to be concave on the right side. The same writer also states that he has seen, in association with writers' cramp, torticollis, occasional strabismus, stammering, and palpitation of the heart.

There is not generally any impairment of sensation. Sometimes the patient complains of "numbness," or "tightness," or "coldness," or of some peculiar sensation which he cannot define in one particular finger, or running from some part of the hand up towards the arm. In some rare cases anæsthesia has been observed. Very commonly the act of writing, if continued for any length of time, causes the hand and wrist to ache, or even the shoulder or the spine. These abnormal feelings distress the patient to an extent which seems altogether disproportionate to that of the actual pain which he has to endure. Indeed, a peculiarity is often observable in the psychical state of those who are affected with writers' cramp in its more advanced stages; they are highly irritable and excitable, and very desponding about their complaint. Dr Poore speaks of one man as having been apparently on the verge of suicide, and of another as having actually taken up a knife with his left hand and stabbed himself on the wrist. Such patients are generally nervous, and they are less than ever able to write when others are looking on, or when the subject-matter is of special importance. Dr Poore was told by one person that he got on better when copying than when composing what he wrote; and another patient had to watch the point of his pen, for if he looked away from it his handwriting at once became unsteady. Those who are affected with this disease are also said to sleep badly, and to be ill-nourished, with soft flabby muscles. Dr Poore has often noticed a thin, brittle state of the nails.

Ætiology.—It is generally assumed that the cause of writers' cramp, and of the affections allied to it, is over-use of the muscles, or working too hard and too long at that particular kind of labour which becomes accompanied with the spasm; and it is undoubtedly true that most of those who are attacked are clerks, or accountants, or in some way professionally engaged in writing. Moreover, as Dr Poore points out, the commencement of the disease often dates from the time at which some heavy task has been accomplished, as when an architect has worked against time to

complete the details of a plan, or a lady to finish an etching for a bazaar. Yet some persons strain their powers to the utmost, and get through extraordinary amounts of writing, without suffering from scriveners' cramp; and others are attacked who have never written more than is safely accomplished by most persons. The author met with two such cases: one in a boy, aged fifteen, whose education seemed to have been rather neglected than otherwise; and one in a lady of fashion, who had at most to write a few notes to her friends. Dr Poore speaks of an instance having come under his observation in which three generations in a direct line were affected. Another of his patients referred the complaint to the severe shock caused by an alarm of fire.

Pathology.—The seat of this neurosis is doubtful. One theory, which is supported by Reynolds and by Erb, is that it lies in the central ganglia which effect the association and co-ordination of muscular movements for the more complex actions. The nutrition of these structures is supposed to be impaired as the result of over-exertion, and the consequence is a perversion of their functions, which expresses itself in irregular spasmodic movements. A strong point in favour of this view is the fact that when one arm has been affected the other is often attacked afterwards; and the relations which writers' cramp bears to torticollis and other spasmodic neuroses afford a further argument.

It seems more likely that the motor centres in the cortex are affected than those in the anterior cornua.

The other theory, which was first proposed by Zaradelli (1857) and is ably advocated by Dr Poore, is that the starting-point of the morbid change is exhaustion of some particular muscle or set of muscles—generally one of the small intrinsic muscles of the thumb or fingers—which are kept constantly in action throughout the whole time that a pen is held in the hand. One or more of these, it is suggested, begins after a time to respond sluggishly to the stimulus of the will. The patient then unconsciously calls into play other muscles, generally those of the forearm. In their turn these too become worn out, and thus the process of substitution is carried on, always with the same result. Dr Poore has in a large number of cases of writers' cramp tested the electric reactions of the muscles which are specially engaged in the act of holding a pen, and has found that often one or more of them showed a marked impairment of irritability as compared with those of the opposite limb.* It might be thought that a strong argument against the theory in question is afforded by the well-known and remarkable fact that a patient who is severely affected with the disease is often perfectly able to perform other actions. But Dr Poore argues that the utility of the affected muscles for other purposes is only apparent; the hand and forearm, he says, are so bountifully furnished that for most movements there are more muscles than are required. If a man finds some of them to be weak, he unconsciously substitutes others in their place to do the work required. This writer maintains that certain among the more delicate actions of the hand are generally interfered with beside those which give to the case its principal character. Thus one of his patients could no longer hold a knitting-needle; another was unable to take up her

* Electrical irritability is generally deficient, as Dr Frank Smith found to be the case in hammer palsy; but Dr Dubois, of Berne, has lately stated that in early cases of telegraph-workers' cramp faradic and galvanic irritability of the affected muscles is exalted, although afterwards depressed or lost ('Brit. Med. Journ.,' 1887, vol. i, p. 1302).

dress between her thumb and her forefinger; and a third could not toss over a letter into a letter-box. So, Erb states that the sufferers from writers' cramp may be unable to button their clothes, to work embroidery, or to play upon the piano. One of Dr Poore's patients had great difficulty in feeding and dressing himself.

Benedikt has proposed to distinguish a spasmodic, a tremulous, and a paralytic form of the disease.

Diagnosis.—The diagnosis of writers' cramp, or of the allied forms of handicraft and reflex spasmodic spasm, is tolerably easy when they have reached an advanced stage. But one must always recollect that impairment of the power of writing may result from a number of other affections of the nerves and muscles of the upper limbs. Progressive muscular atrophy, neuritis of the ulnar, the median, or the radial nerve, compression of either of these nerves by a tumour of whatever kind, may each give rise to this symptom. Dr Poore mentions a case of subclavio-axillary aneurysm in which the first thing noticed by the patient was that he could write only with great effort, and then illegibly.

As a rule, a person affected with writers' cramp goes on for a long time without medical advice, struggling against what he deems a foolish incapacity to carry on his duties. But when it does happen that one is consulted at a very early period, there may be great difficulty in determining whether the disease is really present or not. This is particularly the case if the patient is a nervous professional man, whose mind has for some time been dwelling upon the subject; or if he is a bank clerk, who has heard all about it from his fellow-officers, and who has perhaps been reading a number of medical books. Such persons come to one complaining that the act of writing causes a number of unpleasant sensations; and one may be in great doubt whether to laugh at their complaints or to take them seriously. It seems to me that the best indications are afforded by the effects of rest. If the supposed symptoms show themselves only at the end of a hard day's work, and are entirely gone the next morning or after the interval of a Sunday, they are not likely to be of serious consequence. Thus Dr Poore speaks of an eminent pianist who, after practising for a long time, found that he could not always strike the right note; but a few hours later he could play as well as ever. In such a case it is evident that nothing is seriously wrong.

Prognosis and treatment.—In most cases a right diagnosis is of the utmost importance, for on it may depend the whole future of the patient. At this period the discontinuance of all writing—or of whatever other action may be the exciting cause of the complaint—may completely cure it in the course of a month or two. Even then, indeed, there is always a risk that the disease may return if the muscles should be again overworked. But, most commonly, the prognosis that must be given is very unfavourable. At an advanced stage of the disease there is scarcely any chance of recovery, unless the patient can altogether give up his occupation for six months or even for a whole year. And in many cases treatment fails entirely, the disease advancing steadily in spite of all that can be done.

The only remedy appears to be galvanism. Of this Erb speaks highly, recommending the employment of ascending currents along the cervical spine, as well as peripheral galvanisation of the nerves and muscles specially concerned. But by far the best results have been obtained by Dr Poore. His method consists in making the patient perform rhythmical movements

with the affected muscles, while a continuous current of moderate intensity is passed through them. His first case was that of a man, aged thirty-two, who had suffered from writers' cramp for more than nine years, and in whom it presented its severest form. Dr Poore exercised separately the deltoid, the biceps, the triceps, and the other muscles, as well as those of the hands and fingers. When the treatment was commenced the patient could not finish writing the word George; at the end of five months he was able to write a whole epistle, forming his letters well and firmly. He had been unable to feed himself; he could now do all that he wanted, and had embarked in a small business. Another of Dr Poore's cases is little less remarkable. A man, aged forty, had for four years suffered from the disease, and had for six months been unable to write, the attempt to do so giving him great pain. After the first application of the current this pain disappeared, within a week he could sign his name with tolerable ease, and at the end of ten months his handwriting looked as good as it had ever been, although he was not at that time able to hold his pen quite properly and tightly between the thumb and fingers. Unfortunately, however, Dr Poore records no similar successes in his later papers on the subject.

Wolff's combination of galvanic treatment with massage is commended by Charcot, by Ross, and by de Watteville.

Dr Gowers strongly advocates free writing from the shoulder as a preventive and a remedy, and says that the disease is unknown among shorthand writers.

Bromide of potassium is said to be occasionally beneficial to patients affected with writers' cramp, by removing the depression and irritability from which they suffer. Strychnia is often injurious. Stimulating liniments and douches appear to be almost if not quite useless. Erb says that many patients are relieved by applying a narrow bandage or strip of plaster round the wrists. This cannot, however, be expected to do any good where the only muscles affected are the small muscles of the thumb and fingers.

*Hammer palsy.**—This curious variety of handicraft spasm was described by the late Dr Frank Smith, of Sheffield ('Lancet,' March 27th, 1869; and 'Brit. Med. Journ.,' Oct. 31st, 1874). He observed eight cases among the pen, blade, and file forgers, who use a hammer of three to eight pounds weight sometimes for twelve or thirteen hours, delivering as many as 28,800 carefully adjusted strokes every day.

The patient finds his hand has lost its cunning; he is awkward, and hits awry. Spasms of the flexors and pronators of the forearm appear, then gradual weakness of the whole limb, and finally muscular atrophy.

We lately had a typical case of hammer-spasm in Guy's Hospital (Philip Ward, No. 31, 1887) in a healthy, well-nourished man of twenty-seven. He worked at nail-making with a hammer of two pounds weight, and worked by the piece. His arm, on his attempting to move it, or on the hand being grasped, was at once thrown into clonic spasms, without pain, which affected the pronators and flexors of the forearm, and also the flexors and adductors of the arm. Sensation was perfect. There were no signs of affection of the cranial or other nerves, no wasting of muscles, and no cephalic symptoms.†

In Dr Smith's cases the right leg was frequently weak; there was

* *Synonyms.*—Hammer spasm—Hephæstic hemiplegia.

† This case, we afterwards found, was the same which formed the subject of a lecture by Dr Poore in the 'Lancet' for August 21st, 1886.

neuralgia or numbness, and painful spasm in the affected arm; sometimes ptosis or thickness of speech, while in one instance (No. 7) there was aphasia. Hence he proposed the name Hephæstic *hemiplegia.*

Of one case he writes: Neuralgia and an indescribable centripetal feeling frequently affected the right arm and forearm. The tactile sensibility of the forearm was almost lost; electro-sensibility and electro-contractility were almost extinct. Thermal sensibility remained. The temperature in the right axilla was 1° Fahrenheit below that of the left. There were no mental symptoms; sight, smell, hearing, taste, and speech were unaffected.

The facts of spasms being followed by paralysis, of extension of motor symptoms to the leg and face, and of the affection of speech, all appear to point to the motor region of the hemispheres as the seat of this remarkable disease. No autopsy has yet been made, and it is quite possible that the local lesion is, at least in its earlier stages, either vascular or otherwise unrecognisable after death. A "discharging," becoming afterwards a "destroying" lesion, would probably best explain writers' cramp and the allied spasmodic diseases. At least there seems less reason for placing the seat of the disease, whether "functional" or "organic," in the muscles, peripheral nerves, anterior cornua, or motor tract of the cord.

Some of Dr Smith's cases improved under sedatives and rest. In the case above mentioned in Guy's Hospital, Dr Poore's plan for scriveners' cramp was tried without benefit; also Calabar bean, bromides, and succus conii raised to a two-ounce dose, and continued three times a day for nearly a fortnight. Rest in bed seemed to be most useful, but the patient thought chloral hydrate at night not only gave him refreshing sleep, but made the spasms less severe during the day.

TETANILLA.*—In the spasmodic affections hitherto described the convulsive movements have been of a clonic kind. But there is one member of the group in which tonic spasm is the essential symptom. This is the disease to which Lucien Corvisart in 1852 gave the name of *tetanie:* previously it had been described by Dance in 1831 as "a kind of intermittent tetanus;" and Steinberg had called it "idiopathic" or "rheumatic contraction of the extremities." Trousseau's lecture on this curious affection first drew attention to it in this country, and Dr Moxon recorded a case in the 'Guy's Hospital Reports' for 1870. It differs pathologically and clinically from the terrible disease always known as tetanus.

Tetanilla is generally confined to the limbs, and affects chiefly their distal parts. Sometimes it is limited to the forearms and hands; much more rarely to the legs and feet. As a rule it involves all four extremities at the same time or alternately.

The patient first has a sensation of tingling in the parts which are to be affected, and then begins to find that their movements are no longer free. Soon the thumbs become forcibly adducted; the fingers of each hand are closely pressed together, and are half flexed at the metacarpo-phalangeal joints, but usually extended beyond; the palms are hollowed by the approximation of their inner and outer surfaces. Hence Trousseau's comparison to the conical form of the accoucheur's hand when introduced into the vagina to perform turning. The thumbs may be driven into the skin so

* *Synonyms.*—Tetany—Tonic cramps of the fingers and toes in children—Rheumatic contraction of the extremities—Remittent tetanus.—*Fr.* Tetanie—Tetanille—Contracture rhumatismale des nourrices (Trousseau).—*Germ.* Tetanie.

violently as to produce marks, or even (it is said) to give rise to sloughs. The wrists are half flexed, and the forearms strongly pronated. In the lower limbs the toes are bent down and adducted; the soles of the feet are hollowed, and the backs arched; the heels are pulled up by spasm of the muscles of the calves. The extremities are affected on both sides.

The contracted muscles feel hard; their resistance may be overcome by the employment of some force, which generally gives the patient pain, but sometimes relief. Thus the fingers and thumb may be straightened; but as soon as they are released they again become flexed as before. Another way of arresting the spasms is by means of cold. Trousseau mentions that persons in whom the lower limbs are affected can often regain the free use of them, for the time, by merely standing with naked feet on a stone floor; and the same result can be brought about by immersing the hands and forearms in cold water.

During the paroxysms the movements of the affected parts are much impaired. If, as is sometimes the case, the patient is a woman nursing a child, she cannot hold it in her arms. There is partial anæsthesia, so that the patient cannot judge of the size and hardness of objects. In walking he feels as if his feet were treading upon a thick carpet. Pain is sometimes altogether wanting, but it is usually present in more or less severity, and is compared to that which accompanies ordinary "cramps" of the legs. Another common symptom is that the backs of the hands and feet become œdematous, and sometimes red with swollen veins.

In from five to fifteen minutes the spasm generally passes off, but sometimes it lasts without stopping for an hour or two, or even longer still. As it subsides sensations of formication are again experienced.

After a variable interval another attack commences, and this goes on for several days, or sometimes for two or three months. Even after a long period of health the disease may return. A child, whose case is recorded by Dr Moxon, was first attacked by tetany when five months old, and had it on five distinct occasions, at intervals of from five to twelve months, before it reached the age of three years and a half. One of Trousseau's patients, a young man of twenty-one, had the disease every winter for four years, the contractions coming on every day during a period of two months.

Trousseau found that in a patient affected with tetany one can at any time bring on the spasms by compressing the principal nerve or artery in the upper part of the limb.

The electrical relations of the motor nerves and muscles in this disease have been investigated by Erb ('Ziemssen's Handbuch,' xii, p. 345), previously by Kussmaul and Benedikt, and more recently by Chvostek and Weiss, with the result that the excitability both to faradic and to galvanic currents has been found greatly increased except only where the facial nerve is distributed (see Dr Buzzard's observations, 'Clin. Lectures,' pp. 414, 417). We found in the two cases mentioned below that the closing contraction was apt to cause tetanus instead of an ordinary contraction.*

* The following summary of cases of tetany admitted into Guy's Hospital between 1866 and 1888 was extracted out of our clinical records by Mr Lefevre for the present edition:

1869. *Tetany.*—Boy, æt. 6: swelling of right foot, hot and tender; history of cramps *before* admission only. Relieved.

1870. *Intermittent tetany.*—M., æt. 3, with condition of talipes equino-varus. Relieved.

1872. *Intermittent tetanilla.*—M., æt. 18, with cramps in calves and hands; the diaphragm was also involved. Improved.

1873. *Tetany.*—M., æt. 2: hands cone-shape, feet extended; slight opisthotonos. Cured.

Cases presenting the characters just described are decidedly of rare occurrence; only seven have been seen at Guy's Hospital since the one recorded by Dr Moxon in 1870. One, in a boy three years old, was described by Dr Wilks in a paper in the same Reports for 1872, and in his lectures he recounts two others in youths of sixteen and eighteen. The former was a private patient; the latter was the third of the Guy's cases.

The writer had two boys, brothers, under his care within a few months, with the typical symptoms of tetanilla. One aged five, in Mary Ward, developed severe symptoms. The spasms became general, he lost his power of speech, and his temperature rose. After further local and general treatment he died, September, 1887, the illness having lasted about three weeks.

His elder brother, aged seven, was admitted into John Ward, November, 1888, with the same painful tonic spasms of his hands and feet. He slowly and gradually recovered, and at last went out well in February, 1889. In neither of these cases were there any clear signs of rickets.

In the present year (1890) we have had a ninth case in Mary Ward in an adult which resembled tetanilla in its localisation, and in being in all probability functional. A woman of about forty was suddenly attacked, as the result of strong and sudden emotion, by tonic contractions of her fingers and toes. This had lasted several months when she came under the writer's care, and two fingers of one hand had become contracted. The other hand was nearly well, and the toes could be flexed though with some pain. The case might be called carpo-pedal spasms in an adult. It had the character of an "hysterical" disorder.

In tetany the spasms are not always limited to the extremities. Trousseau describes instances in which the face and trunk were also affected. In these cases the face became distorted; the eyes acquired a squint; the sterno-mastoidei and the pectorales were rigid; the recti abdominis stood up like tense cords. The jaws were firmly clenched, and the speech was impaired. Laryngeal spasm sometimes occurred, causing lividity and apparent danger of suffocation. Febrile disturbance was also present. Yet even in such severe cases, the patient would often get up; and if an adult she would attend on other patients, although suffering from pains in the loins, and feeling bruised and exhausted. Sooner or later recovery almost invariably took place. Trousseau, however, mentions one instance in which death occurred from phthisis during a relapse of tetany, and another which was supposed to have ended fatally within a few hours of its commencement. Dr Moxon suggested that this was really a case of tetanus, but the hands and feet were characteristically affected; and these are the very parts which constantly escape the spasms of tetanus.

It appears likely that a very common affection is really a minor variety of tetany, namely, "*carpo-pedal contractions*," or "turning in" of the thumbs and great toes, which one looks for almost as a matter of course in rachitic children. Dr Jackson regards these as rudimentary epileptiform convulsions; but surely they are more closely related to tetany.

Notwithstanding occasional extension to other parts, tonic spasm of the fingers and toes is the distinctive feature of tetany, and this approximates

1876. *Tetany* (associated with rickets and diarrhœa).—Boy, æt. 6. Cured.
1883. ? *Tetany*.—Girl, æt. 10½. Right hand only affected; thumb flexed and adducted; fingers semiflexed; hand drawn to ulnar side. Rapidly disappeared.
1887. Boy, æt. 5. Hands and feet affected. Fatal by dyspnœa.
1888. Brother of above, æt. 7. Hands and feet, with much pain. Slowly recovered.

it to another affection of early life, *Trismus neonatorum*. It occurs in infants within a week after birth, and sometimes even in the first twelve hours. From Dr West's description of its symptoms it appears that, besides opisthotonos, there is clenching of the hands, flexion of the feet upon the ankles, and bending of the toes; and he goes on to say that "when the fit subsides the child still lies with its hands clenched and its thumbs drawn into the palm, the legs being generally crossed, and the great toe separated widely from the others." The head is thrown back, and the opisthotonos continues, although in a diminished degree. Before long the little patient becomes unable to swallow, and perhaps comatose; and death quickly ensues.

The frequently fatal result and the presence of opisthotonos seem to separate trismus neonatorum from tetany. But tonic spasm of the back of the neck is common in children with "carpo-pedal contractions."

The relation of these two affections to tetany is confirmed by their ætiology. The trismus of newly born children was at one time supposed to be a traumatic tetanus, excited by irritation which started from the umbilical cord. But that notion was refuted by the subsidence of the disease in the Dublin Lying-in Hospital, in consequence of the introduction of an effective system of ventilation. Previously, one in every six of the infants born there had died when less than a fortnight old; and nineteen deaths out of twenty were due to trismus. Afterwards the mortality was only one in fifty-eight and a half, and but a ninth part of it was from the disease in question. In the West Indian islands it is still common, and also in St Kilda; probably it is due to a vitiated state of the air in the lying-in chamber. In London it must be very rare, for Dr West saw only one case.

In a valuable paper in the 'Lancet' (May, 1887) Dr Cheadle relates a case of tetany in an adult, and remarks on the association of this disease with previous diarrhœa or lactation in women. He believes that rickets in infants is apt to produce various spasmodic affections, which sometimes take the form of infantile convulsions, sometimes of carpo-pedal contractions, sometimes of laryngismus stridulus, and sometimes, though less commonly, of tetany. Dr Eustace Smith and Dr Goodhart take the same view; and the latter writer quotes Mr Hutchinson's observation of the frequent coincidence of zonular cataract with infantile fits and rickets. Rickets has been present in most cases of tetany seen at Guy's Hospital.

Age and sex.—The majority of cases of tetanilla seen in England occur in children. In 142 collected from various sources Dr Gowers found 42 between one and nine years of age (and of these 34 under five), and 36 between ten and nineteen; making 78 under twenty, to 64 adults. Of the latter the majority were between twenty and forty (34 women to 13 men), 13 between forty and fifty (8 women to 5 men), and 4 (all men) above fifty. In children it is more common in boys than in girls. It occasionally occurs in families.

Trousseau speaks of tetany as occurring most frequently in women between the ages of seventeen and thirty, particularly in those who are suckling their children. One is tempted to think that there may be something in the diet adopted during the puerperal period by French physicians which lowers the health, and so brings on a disposition to the disease. Trousseau, however, speaks of it as being the direct result of exposure to cold. Tetany has been observed as a sequence after removal of the thyroid,—in seven out of seventy cases according to Wölfler. Trousseau also described diarrhœa as a frequent predisposing cause of tetany; he had also seen it

follow enteric fever and cholera. He had met with instances of it in women over forty years of age, and even in adult males.

Tetany sometimes occurs as an epidemic neurosis in girls' schools and in convents, as in the school at Gentilly in 1876. Such cases bring it into relation with hysteria, chorea major, and the extraordinary attacks of "convulsionnaires."

The immediate *pathology* of tetany is quite unknown; and it has no ascertained morbid anatomy. In the fatal case of the writer's referred to above, the brain and spinal cord were both normal to the naked eye, and after hardening and staining nothing abnormal could be found.

The malady has no connection whatever with rheumatism in any intelligible sense of the word; all that French and German writers mean by calling the contractures "rheumatic" is that they occasionally follow exposure to cold. Dr Hughlings Jackson suggests the hypothesis that cerebral influence is diminished, and that (supposed) cerebellar influence over the motor nerves is increased.

Dr Moxon remarked that the disease which has been called *spasmodic ergotism*, and which was prevalent in certain parts of Germany in the earlier part of the present century, was similar in its symptoms to tetany. Mr Wright's account of it ('Edin. Med. Journ.,' 1839) almost proves that the two disorders are identical, and that the former was not due to a specific poison, but to general deterioration of the health from a defective supply of nutriment. He states that in the eighteenth century a similar disease occurred in Sweden. (See also on this point a paper by Bauer in the 'Berliner klin. Wochenschr.,' 1872.)

The *diagnosis* of tetany is not difficult, spinal meningitis, especially epidemic cerebro-spinal fever (p. 653), being the only affection with which it is likely to be confounded. There is no rise of temperature in tetany, and when it occurs epidemically it is obviously a neurosis.

The *prognosis* is probably always favourable in genuine cases, but death may occur from general convulsions or other complications to which rachitic children are liable.

The *treatment* which Dr Moxon adopted in his case of tetany consisted in the administration of bromide of potassium in five-grain doses; in four days the spasms ceased to return, and the child afterwards took cod-liver oil and steel wine. There can be no question that such remedies are to be preferred to bleeding and cupping as suggested by Trousseau. Indeed, that writer himself makes an exception for weakly and debilitated subjects, for whom he recommends quinine with small doses of opium or belladonna; but cod-liver oil and good food are the best medicines. In severe cases he says that the inhalation of chloroform has sometimes given marked relief. He mentions a case which Aran cured by applying to the affected parts pieces of linen soaked in chloroform. Dr Wilks also finds cold applications the best. Internally chloral has been found most useful at Guy's Hospital.

Fresh air and exercise and moral discipline are probably the best treatment in many cases. In infants *chloral hydrate* (with or without bromides), followed up by good milk, cod-liver oil, meat juice, and other nutritious food, will in most cases cure by removing the cause as well as the symptom, in cases of tetany, or carpo-pedal contractions, or trismus. Dr Cheadle believes that Calabar bean will sometimes succeed in overcoming the spasms when other measures fail.

THOMSEN'S DISEASE.*—The first full and particular account of this remarkable affection was given in 1876 ('Arch. für Psychiatrie u. Nervenkr.') by Dr Thomsen, a Danish physician, who was himself, with several other members of his family, the subject of the infirmity. In the same year Seeligmüller published a characteristic case. Several others have been observed in Germany, France, Denmark, and Italy, and Dr Buzzard has recently described two cases in our own country ('Lancet,' May 14th, 1887). Erb published a monograph on the disease in 1886. A full bibliography was given by Dr Paul Chapman in the sixth volume of 'Brain' (April, 1883), and by Dr Hale White in the same periodical for April, 1886, and in the 'Guy's Hospital Reports' for 1890.

Symptoms.—The peculiar features of the affection are that when the patient endeavours to perform voluntary movement after rest, the muscles of the limbs when used are thrown into tonic contractions, which prevent motion and keep the parts in a state of stiffness, like that of cramp, though without pain. As in ordinary cramp, the contractions, though violent, are ineffective and irregular, portions only of the muscle being raised into bulging elevations. After a time the difficulty is overcome, and the patient is able to rise from his chair, and walk, run, or perform any action he desires with his limbs. But the difficulty returns again and again, so as seriously to interfere with his daily life. Seeligmüller's patient was a healthy young recruit, who found himself slow and awkward in performing his military exercises. Not only the arms and legs, but the muscles of the trunk, are subject to this difficulty, and also those of the tongue and lips, so that there results a kind of stammering—a violent effort to speak, inability to do so, and at last an explosive utterance, which passes into ordinary easy articulation for the rest of the sentence.

The involuntary muscles, as those of respiration and micturition and the sphincters, are quite unaffected. There is sometimes a feeling of numbness or "pins and needles" in the limbs, and occasionally pains, but these appear to be often absent, and never severe or long continued. There is no anæsthesia. The knee-jerk and plantar reflexes are usually reported to be present, and sometimes excessive; but in one of Dr Buzzard's cases the former was completely and the latter almost completely abolished. The galvanic and faradic irritability of the muscles is greater than normal, and they readily respond to mechanical stimuli, but contract and relax very slowly. The nerves react normally; there is no R.D. Fibrillary tremors have often been observed, and the contraction of a muscle after stimulation continues long enough to be photographed. Erb says that irritation of the ulnar nerve causes the muscles it supplies to contract, but Dr White finds that this may occur in healthy persons.

There is no atrophy of the affected muscles. On the contrary, they undergo true hypertrophy, especially, it would seem, the gastrocnemii, external vasti, glutæi, and trapezii, but always in a symmetrical manner. Yet the muscular strength of the patients is not at all proportionate to this apparently athletic development.

Some authors have described the mental faculties of the patient as deficient, but there seems no reason to regard this as a character of the disease, particularly since one of them taught us to recognise it. The patient is often intelligent and anxious to be relieved of his infirmity; but he says

* Called by Thomsen "Tonic contractions in voluntary muscles, as the result of inherited psychical predisposition." Strümpell and Erb call it "Myotonia congenita."

he feels awkward and unready; "before doing anything he must get his muscles ready," and hence he is incapable of prompt and rapid movement in reponse to the will. The general health is unimpaired.

We have lately had a typical case of this curious disorder in Guy's Hospital, in a healthy and well-made young Welshman. He was apparently strong, and of more than average intelligence, but he could not "start" walking or rising from a chair. A full account of his case is given by Dr W. H. White, 'Guy's Hosp. Rep.,' vol. xlvi.

Pathology.—No autopsy has yet been made in a case of Thomsen's disease, so that its seat and morbid anatomy are completely unknown. The primary change may be in the muscles or motor nerves, in the motor tracts of the cord, or in the cerebral cortex, or it is quite possible that there is no lesion discoverable by the scalpel or the microscope. Fragments of muscles have been removed by the "harpoon" (p. 498), and the fibres are found to be decidedly thicker than usual; the striation is unaffected, and there is no fatty or interstitial overgrowth as in pseudo-hypertrophic paralysis. (See the drawing in Dr White's article, pl. iii.)

Dr A. McL. Hamilton, of New York, and some other writers, have argued against the symptoms of Thomsen's disease being more than accidental or secondary. Dr Thomsen himself, as appears by the title under which he described it, regarded the affection as rather mental or emotional than organic in origin. The nervous centres have never been examined, for no patient has yet died.

Ætiology.—The reality of the disease as a recurrent combination of symptoms is established by its hereditary origin. Dr Thomsen saw signs of it in his own children while still in the cradle; his own mother was slightly affected, and two of her brothers typically so; of her thirteen children, six, besides Dr Thomsen himself, were subjects of the infirmity; and of his own five sons, four inherited it from their father. Benedikt, Leyden, Eulenburg, Pitres, Dalledet, Erb, all found the same hereditary and family distribution. It is more constant than in any other affection of the nervous system, except perhaps Friedreich's disease; but, like that rare and obscure condition, its duration is more extended laterally than vertically, *i. e.* it affects many brothers and sisters in a family, but does not so readily pass to the next generation, and rarely to a third.

Both sexes are liable to be affected, but males much more frequently. The disease is probably congenital, but the symptoms first become obvious when a boy goes to school and finds himself unable to take part in games, —in a case reported by Dr Banham, of Reading, only at the age of twelve.

Course and prognosis.—The symptoms do not appear to be aggravated after they are once fully developed, but there is no instance of their having disappeared, nor is any treatment even supposed to be efficacious. The condition is probably as innate and ineradicable as stammering, or timbre of voice, or tricks of speech and gesture; and, like them, if ever "grown out of" the result is not obtained by direct therapeutical means, but by the gradual effect of circumstances and of time.

PARALYSIS AGITANS.*—Unlike the preceding spasmodic disorders, the curious malady called shaking palsy has been known from an early period,

* *Synonyms.*—Shaking palsy—Paralysis cum tremore—'Parkinson's disease'—Scelotyrbe festinans—Chorea procursiva, senilis v. festinans—*Anglice,* The trembles.—*Fr.* Tremblement paralitique progressif.—*Germ.* Schüttellähmung.

though its true clinical features were only recognised by Parkinson in 1817. It consists in more or less violent oscillatory movements affecting especially the limbs, but sometimes the neck and the tongue also. It usually begins in one of the arms, and sometimes in a leg. Charcot says that it may for a time be limited to one thumb. The movement is at first very slight, a mere tremor; but gradually the amplitude of the oscillations becomes greater and the whole limb is involved, so that the patient is unable to go on with his work, and has to seek medical advice. Sometimes, however, the agitation of the muscles is severe from the very first. It is said to be sometimes the result of a sudden shock or terror, but in two cases under the author's care the patient went to bed as usual and woke in the morning with the disease fully developed, the right arm being in one instance the part affected, in the other both the left arm and the left leg. In two other recent cases at Guy's Hospital one side of the neck was the earliest seat of the movements. After a variable interval—generally after some months—the oscillations appear somewhere else, most frequently in the leg of the same side as the affected arm, but sometimes in the other arm. Presently the remaining limbs suffer in their turn. The head also begins to shake, and the tongue becomes very tremulous when it is protruded, or even when it is lying within the mouth; but nystagmus, or oscillation of the eyeballs, seems never to occur, nor do the jaws ever take part in the movements.

During the early part of its course paralysis agitans is almost always paroxysmal. Each attack generally lasts some hours, and it is followed by a feeling of great fatigue, which gradually passes off in the interval of rest before the next one begins. At a later period the movements become continuous.

The oscillations bear to voluntary motions of the affected parts a relation on which great stress has been laid by successive writers, but which is hardly so simple as has been represented. Mr Parkinson began his monograph by defining it as follows:—"Involuntary tremulous motion, with lessened muscular power *in parts not in action*, and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellect being uninjured." He goes on to distinguish the disease from the tremor of old age, as well as from that caused by the vapour of mercury (cf. p. 521).

During the early period of the disease the hand may become perfectly steady when it takes up a tool or a pen, and the patient is able to control the agitated limb by an effort of will; moreover, the oscillations often go on without intermission when the elbow rests upon the knee. But at this stage the affection is usually dormant so long as the patient lies quietly on his back in bed, and it is certain that no movements occur during sleep except in the most advanced stage of the disease. Parkinson mentions that one of his patients had an intercurrent attack of right hemiplegia, in which the face was drawn to the left side, and this lasted a fortnight; during that time neither the arm nor the leg on the paralysed side was in the least affected with the tremulous agitation, but it returned as the limbs regained their power. In the later stage of shaking palsy the attempt to perform any voluntary movement brings on the oscillations with greatly increased violence. They are also much augmented by emotion or excitement. Even the presence of a looker on often affects such patients to a remarkable degree; a man who can write quite well when alone may be unable to form a letter while he is being watched by a clinical clerk. If

we hold the agitated limb, so as to check its movements, they are aggravated; and if we forcibly restrain them, similar movements appear in the opposite arm or leg.

Paresis.—According to most writers there is in paralysis agitans, beside the oscillatory movements, a certain degree of impairment in the muscular power of the affected parts. Eulenburg, however, has found that even in severe cases of long standing the reaction to induced or galvanic currents is perfect, and both Trousseau and Charcot have taught that the muscles really retain their full force. The former writer relates a case in which the patient was able to exert much more power with the affected hand (which, however, was the right one) than with the other, which was healthy—considerably more power than Trousseau himself could exert. But this must not be taken as showing that the disease had actually augmented the force of the limb, since it was not known what the man's strength had previously been. Another point observed in that case was that the contractility of the muscles was very quickly exhausted; when told to open and shut his hand in quick succession the patient at first did so rapidly, but after fifteen seconds more slowly, and soon not at all. Charcot further remarks that there is often a retardation of the influence of the will, shown by an unduly long interval between a thought and its expression in words.

Recently M. Bourneville, the editor of 'Charcot's Lectures,' has made observations on six patients with the dynamometer, which appear to show that their strength was really diminished to a considerable extent.

In most cases limbs affected with paralysis agitans have their cutaneous sensibility unimpaired. Charcot, however, says that a feeling of pins and needles in the hands and feet is sometimes complained of; this was the case in a patient of the author's, who also said that he was unable to feel the ground. Headache and vertigo are not uncommonly present; and at an advanced period of the disease there may be loss of memory, and even delirium.

Tonic spasms.—One impediment to voluntary movements in the more advanced stages of paralysis agitans is the occurrence of *rigidity* in the muscles of the affected parts. Charcot has pointed out that the patient commonly maintains an attitude which is characteristic of the disease. The head is bent so that the chin approaches the sternum, and he can with difficulty raise or turn it to left or right. The body is almost always bowed forwards when he is standing. The elbows are generally drawn slightly away from the chest, and partially flexed; the hands present a deformity somewhat like that which occurs in osteo-arthritis, the three inner fingers being inclined towards the ulnar side of the hand, while the thumb and forefinger are stretched out and brought close together as in holding a pen. In the lower limbs the rigidity is sometimes very marked; they are semi-flexed, the knees are brought together by a movement of adduction, the feet are curved inwards as in talipes equino-varus, and the toes are arched like the three fingers.

Articulation is unimpaired until the last stage, but the speech is slow and jerking, as though a considerable effort of the will were needed for the pronunciation of each word. Charcot compares it to a bad rider trying to talk when mounted on a high-trotting horse.

The way in which the head and body are bent forwards in the more advanced stages of paralysis agitans accounts for a symptom which Parkinson noticed, namely, that the patient tends to fall upon his face when he attempts to walk, and that his steps are consequently hurried, so that he

runs instead of keeping to his ordinary pace. This had, indeed, been previously described by Sauvages, but as a separate complaint, under the name of *Scelotyrbe festinans.** The man has been said to be constantly trying to overtake his own centre of gravity. Parkinson mentions a case in which an attendant was obliged to walk backwards in front of the patient with one hand on each of his shoulders to prevent him from falling. When he first gets up from his seat, which he does very slowly, the patient makes a few hesitating and ineffectual steps before he seems to be able to start off, and in walking he treads only upon his toes, being unable to bring his heels to the ground. But the most remarkable symptom is that some persons affected with paralysis agitans walk backwards without intending it and when they mean to go forwards. Some years ago a striking instance of this occurred at Guy's Hospital. The patient, after a few ineffectual efforts to rise from his chair, would stand up, pause, make two or three abortive attempts at starting, and then succeed in walking a few steps towards the door, when suddenly he found himself hurried against his will backwards into the umbrella-stand in the corner of the room. It was like the sudden reversal of the engines of a steamboat. Charcot met with a case in which he could at any time induce such retrograde movements by unexpectedly giving a gentle pull at the patient's dress when she was standing up, and Dr Buzzard remarked exactly the same thing in an elderly woman, whose appearance and symptoms he graphically describes in his lectures. The movements cease during sleep, except in the latest stages.

In the more marked cases of paralysis agitans the movements of the hands are not merely oscillatory, but are to some extent co-ordinated. The finger and thumb move as if the patient were rolling a cigarette paper, or crumbling a morsel of bread.

Profuse sweating is an almost constant symptom when the oscillatory movements are at all severe. The patient may become so bathed in perspiration as to have to change his clothes many times a day. He often suffers greatly from a feeling of heat, especially about the epigastrium and in the back, so that he insists on being kept lightly covered. But with the thermometer the temperature is always found to be normal.

The features are immobile, and the face has a remarkably impassive expression. The voice is often shrill and piping. Dr Buzzard believes that this change of an old man's voice to "childish treble" is an almost decisive symptom of shaking palsy.

Pathology.—It is still uncertain whether paralysis agitans should be regarded as an affection of the brain or of the spinal cord. Parkinson supposed its seat to be in the cervical region of the cord, extending up to the bulb. But perhaps indications adverse to such a view may be found in the fact that after one upper limb the corresponding leg is generally next affected, rather than the opposite arm; and still more in the cessation of the oscillations during sleep, and throughout the continuance of a hemiplegic attack, so far as the paralysed limbs are concerned. Hitherto anatomical observations have thrown no light on the question. The author made one autopsy, on a woman, aged forty, who died of phthisis after having suffered from paralysis agitans for eight years; for a year her speech had been impaired, and at last it was unintelligible. The only unusual appearance detected was that the substance of the pons towards the floor

* Σκελοτύρβη (disorder of the legs, staggering) occurs in Strabo, Pliny, and Galen. The term has been also applied to chorea.

of the fourth ventricle looked unduly grey. Westphal and Wilks also failed to find any lesion. Slight thickening of the cortical layer and reticulum were found by Cayley in the cord of a patient of Murchison's, aged seventy-one, who died of typhus after being twelve years the subject of paralysis agitans ('Path. Trans.,' 1871, p. 24). Charcot has been able to examine the nervous centres in six cases: in three they were perfectly healthy; in three they presented slight microscopical changes which were believed to be merely senile. In some cases recorded by other observers there has been an obvious atrophy of the brain, but not more than is often found in persons of the same age. Eulenburg, in his paper in 'Ziemssen's Handbuch,' places paralysis agitans among "neuroses."

It is by no means a common complaint. During the ten years from 1866 to 1875 only fourteen cases were admitted into the wards of Guy's Hospital, from 1876 to 1885 eighteen, and from 1886 to 1889, eight.

Age.—Parkinson speaks of paralysis agitans as seldom occurring in persons below the age of fifty; but among the forty cases at Guy's Hospital there were twenty-one in whom it began at an earlier age, and in nine of them before forty. Of these nine, four were thirty-six, one thirty-two, three thirty-eight, and the youngest was but twenty-one years old. This last instance is not without precedent; for Charcot mentions a case of Duchenne's in which the patient was only twenty, and one of Fioupe's which occurred in a girl of fifteen or sixteen who had been terrified by a bombshell during the siege of Paris. In thirteen of our forty cases the disease began between the fiftieth year and the fifty-ninth, and there were six in which the patient was still older when first attacked, the age varying from sixty-one to seventy-three.

Sex.—Charcot says that paralysis agitans is equally common in women and in men; but in all Parkinson's cases, and in twenty-nine out of our forty cases, the patients were of the male sex.

Origin.—The disease seems frequently to arise without any definite exciting cause; but sometimes it follows close upon some violent shock of terror or other emotion. Charcot says that in many of his female patients it developed itself during the political commotions so frequent in France. The prolonged action of cold and moisture has been mentioned as occasionally giving rise to paralysis agitans, but apparently on no satisfactory grounds. It has sometimes followed a local injury, as in a case of Charcot's, that of a lady who severely bruised her left thigh in falling from a carriage, and in whom shortly afterwards that leg began to shake, and at a later period all the other limbs.

Diagnosis.—The recognition of paralysis agitans is seldom difficult. A disease which was until recently confounded with it is the insular or multiple sclerosis described in a previous chapter; but the points of distinction between them are clear (cf. p. 545).

In the tremors of age the head shakes when the patient is sitting at rest, and the hands when he moves them. But it is doubtful whether a strict line of separation can be drawn between paralysis agitans and the tremor which is so common in old people, and which (as Dr Maclachlan found among the inmates of Chelsea Hospital) has little or no tendency to shorten their lives. He mentions one case in a pensioner, aged 107, who had been affected with it ever since he was sixty. But it may be that the same affection, which in young subjects is progressive, is in older persons comparatively stationary, or runs so slow a course that death over-

takes them before it has had time to develop itself fully. Dr Handfield-Jones has, indeed, expressed an exactly opposite view, namely, that shaking palsy in young subjects is a less serious form of the disease and curable, whereas in older patients it is incurable; but there seems to be no adequate evidence in support of this opinion.

One must not forget that local organic disease of the brain—a tumour, for example—may give rise to paroxysmal attacks of spasm in one arm, or in one arm and leg, which are not unlike those of the commencement of paralysis agitans; but the history of the case and the other symptoms will generally prevent one from making a mistake in this direction.

Hysteria may simulate this disease. Thus, in a girl of eighteen, the right arm began to shake three weeks after a fright. When she was admitted into the hospital it was in a state of continuous agitation, and if it was held the other limbs began to move in a similar way. However, she had a screaming fit the very day after her complaint began, with globus and headache. She was treated with frictional electricity, and quickly recovered.

Prognosis.—Paralysis agitans is a disease which runs a very slow course. Charcot speaks of it as sometimes lasting thirty years. But even at an advanced period of the disease recovery sometimes takes place. Towards the end the patient sometimes falls into a very sad condition. The movements, which are now incessant, at least while he is awake, may be so violent as to shake the bed in which he lies. He is unable to get up or to dress or feed himself without assistance. His speech may become unintelligible. His mouth remains open, and the saliva runs from it. His fæces and urine are passed involuntarily. Bedsores may form, and he may die of sheer exhaustion, or he may be carried off at an earlier period by pneumonia or some other intercurrent disease. Charcot remarks that a few hours before death the movements sometimes cease entirely.

Treatment.—Dr Elliotson supposed that he cured one case with the subcarbonate of iron; Brown-Séquard that he cured another with chloride of barium. In three of our cases the last-named salt was given in doses of gr. ¼ to gr. j. In two it seemed useless; in the other marked improvement took place, the patient (who was under Dr Moxon's care) being able within six weeks to walk twice the length of the ward merely holding a nurse's hand, whereas he had been so helpless as to be unable to get in or out of bed, and for five years he was dressed by others. A man, after taking two grains of valerianate of zinc three times a day, went out of the hospital "cured," but the noise of a passing waggon, as he was walking home to Bermondsey, brought back the jerking movements, which had ceased entirely for several days. Dr Ramskill (Syd. Soc.'s translation of 'Trousseau,' vol. i, p. 499) had a well-marked case in which recovery occurred, after the failure of other treatment, under the administration of four minims of phosphorised oil of the Prussian pharmacopœia in a drachm of cod-liver oil three times a day.

A plan recommended by Eulenburg consists in the subcutaneous injection of liquor potassæ arsenitis diluted with two parts of water. The quantities which he employed would correspond with from 1½ to 2½ minims of Fowler's solution. But Charcot has made trial of this treatment, and found it useless. Dr Shaw, the medical registrar at Guy's Hospital, in a report made for the second edition of the present work, states that nitroglycerine and physostigma have been tried without effect.

Eulenburg says that he has employed galvanism without any benefit—the constant current passed through the head or along the sympathetic nerves. We have had at Guy's Hospital one or two cases (among more failures) in which the application of this form of electricity down the spine caused temporary improvement. The most striking was in a patient of Dr Habershon's. It was first found that galvanisation for ten minutes was attended with marked benefit for the time. The electrodes were therefore fixed upon the neck for three hours without intermission. After this treatment had been continued for some time the limbs became much steadier, but he was not cured. Dr Wilks used to say that no medicines do any good, but that galvanism sometimes relieves.

At an early stage, when one limb only is affected, it might be worth while to try the effect of keeping it at absolute rest for a period of several weeks, if necessary by the aid of a mechanical appliance.

TETANUS.*—We now come to a dangerous and happily rare neurosis accompanied with most severe tonic spasms. Its history dates back to classical times; and so completely has the word become identified with *tonic spasm*, that it is applied by physiologists to any continuous contraction of a muscle. There are, however, several distinct neuroses which are all attended with continuous spasms—Tonic Wryneck, Tetanilla, Trismus neonatorum, and Hysterical Contractions of the Limbs. Evidently, therefore, something more than the presence of tonic muscular spasms is required to characterise tetanus. Further definition is found in the *distribution* of the spasms, in their more or less constant *order of development*, in the traumatic *origin* of the disease, and above all in its rapid *course* and fatal *issue*.

Symptoms.—In the great majority of cases tetanus begins with stiffness or rigidity of certain muscles of the face or neck. Very commonly those first affected are the masseters and the other muscles of mastication; the consequent inability to open the mouth is known as *trismus*, and has given to tetanus the English name of "lockjaw." Sometimes the earliest symptom is a "stiff neck;" or it may be a peculiar grinning expression of the face, as in a little girl who some years ago died in Guy's Hospital: her mother found fault with her for making faces; but soon afterwards the poor child was nearly choked by spasms in attempting to swallow food. The disease is often first discovered when the patient wakes up in the morning. As a rule it is preceded by a wound, or by an injury of some sort, sometimes not sufficiently severe to require surgical treatment. Occasionally before the tetanus sets in darting pains are complained of in the injured part, which may shoot up the limb; there are four instances of this among seventy-two cases which were collected from our books for the 'Guy's Hospital Reports' in 1857 by the late Mr Poland. Some cases are said to have been ushered in by rigors, but, as a rule, there are no premonitory symptoms whatever.

Possibly the trismus and other early symptoms, after lasting a few days, may (it is said) subside, and the disease aborts; but far more frequently the tonic spasm increases and spreads to the muscles of the trunk and limbs. The face acquires an unnaturally aged appearance, the forehead is wrinkled, and the features drawn. The angles of the mouth are wide apart, and the lips are stretched over the closed teeth, so as to produce a fixed smile,

* Τίτανος (a stretching, strain, tension, from τείνω), used by Hippocrates and late classical writers—Rigor nervorum (Celsus)—Lockjaw.—*Fr.* Tétanos.—*Germ.* Starrkrampf.

which is known as the *risus sardonicus.** The naso-labial furrows are exaggerated. The eyelids are half closed; their muscles are seldom affected by the cramp. The jaws are sometimes clenched so firmly that not even a paper-knife can be wedged in between the teeth. The trunk is rigid, and it is almost always curved, so that the back forms a deep hollow. Thus the occiput is buried in the pillow, and the throat is stretched upwards. If the patient were to attempt to lie straight his body would be supported upon the head and the heels. This condition is called *opisthotonos.* The chest is thrown forwards, and is more or less fixed in a state of expiration, while the abdomen is flat or hollow. The tension of the affected muscles is obvious to the touch and sight; this is particularly the case with the recti abdominis, which feel "as hard as boards," and stand out in knotty masses. Sometimes their fibres give way, and blood is extravasated so as to form a palpable swelling. The limbs are commonly extended, and there may be a marked stiffness of the shoulders and hips, and less often of the elbows and knees. Beyond a little undue resistance to passive flexion of the wrists, there is seldom any impairment of the movements of the hands or fingers. In one case it was recorded that the soles of the feet were arched and the extensor tendons rigid.

From an early period of the disease there is *pain* resembling that which is experienced in a limb affected with cramp, and one of the first symptoms is often an acute pain at the lower part of the sternum piercing through to the back; this is supposed to be due to spasm of the diaphragm. There may also be a distressing sense of oppression from embarrassment of the breathing, and the same cause may reduce the voice to a whisper. The patient is frequently unable to micturate in consequence of the rigidity of his abdominal muscles.

According to Rose, of Zürich, many cases of tetanus end fatally without being attended by any symptoms beyond those which have been already described, but as a rule the spasms undergo aggravation from time to time at longer or shorter intervals; during these paroxysms, which last from a few seconds to three or four minutes, the aspect of the patient becomes frightful. The contraction of the features and the opisthotonos are greatly augmented. The tongue is often caught between the teeth and severely bitten. The face and even the hands become livid from interference with the respiration. These paroxysms sometimes seem to arise spontaneously; sometimes they are obviously reflex, being brought on by a touch, a draught of cold air, a sudden noise, or some voluntary effort, such as the attempt to turn round, to speak, or to swallow. They are generally attended with great increase in the pain, often by extreme anguish. Sir Gilbert Blane, however, met with a case in which, although it terminated fatally, there was merely a tingling sensation of rather a pleasurable kind.

Sleep is generally impossible from an early period, but Watson relates how, when a patient fell asleep, the spasms ceased for the time; even the abdominal muscles became perfectly soft and yielding, but instantly resumed their contracted state as soon as he awoke. The mind is clear and unclouded; only when the end is near at hand is there sometimes slight delirium.

* *I. e.* as usually interpreted, the Sardinian laugh: from the classical tradition of an herb growing in Sardinia (Σαρδώ) which produced involuntary facial spasms. Cicero has *ridere γέλωτα σαρδάνιον*; and this form, used by all Greek writers from Homer to Plato and afterwards, makes the above derivation doubtful.

The pulse is at first natural, but towards the last it becomes very rapid, perhaps 160 or 200 in the minute.

It was long a disputed question whether tetanus is attended with fever. Recent observations have shown that the temperature may be normal throughout the whole course of even the most acute and severe cases; while in those which are comparatively slow in their progress the thermometer occasionally indicates 102° or 103°. Before death hyperpyrexia sometimes rapidly develops itself, temperatures of 110° or 112° being registered. The suggestion has been made that the heat evolved is due to the tonic muscular contractions. But only a small part of the rise can be accounted for in this way, and its great height must be ascribed to disturbance of a central regulating machinery, exactly as when the same thing occurs in a case of fractured spine, of cerebral hæmorrhage, or of the *status epilepticus*. Tetanus is among the few diseases in which the temperature has been observed to rise one or two degrees after death.*

The skin is often bathed in sweat, and an eruption of sudamina is not infrequently present. Dr Wilks has recorded in the 'Guy's Hospital Reports' for 1872 an instance in which the perspiration from the forehead gave to white linen a reddish stain, which, however, Dr Stevenson found not to be due to the presence of blood.

Varieties.—The symptoms of tetanus sometimes deviate from the ordinary type. In ancient times several species were distinguished. Thus it was said that the body may be arched forwards instead of backwards, so that the head and the knees meet in front of the chest; and this was called *emprosthotonos.*† In another variety, for which the name *pleurothotonus* has been invented, the curve is on one side. But it has long been known that, in comparison with opisthotonos, both are exceedingly infrequent, and it is absurd to make of them separate kinds of tetanus. Indeed, Rose has lately maintained that they are really never seen in this disease, although they may occur in hysteria.‡

In 1870, however, a woman, aged forty, died in Guy's Hospital of tetanus, in whom it is said that "the anterior muscles were mainly affected, so that there was a condition of emprosthotonos."

There is one aberrant variety, in which the earliest symptom is muscular spasm of the part originally injured, and in which the paroxysms, when they set in, affect those muscles far more than others. Such a case was recorded by the late Mr Key in the third volume of the 'Guy's Hospital Reports,' and we have since had a well-marked instance of the same kind.

An occasional complication of tetanus, to which Rose has drawn attention, is facial paralysis. In 1871 a woman died under Mr Poland in Guy's

* This heat has then been supposed to be derived from the stiffening of the muscles in *rigor mortis*. This explanation, however, does not seem to be quite satisfactory, for although the *post-mortem* rigidity is said to begin early, and also to last longer than usual, there appears to be an interval of relaxation from the vital spasm before it sets in.

† "Neque tamen alius importunior acutiorque morbus est quam is, qui quodam rigore nervorum, modo caput scapulis, modo mentum pectori annectit, modo rectam et immobilem cervicem intendit. Priorem Græci *ὀπισθότονον*, insequentem *ἐμπροσθότονον*, ultimum *τέτανον* appellant: quamvis minus subtiliter quidam indiscretis his nominibus utuntur" ('Celsus,' lib. iv, cap. iii; also 'Cælius Aurelianus de Acut. Morb.,' lib. iii, cap. vi).

‡ The French military surgeon, Larrey, has been the great authority for the occurrence of emprosthotonos in tetanus; but Rose shows by detailed criticism that none of his cases belonged to any but a very mild form of tetanus. Moreover, this position of the body is, after all, frequent in patients suffering under irritation of the nervous centres. Larrey entertained the notion, certainly without foundation, that the distribution of the spasms varied according as the wound which caused the tetanus was in front or behind.

Hospital, in whom this symptom was present on the left side, and whose ocular muscles were also affected in a strange way, the left eye being immoveable, turned upwards and outwards, while the right one was as rigidly set straight forwards. Neither meningitis nor any lesion of the brain was discovered at the autopsy. The original accident was a fracture of the orbital plate of the frontal bone, caused by the point of an umbrella. This corresponds with a statement of Rose's, that the starting-point of the tetanus in such cases is always within the distribution of the facial nerve; he supposes that the trunk of the nerve becomes swollen, and is compressed within the bony canal through which it has to pass. If this view is correct, the symptom in question affords a proof of the occurrence of an "ascending neuritis" in tetanus, and is thus of great theoretical interest. Indeed, in some few cases nerve-trunks are said to have been found reddened and swollen in parts of their course; but as such changes are often not to be discovered, it is doubtful whether they are of much importance.

Ætiology.—In reference to the *causes* of the disease there are various questions, which are fully discussed in surgical works, as to how far the extent or the seat of an injury depends on the healthy or unhealthy state of the lacerated structures, or on injury to a nerve, or the introduction of foreign bodies.* But it is important for the physician to be alive to the fact that tetanus often follows very slight cuts and trifling abrasions, which are apt to be forgotten by the patient. Rose says that nearly half of his cases were sent into the hospital as cases of "rheumatism" or of some internal disease. Instances have been recorded in which tetanus has apparently been set up by the extraction of a tooth, by venesection, by the application of a blister or a cupping-glass or a seton, by the sting of a bee, and by a cut from a whip. It has now and then been observed after simple fractures of the limbs, or after blows or falls upon the back of the neck, without any breach of surface. In 1860 a girl, aged five, died in Guy's Hospital, who on the day before she was attacked by the disease had a fall in which she slightly strained her back and grazed her elbow. Three other patients showed, one a slight scar over the knee, another a small sore on the elbow, and the third a little scab with pus beneath it at the elbow, in addition to a cicatrix half an inch long on the forearm, due to a cut received about six weeks previously. In 1873 a boy was admitted under the author's care in whom stiffness of the neck and jaws had come on four or five days after the healing of a small "gathering" on one great toe, due to irritation from a nail in his shoe. This patient recovered. Again, tetanus has sometimes occurred soon after parturition or abortion. In a case which was observed at Guy's in 1870 no cause could be discovered but a prolapsed and excoriated cervix uteri.

There is a special ætiological variety of the disease—*tetanus neonatorum*—which affects infants, usually in the end of the first or during the second week after birth. It is believed to be the result of an ulcer of the navel.

In two of our cases, in which the disease appeared to be referable to a trifling injury, it is possible that *cold* helped in its production. One was a patient of the author's who, besides having a festering sore on the elbow, had got wet through in a shower of rain three days before the tetanus set in; this perhaps affected him the more because his work was behind the ovens at a biscuit manufactory. The other was a man under Mr Bryant, who had a slight scratch on the little finger, which soon healed;

* See M. Bowlby's remarks on this subject (*loc. cit.*, p. 304).

he also was exposed to wet two days before being attacked with stiffness of the shoulders.

It seems to be an established fact that in some cases in which tetanus is clearly traceable to a severe wound it is, nevertheless, also due, in part, to changes of temperature. Army surgeons have often noted that after a battle the wounded are especially apt to be attacked when they lie in tents on a damp surface, or when cold nights follow hot days.

The liability to exposure to draughts in tropical climates may be one reason of the frequency with which, as is well known, the disease follows all sorts of injuries among the coloured populations of the East and West Indies; and the same seems to have been true in ancient Greece and Italy, where tetanus was well known. It is among the diseases which Hippocrates ascribes to cold (Aphor., v, 17).*

Moreover, there appears to be no doubt that cold sometimes gives rise to tetanus directly, in persons who have received no injury whatever. On July 17th, 1863, a man sat in a draught, and afterwards felt a stiffness in his limbs; this continued during the 18th and the 19th; on the 20th more marked tetanic symptoms set in, and he came to Guy's Hospital and was admitted; at 7 a.m. on the 21st he had a severe paroxysm affecting his chest, in which he died. Another patient, in 1869, had got his feet wet the day before the disease began. Sir Thomas Watson cites a case from Dr Gregory of a man who, "having fallen asleep in moist grass, awoke with a stiff neck, which afterwards went on into regular tetanus."

But it now and then happens that not even this cause can with any plausibility be assigned, so that the occurrence of tetanus remains altogether inexplicable in the present state of our knowledge. Three such cases have ended fatally in our wards between 1863 and 1875.

Whenever the disease arises independently of an injury—whether or not after exposure to cold or wet—it is called "idiopathic," to distinguish it from ordinary "traumatic" tetanus.

On the whole, tetanus is more common in men than in women, in young persons than in those who have passed the middle period of life, and in the robust and the healthy than in those who are weakly or diseased.

Pathology.—Formerly pathologists were agreed that there are no obvious changes in the nervous centres in cases of tetanus. But in 1865 Mr Lockhart Clarke recorded in the 'Med.-Chir. Transactions' (p. 255, pl. iv) some cases in which he believed that he had discovered areas of semi-fluid disintegration, or white softening, in the grey matter, but also in the white columns of the cord. Similar observations were afterwards made by Benedickt and by Dr Dickinson, who considered that the leucocytes found in the peri-arterial sheaths of the same structures denoted an inflammatory exudation. Still more recently Dr Coats, of Glasgow, has demonstrated like appearances in the bulb and the pons. These pathologists also lay stress on the congested state of the blood-vessels, but that had long ago been shown to have no real significance, being attributable either to the mode of death by dyspnœa or to gravitation after death. Whether greater importance is to be attached to the supposed lesions above referred to is still doubtful, and the doubt applies to other diseases beside tetanus.

In his article on Tetanus in 'Holmes's System of Surgery,' the late Mr

* "Frigus modo nervorum distentionem, modo rigorem infert: illud σπασμός, hoc τέτανος Græce nominatur" ('Celsus,' ii, 1). The context shows that this statement is directly taken from Hippocrates.

Poland argued against a primary central lesion (or an ascending peripheral neuritis, as it would now be called—*a neuritis migrans*, as Lockhart Clarke supposed), and in favour of the view that tetanus is really a "specific" disease, a form of blood-poisoning. This hypothesis has lately been supported by Professor Coats, by Mr Bowlby, and by many pathologists abroad. Experiments to ascertain whether it is transferable by inoculation, and to isolate the contagion of bacteria if present, have been undertaken by Rosenbach, Rietsch, Abadie, Brieger, and others. See a review of this part of the subject by Mr Wm. Anderson, of St Thomas's Hospital ('Lancet,' Feb. 4th, 1888). In Eichhorst's 'Handbuch' tetanus is no longer found with other affections of the nervous system, but with infective diseases.

Diagnosis.—This is seldom difficult. It is only at the very commencement of *trismus* from dental irritation in infants that one could mistake it for true "lockjaw." Rose says that even at the earliest period of tetanus he has always been able to discover a certain degree of stiffness of the back of the neck, the patient being unable to bring the chin freely down to touch the chest. Another practical suggestion of his is that by introducing one's finger into the patient's buccal cavity on each side one can feel the hard edge of a rigid masseter much more distinctly than from outside the cheek. Inability to open the jaws, from any chronic affection of the temporo-maxillary joints, is in this way easily distinguished from all forms of trismus.

Tetanus is sometimes simulated by *hysteria*. Sir Thomas Watson mentions an instance of this kind in a girl, who "would all at once be drawn into a position such that the top of her head and her feet alone supported her, while her body was bent backwards like a bow; then, after a time, with equal suddenness, the opposite position was assumed, her forehead and her knees being brought together." His statement suggests the criterion which is applicable to all cases of this kind; namely, the irregular and inconsistent nature of the symptoms. Another affection which has perhaps to be considered is *spinal meningitis*. A case given in 'Reynolds' System' as a typical example of the latter disease would show that it may very closely resemble tetanus; but, as Moxon argued, that may, after all, have been tetanus.

The most important point of diagnosis is between tetanus and the effects of *strychnia*. A boy, aged twelve, was brought into Guy's Hospital at nine o'clock one morning, suffering from opisthotonos, and from spasms of the respiratory muscles, so severe that he almost ceased to breathe. He was a druggist's boy, and confessed that between 7.40 and 8.30 a.m. he picked up some black stuff (afterwards ascertained to be extract of nux vomica) and put it into his mouth for liquorice, until he found it bitter, and spat out as much as he could. Ice applied to his spine gave him relief. From some urine passed at 1.20 p.m. Dr Stevenson succeeded in obtaining the colour reactions of strychnia and brucia. By that time his symptoms had passed off, and he was discharged from the hospital a few days later. The characteristic features of such cases are the suddenness with which opisthotonos and the most violent general spasms set in, the absence of persistent rigidity of the affected muscles during the intervals (so that the mouth can be freely opened), the fact that the hands are involved, and the rapid death or recovery which ensues. Even if the poison be administered in small doses, and repeated at frequent intervals, there is no reason to suppose that strychnia could simulate the progressive development of tetanus.

Fatal event.—As a rule tetanus ends fatally between the third and the

seventh days. The occurrence of death within twenty-four hours from the commencement of the disease is very infrequent. Among Mr Poland's seventy-two cases there were only two instances of it: one patient, a young man, who was attacked six days after admission with a compound fracture of the leg, died in nineteen hours; the other, a girl, who had been burnt, is said to have lived only four or five hours. The most quickly fatal case on record is one of a negro servant, who lacerated his thumb in breaking a china dish, and who was almost instantly seized with convulsions, and died in a quarter of an hour; but Sir Thomas Watson, although he cites it, expresses a doubt as to whether this was a genuine example of tetanus. In most instances death occurs during a paroxysm, and is believed to be often due to spasm of the diaphragm or of the glottis. Mr Poland mentions that in one of his cases the heart's action continued for a short time after the breathing had ceased. In 1875 a patient suffering from tetanus in Guy's Hospital on several occasions turned perfectly livid, so that he seemed to be at the point of death, although a clenched state of his hands was the only outward indication of an increase of spasm. At last, after four days, one of these seizures did in fact prove fatal. Bauer, in Ziemssen's 'Handbuch,' however, expresses the opinion that the danger from failure of the respiration in tetanus has been generally over-estimated, for carbonic acid poisoning, he says, relaxes the muscles before death can take place.

There is no doubt that the immediate cause of death is sometimes sudden failure of the heart. Rose relates an instance in which he had his finger on the pulse when it suddenly stopped for ever; but here there was also hyperpyrexia present. Some observers have supposed that in cases of this kind the ventricles are seized with spasm. Stress has been laid on the fact that after death the heart is often found closely contracted, but this is in all probability the result of rigor mortis. Rose argues that the real cause of death is cardiac paralysis; he thinks that the muscular contractions oppose a resistance to the circulation through the arteries which the heart is unable to overcome.

Lastly, there are cases in which life is prolonged for three or four weeks, but which yet ultimately prove fatal. The cause of death is then generally exhaustion. Sometimes the spasms have altogether subsided, so that the patient is thought to be in a fair way towards recovery, although he is excessively weak and emaciated and prostrate, with sunken features and a scarcely perceptible pulse. Among the cases collected by Mr Poland there are two which proved fatal during a paroxysm as late as the twenty-first or the twenty-second day, and two others in which death was referred to exhaustion on the sixteenth and the thirty-second days respectively.

Convalescence.—When recovery from tetanus takes place a certain degree of stiffness of the muscles often remains for a considerable time, and very slowly passes off. Mr Poland mentions a case in which there was still some stiffness of the jaws after a year had elapsed. In 1871 Mr Golding-Bird had a boy under his care in Guy's Hospital in whom the spasms lasted for fifty-one days, and even after they had subsided his limbs still remained rigidly flexed, he became exceedingly emaciated, and bedsores developed, so that, although he was floated on a bath on water, he ultimately died on the one hundred and seventh day.

Course.—Although almost all writers recognise an acute and a chronic form of tetanus, they generally admit that no line of demarcation can be drawn. It is true that when successive cases are arranged in groups according

to the length of time which happened to elapse between the injury and the onset of trismus or of stiffness of the neck, a rapidly fatal termination is found to occur more frequently in those in which the trismus is early than in those in which it is late. A similar difference in intensity, although not so marked, seems to be observable in cases in which the primary lesion is extensive and severe, as compared with those in which it is slight. It is therefore not surprising that instances of "idiopathic" tetanus should, as a rule, be less urgent in their symptoms than those which are of "traumatic" origin; and since the subsidence of the disease, in those cases which recover, is never otherwise than gradual, one can understand how it has come to pass that the idea of chronic tetanus has become associated not only with a hopeful prognosis, but also with the absence of a severe injury, or of any injury at all; or, again, with the occurrence of a long interval since the date of an injury, rendering the connection between them doubtful. But although this is tolerably correct, so far as groups of cases are concerned, it is very apt to mislead when applied to an individual patient. In some instances of idiopathic tetanus death ensues as rapidly as in most of those which follow quickly upon a severe compound fracture or a burn. The division into acute and chronic tetanus had better be given up altogether.

Prognosis.—The only ground on which a forecast of the disease can with any safety be based is that afforded by the rate at which the symptoms of the disease are developing in the particular case under observation. Unfortunately it must be added that rapid progress is much more surely indicative of a fatal ending than is a slow course of a good prospect of recovery, or even of certainty that death is very near at hand. Spasm of the respiratory muscles sometimes destroys life suddenly and almost without warning. The average mortality of the disease among the seventy-two cases collected by Mr Poland in 1857 was 86 per cent.*

Treatment.—The difficulty of prognosis in tetanus leads to a corresponding difficulty in estimating the results of treatment. There are, indeed, certain measures the propriety of which is obvious. The patient should be placed in a quiet, dark room, and should be withdrawn from all noise and bustle, and from the visits of too many friends. He should speak as little as possible. The food which is given to him should be nutritious but in a fluid form, with wine or brandy in full quantities. If he is unable to swallow, enemata of beef-tea and brandy may be administered at regular intervals, unless even this brings on paroxysms of spasm, as is too often the case. Rose makes the practical suggestion that anæsthesia should be induced regularly once or twice a day by chloroform, for the purpose of injecting food into the stomach through a tube. Purgatives should not be given after the commencement.

Probably amputation of an injured limb or division of a nerve leading

* Some years ago Dr Wilks and the author saw with Dr Anderson, of New Cross, a gentleman whose symptoms seemed to deviate from the ordinary features of tetanus. It was one of those instances in which the paroxysms constitute the most marked element of the disease, but in which, although they recur but seldom, they destroy life early. The patient's main complaint was of a spasmodic pain in the right hypochondrium, which he attributed to wind. When it came on he used to get up and walk about, declaring that he could not lie down; he would stand leaning against the bedpost, and would call to his wife to rub his back. About a fortnight previously he had run a garden fork through his great toe; four days before we saw him he began to complain of stiffness of his jaws, and from that time he was unable to open his mouth fully. These facts led to a strong suspicion that the abdominal pain was due to tetanic spasm. He died three days after our first visit, and about twenty-four hours after sitting up in bed to make his will.—C. H. F.

from a wound are of no avail. But it must be remembered that a splinter of wood or some other foreign body has been now and then unexpectedly found under a cicatrix, or embedded in a nerve-trunk.

As regards the treatment of tetanus by drugs, we know of no medicine sufficiently potent to arrest the course of this terrible malady. When it runs a rapid course, one can often do nothing better than keep the patient continuously under chloroform, so as at least to secure euthanasia.

On the other hand, when the progress of tetanus is slow, it often seems that the death of the patient is little more than an accident, from the supervention of a paroxysm which happens to exceed a certain limit of severity. In such cases one might hope that narcotic medicines, or those which relax the muscles, would save life. With this object, as well as to relieve the patient's sufferings, *opium* has been often employed. Sir Thomas Watson mentions the case of a lady who took more than four ounces of laudanum a day during twenty days, and who recovered. And he refers to another case in which an ounce of solid opium was swallowed in divided doses every day for three weeks. Of late years the hydrate of *chloral* has sometimes been used in cases of the same kind with apparent success. In 1870, for instance, Mr Birkett had under his care at Guy's Hospital a man, aged twenty, who on June 24th had received a kick on an ulcer. Next day he felt rigidity of the muscles of the face and was unable to swallow solids. He was admitted on the 28th. At first thirty grains of chloral were given every four hours, but on the 30th, thirteen doses having been taken, a grain of opium was ordered to be taken every three hours instead. However, the spasms became more severe; and on July 4th a drachm of chloral was prescribed at one dose, and half a drachm on the following evening. After this fifteen grains were given every other hour until the 11th, when the quantity was increased to twenty-five grains every other hour. Two days later it was reduced again, and the disease now gradually subsided in the course of the next three or four weeks. A full report of this case will be found in a paper in the 'Guy's Hospital Reports' for 1878 by Dr Frederick Taylor, who remarks that in some other instances chloral has been found to give rise to great drowsiness, without much affecting the spasms.

Among the depressants of muscular action *physostigma* has been employed at Guy's Hospital in one case which ended favourably. The patient was a man aged twenty-one, who, having got drunk on November 30th, 1874, began to suffer from stiffness in the back on December 1st. He was admitted, under Dr Wilks, with fully-developed tetanus, on December 5th, and spasms recurred every three or four minutes. The extract of Calabar bean was given at first in small doses, but afterwards one grain every two hours; and on the 10th, after a very severe seizure, seven doses of a grain each were administered at intervals of fifteen minutes in succession. He began to improve about December 17th, but the stiffness of the joints remained until after the middle of January, 1875. Against this case must be set another one under the author's care in the summer of 1875, in which death occurred on the sixteenth day, in spite of two and a half grain doses of extract of physostigma repeated at intervals of only an hour. Previous to these two cases was one published in the 'Practitioner' in 1874 (vol. xiii, p. 345), in which enormous doses of physostigma were followed by recovery.

Subcutaneous injections of *curare* were administered to a man under the care of Mr Durham in 1876; but it seems doubtful whether the doses

which were employed were sufficiently large to be really efficacious, although the report says that they were followed by sleep and relief of pain.

Another medicine which has now and then been used in cases that ended in recovery is quinine. An instance of this was recorded by Dr Bright in the first volume of the 'Guy's Hospital Reports.'

In the collection of cases published by Dr Taylor in the same 'Reports' for 1878, there are recorded nine in which death occurred under chloral (six by the fourth day, and the other three on the eighth, tenth, and twelfth days), and seven which ended fatally under Calabar bean (six by the fourth day, the seventh on the eighth day).

CHOREA.*—The term St. Vitus's dance—*chorea Sancti Viti*—was originally applied to the dancing mania which prevailed in certain parts of Germany in the fourteenth and fifteenth centuries. It was also known as St John's dance. It is said that the designation is first met with in the account of an epidemic at Strasburg in 1418, when those who were attacked were sent to the chapel of St Vitus, at Zabern, to be calmed by religious processions and masses. Such cases will be hereafter mentioned under hysteria, and are related to some forms of saltatory spasm (p. 701).

The disease now to be described was named chorea first by Sydenham,† and for a long time afterwards Sydenham's chorea was distinguished as "chorea minor," or "chorea Anglorum."

Symptoms.—These are chiefly two: inability to keep at rest while awake, and incapacity to perform voluntary movements with precision. If the patient means to sit or to stand still, she (for chorea is more common in girls than in boys) begins after a few seconds to fidget. She shuffles her feet over the floor, or throws one foot over the other, or twists it first outwards and then inwards. Or, perhaps, she lays her hand palm upwards upon her lap, and then suddenly reverses it. Or she may shrug up one shoulder, or throw it forwards, or open and close her mouth or her eyelids without purpose. The muscles of the face are continually working. She probably is herself unaware of some of the movements, as, for instance, when a squint develops itself, or when her eyeballs are jerked from side to side. If one asks her to show her tongue she often seems at first unable to put it out, but afterwards she suddenly thrusts it forwards, and then as suddenly withdraws it, her jaws snapping together in front of it. If she wishes to carry a cup to her mouth she cannot help throwing her arm in various directions, and accomplishes her object only after several unsuccessful attempts, and then she perhaps seizes the edge of the vessel with her teeth, and is obliged to gulp down all its contents at once for fear of spilling them. When she tries to walk she moves by fits and starts, and jerks her body and limbs first to one side and then to

* *Synonyms.*—Chorea Sancti Viti minor—Chorea minor—Chorea Anglorum.—*Fr.* Danse de Saint Gui.—*Germ.* Vitustanz.

† "De Chorea Sancti Viti: Convulsionis species quædam est, pueros puellasve a decimo ætatis anno ad pubertatem invadit, &c. . . . . Antequam poculum ad os possit addacere, mille gesticulationes, circulatorum instar (like a mountebank), exhibebit. Non enim rectâ lineâ ori admovet, sed deductâ a spasmo manu, huc illuc aliquandiu versat, donec tandem forte fortunâ labris proprius apponens liquorem, repente in os injicit et avide haurit" (Sydenham: 'Processus integri in morbis curandis,' 1693). It is remarkable, that while Romberg calls Sydenham's original description of chorea masterly, and von Ziemssen says that it leaves nothing to be desired for clearness and precision, Dr Sturges thinks we must suppose either that the period and manner of its attack have changed since Sydenham's time, or else that this observer was in gross error in his description of what he saw.

another. Attempts at voluntary movement bring on the choreic symptoms worse, whereas in slight cases a child may lie quiet in bed or sit still, without anything seeming to be wrong until she is spoken to.

The respiration is disorderly in rhythm. The articulation is apt to be hurried. The patient, if told to count, may count several numbers one after another with explosive violence, and then pause to take a deep breath; or she utters only one sound with each expiration, drawing in air hastily before she goes on to the next. Von Ziemssen says that he has seen with the laryngoscope unsteady and quivering movements of the muscles of the glottis, and that an imperfect degree of tension of the vocal cords is shown by the low pitch and monotonous character of the voice, and by the short time that a note can be kept up in singing. Romberg relates a case in which there was annoying hiccough, and two others in which inspiration was attended with a whistling noise; in one of them the chest was observed to be suddenly drawn inwards by spasm of the intercostal muscles. According to Trousseau deglutition is sometimes impaired.

During sleep the choreic movements cease. Jaccoud also remarks that in slight cases they sometimes remain absent for a while after the patient awakes. Marshall Hall, however, is said to have observed that if she dreams they may for the time return during her sleep.

Rosenthal and Benedikt have tested the reaction of the muscles to faradic and galvanic currents, and have found their excitability greater than under normal conditions. There is no reaction of degeneration.

When chorea is slight or of moderate severity the spasms are often confined to one side, so far as the limbs are concerned. This has been called "*hemichorea*," but a special name is hardly needed. Dr Hughlings Jackson has pointed out that, even in these cases, the muscles of the trunk and face are always affected bilaterally, a fact of much theoretical interest. Different statements have been made as to the relative frequency with which the right and the left limbs are affected. According to Reynolds and Jackson the former more commonly exhibit the movements; according to Austin Flint, Trousseau, and Jaccoud, the latter. Dr Anstie also remarks that one may often at the first glance recognise a child affected with slight chorea from her sitting with her right hand grasping her left wrist to keep it still. Among thirty-three cases out of 150 in which the affection was unilateral, there were eighteen on the left to fifteen on the right side ('Guy's Hosp. Reports,' 1873, "Rheumatism and Allied Disorders"). Since then, among 164 additional cases, the writer found choreic movement decidedly predominant in the right arm or leg, or both, in seven cases; and in the left in thirteen.

Trousseau states that the patient experiences formication and tingling, or even anæsthesia; but these symptoms are very rarely mentioned in the reports of cases admitted into Guy's Hospital. Choreic patients often complain of headache, and Sir Thomas Watson remarks that he has sometimes known the pain to be limited to the side of the head opposite to the affected limbs. The pupils are generally dilated and torpid, and in one case von Ziemssen observed that the pupil was much larger in the eye corresponding with the affected arm and leg. Rosenthal noticed that the pupils return to their normal condition when the chorea subsides.

Children affected with chorea are generally pale, often decidedly anæmic. They may also be thin and delicate in appearance, but this is often due to

previous rheumatism and cardiac disease; and chorea may be seen in stout and rosy children. We must remember that the favourite age of the disease is not early childhood, but the period between eight and fourteen years of age, when both boys and girls, even when in sound health, are usually slender and often pale if their appearance is compared with the red cheeks and plump limbs of their earlier childhood.

The temperature is normal.* The urine is sometimes scanty and high coloured, in marked contrast with the pale, abundant urine of hysteria. According to Dr Bence Jones there is an excessive secretion of urea, and others have said that the amount of lithates is increased. Analyses made in two cases by Dr Handfield Jones ('Clin. Trans.,' iv) yielded contradictory results.

The pulse is commonly quickened, especially when the movements are severe. Some writers assert that the heart's action may be irregular or intermittent, but this is quite exceptional, even when there has been previous organic disease.

On auscultation a blowing *systolic murmur* is often heard at the apex. With regard to the significance of this sign widely different opinions have been held. Many observers have maintained that the bruit is functional; some that it is anæmic; others that it is due to choreic spasms of the musculi papillares interfering with the due closure of the mitral valve. But when the disease terminates fatally that valve is almost invariably found studded with vegetations like those of rheumatic endocarditis. Since the murmur also is the same in the two diseases it seems reasonable to attribute it to the endocarditis in both. In many cases no bruit is audible. Its absence, however, is not to be taken as a proof that the valve is unaffected, for Kirkes and Wilks have both recorded instances in which vegetations were found after death, but in which the heart-sounds during life had been normal. Again, in some cases in which a murmur is present it passes off as the patient recovers from the chorea. This fact has been regarded as a proof that at least in these cases it is due to functional disturbance, and not to a valvular lesion; but a precisely similar disappearance of the murmur is often observed in cases of rheumatism.

Among 150 cases of chorea in Guy's Hospital during the years 1870–71–72, the state of the heart is not mentioned in eleven, and the presence of any abnormality is expressly denied in eighty. A systolic bruit was heard in the remaining fifty-nine, and in forty-three of them it was distinctly loudest at the apex. In only nine is it described as basic; two of these were indistinct and two were combined with a diastolic murmur.

Among 180 fresh cases in the same hospital during the five years 1874–78, collected for the writer by his house physician Dr George Halstead, sixty-two were reported to have a murmur of some kind (again about a third of the cases); of these it was an apex systolic bruit in fifty-two, a basic bruit in seven (of which two were diagnosed as aortic and two as pulmonary), a præsystolic apex bruit in two, and a pericardial rub in one.

* The temperature cannot be taken in the mouth or the axilla. The rectum, or the stream of urine (p. 37, note), is the proper place for the thermometer. The fact that the constant muscular movements do *not* raise the temperature of the body is of great physiological interest. Dr Woodman and Dr Long Fox (Wunderlich, Syd. Soc. Trans., p. 426) have found it as low as 97° or 96°, although on the other hand is quoted Dr Finlayson's case (probably a complicated one), in which the average evening reading of the thermometer *in recto* was 103·2° (cf. *infra*, p. 744).

In the next ten years (1879–89) Dr Goodall reports a bruit in 143 cases out of 262.

Varieties.—In exceptional cases of chorea the symptoms are but little marked at its commencement, or even throughout its whole course; but there is marked *loss of power* in one or more of the limbs. Thus, the patient may complain that her arm feels heavy, or may drag her foot slightly in walking. Such cases are often brought to one as paralytic. In other instances she merely has a trick of bringing a particular set of muscles unexpectedly into action, so that she makes a grimace, or throws her hand or arm into an odd position. Or she may let a jug fall, and this may happen two or three times in succession in the course of a week or ten days. Or it may be noticed that she does not write her exercises so well as she used, or that she no longer plays steadily on the piano. All these things are apt to be set down to carelessness, and her parents and teachers are the more likely to blame her because she often becomes inattentive and forgetful, apathetic or peevish.

Severe and dangerous cases are happily the exception. They have been separately described as *chorea gravis*. The patient becomes unable to stand; even when recumbent her limbs are tossed about, so that boards well padded have to be fixed on each side of her bed to prevent her throwing herself on the floor. In spite of all the precautions that can be taken, she is very apt to bruise herself and to rub off the skin from her elbows and knees, so as to cover them with crusts and sores. She ceases to sleep altogether, and the violent movements go on day and night without intermission. In such cases emaciation takes place with wonderful rapidity. Dr Tuckwell has related the case of a boy who was wasted to the utmost at the time of his death, but who six days before had borne the appearance of vigorous health, with remarkably well-developed muscles; and conversely von Ziemssen mentions one in which a girl, eleven years old, gained during convalescence 5½ lbs. in ten days, her weight rising from 55½ to 61 lbs. in that time. One cause of the extreme wasting which accompanies severe chorea is doubtless the difficulty with which food is administered. The patient often seizes a spoon, or the spout of a feeding-bottle, as if she would bite it in two, and so injures her own teeth, or her nurse's fingers.

The mental state of a patient affected with severe chorea is often one of mania. She may be continually shouting, singing, and talking at random. Or she may unexpectedly perform some strange action; for instance, a girl who was in hospital some years ago, suddenly got out of her own bed, and turned a somersault across that of another patient. There is no necessary relation between the intensity of the choreic movements and that of the psychical disturbance. Dr Wilks says that the mind remained perfectly clear in one of his patients, who had the worst attack of chorea which he ever saw terminate favourably. On the other hand, there occurred some years ago in Guy's Hospital a fatal case in which there were such marked mania and so complete a loss of consciousness, while at the same time the movements were so like those seen in epilepsy, that the diagnosis remained doubtful until the autopsy was made, when recent vegetations were found upon the mitral valve. Dr Hills, of the Norfolk County Asylum, knew more than one choreic patient who was sent to that institution as insane.

Loss of power of speech is common in all but slight cases of chorea, and is constant in chorea gravis.

Course.—Chorea may be said to be generally a chronic disorder, but its

duration is very variable. Almost all the statistics which have been published concur in stating a period of from two to three months as the average. Thus Wicke found it eighty-nine days in a series of 125 cases, and Sée sixty-nine days in one of 117 cases; while from much smaller data Gray, Tuckwell ('Lancet,' 1871), and Hillier in this country each made it about ten weeks. Individual cases, however, range widely on each side of these limits; but far more widely *beyond* the average duration than *within* it. Hence the introduction of one or two very prolonged cases may greatly disturb the statistical result; patients sometimes apply for treatment in whom the disease has already lasted for many months or even for two or three years. A single instance of this kind would inevitably spoil, for the purposes of comparison, any series which did not embrace a very large number of cases. As a rule, chorea is more likely to last long when its symptoms are comparatively mild; patients who are obliged to have boards placed along the sides of their beds often get well before those who are able to be up and about the ward throughout their stay in the hospital.

Fatal event.—Children very rarely die of chorea. At Guy's Hospital between the years 1848 and 1875, there were only twenty cases of the disease which terminated fatally. And of these five must be left out of consideration, since death was due to an accidental complication, dysentery, diphtheria, rheumatic pericarditis, or cardiac dropsy. Of the remainder there are only four in which the patients were below the age of fourteen; two were seven years old, one twelve, and one thirteen. The remaining eleven patients were more than fourteen years old, nine of them between the ages of fifteen and eighteen, one forty years old, and one fifty years old. Few of the women seem to have been pregnant; but it is an ascertained fact that the mortality is very great in this condition, which frequently ends in abortion or premature delivery. It seldom happens, even in the most severe forms of chorea, that the patient dies within three or four weeks from the commencement of the disease. One of the most rapid cases on record is the one referred to above, in which there was at first a doubt between epilepsy and chorea; the whole duration of the patient's illness was said to be only two or three days. In 1853 a girl aged sixteen, who was already in the hospital for syphilis, was attacked with maniacal chorea, and died in six days. About two months previously, another case, in a boy of the same age, had terminated fatally in nine days. The immediate cause of death seems to be almost always the intensity of the nervous symptoms themselves, but the movements often subside, and may even cease entirely during the last few hours, the patient lying comatose and passing urine and fæces involuntarily. The temperature of the body may rise during this period of the disease. In one case it was 104·7° before the patient died, and Dr Frederick Taylor observed in a case in our clinical ward the thermometer register a temperature of 108° immediately after life was extinct. It does not appear that endocarditis or pericarditis is directly concerned in bringing about the fatal issue. The presence of bronchitis has been noted in several of the reports of autopsies at Guy's; in one instance it perhaps accelerated the patient's death. In 1873 Dr Habershon had a fatal case in a boy aged twelve, who had been three weeks in the hospital when his breathing became obstructed by swelling of the tongue. Tracheotomy was performed, but without saving the patient's life. Probably the glossitis was the result of injuries inflicted by the teeth, for in another instance, in which the tongue had been severely bitten in two places, there was dis-

covered after death a foul ulcer which exposed the sublingual gland and extended for the depth of an inch into the muscular and other tissues.*

Four fatal cases occurred among the 150 tabulated by Mr Manser for the 'Guy's Reports' (Third Series, vol. xix, pp. 329—332). 1. F., thirteen, 5th March. Mitral and aortic vegetations, ulcer from biting tongue. 2. F., seven. Aortic vegetations, faucial diphtheria. 3. F., pregnant; mitral vegetations. 4. M., thirty-eight. Aortic and slight mitral endocarditis.

Among 180 cases collected by Dr Halstead from the records of the same hospital, during the five years following those which furnished the above statistics, there were only five deaths. 5. In a little girl of seven, who died after being six weeks in the ward, there were found the usual fibrinous vegetations on both mitral and aortic valves. 6. In a girl of fifteen, after a month's treatment, there was, beside the same almost constant lesion, lobar pneumonia and a bedsore. 7. In a girl of eight there were mitral and aortic nodules with broncho-pneumonia and adherent pericardium. 8. In a girl of fifteen there was mitral endocarditis, adherent pericardium, and a "cardiac" lung." 9. In a lad of seventeen, who had suffered again and again from chorea since he was nine years old, no lesion but mitral vegetations was found.

If to these nine fatal cases we add the seven additional cases numbered 5—10 in the paper above referred to (vol. xix, p. 333), we have a total of sixteen *post-mortem* examinations. In every one of these there were fibrinous nodules found on the mitral or aortic valves. In only two of these was there a history of rheumatic fever, and in seven its occurrence was explicitly denied. Diphtheria was apparently the cause of death in three cases. In three cases there was acute lobar pneumonia, and in a fourth lobular inflammation of the lungs. Only one of the patients was pregnant.†

Recovery.—This is generally gradual. Sometimes, however, the breaking out of an exanthem, or of some other febrile disease, is followed by the sudden subsidence of the movements. This is in accordance with an aphorism of Hippocrates: *spasmos febris accedens solvit.* Dr Radcliffe states that he has met with seven cases in which it occurred; and some striking examples of a similar kind are recorded by Rilliet and Barthez. According to the observations of Sée, however, the neurosis is not likely to be cut short unless it had reached its acme of development before the commencement of the fever. In many instances the first effect of the latter is to aggravate the movements, and yet in those very cases they may cease when the crisis of the fever has passed.

It sometimes happens that the subsidence of the spasms in severe cases of chorea is not followed by any evident amelioration in the patient's general condition. She may become perfectly quiet, and yet may remain for two or three weeks unable to stand, and with little power in her arms. This paresis may be limited to a single limb, or to the two limbs on one side, the "choreic hemiplegia" of Dr Todd. In association with such symptoms, or independently of them, the intelligence may remain defective; or a condition of mania or melancholia may develop itself. Dr Barnes met

* According to Dr Radcliffe and von Ziemssen meningitis occurs in some cases of chorea, and in others myelitis; and they accordingly mention those affections as sometimes bringing chorea to a fatal termination; but we may venture to doubt the diagnosis of any case in which unmistakable inflammatory changes were not discovered either in the membranes of the brain or in the spinal cord.

† Among 439 cases published by the Collective Investigation Committee only nine deaths occurred ('Brit. Med. Journal,' February 26th, 1887).

with a case in a pregnant woman, who as she regained her strength became insane and had to be removed to Bethlem. Other patients, after the choreic movements have passed off, still remain absolutely silent for days together, making no attempt to reply to the inquiries of their friends. Dr Hughlings Jackson associates such defects of speech with right hemiparesis.

Alarming as these various symptoms are, they almost always pass off in their turn, and the patient ultimately regains a state of perfect health.*

The strong tendency of chorea to relapse will be mentioned further on.

Pathology.—The nature and causes of chorea are so intimately connected together that it is impossible to discuss the one apart from the other. They involve problems which are of great interest, and have important bearing upon the ætiology of neuroses in general.

In the first place, there are still differences of opinion as to whether the seat of the disease is in the spinal cord or in the brain. When the functions of nervous centres in the cord were first discovered, and when it was found that frogs and other animals could perform co-ordinated movements after removal of the cerebrum, it was natural that attempts should be made to refer to disorder of those centres all affections of which the main symptoms are irregular motions of the body and limbs. Thus Romberg, writing in 1851, placed chorea among the "spinal spasms;" and so recently as 1873 Jaccoud defended a similar doctrine in an elaborate argument. In England, however, the opinion has for some years prevailed that the disease is seated in the sensori-motor ganglia, at the base of the brain, and especially in the corpora striata.

In favour of the spinal theory of chorea, certain experimental results have been adduced. Dogs are liable to a similar complaint, and Chauveau conceived the idea of cutting through the cord close to the skull during the progress of canine chorea; he found that after this had been done the movements went on in exactly the same way as before, the spasmodic contractions of the diaphragm being in one instance sufficient to keep the dog alive for three hours. In two other experiments artificial respiration had to be maintained; but the result was the same as in the first observation, so far as concerned the choreiform spasms. Moreover, a second division of the cord, in the lower dorsal region, put an end to the motions in the tail and in the other parts beyond the section. Chauveau's experiments have been repeated by Legros and Onimus; and they have ascertained, in addition, that by irritation of the posterior columns of the divided cord the spasms were increased, while they were arrested by complete section of the same structures.

It is, however, now ascertained that canine chorea is not identical with the disease of that name in man,† and, even if it were, there remains the question, whether in the human subject the higher nervous centres do not assume functions which in brutes are performed by lower ones. English pathologists have been able to make out a strong case in favour of their own

* Trousseau, however, speaks of children who have never again shown the same degree of intelligence as before; von Ziemssen says that slight defects in the co-ordination of the movements, a precipitancy in the performance of certain manual actions, or a tendency to facial grimaces, may persist for years or even be permanent; and Dr Radcliffe is inclined to think that chorea is apt to be followed by other neuroses, particularly epilepsy, at a later period of life. Dr Bristowe mentions a case in which there was a degree of rigid flexion of the hip- and knee-joints, with overlapping of the knees from preponderant action of the adductors of the thighs, and a tendency to talipes equino-varus,—conditions which seemed to him to indicate degenerative changes in the lateral columns of the cord.

† See Mr Victor Horsley's Lectures ('Lancet,' 1886, vol. i, p. 54).

opinions. Some of the points were stated by Dr Russell Reynolds as far back as 1855. As he remarked, the spasms produced by persistent irritation of the cord are tonic rather than clonic; and it is inconsistent with a spinal origin that the choreic movements are in any degree capable of being controlled by the will, that they are increased by emotions or by voluntary efforts, that they cease during sleep, and that they should be diminished by direction of the patient's attention to other objects. In addition, Dr Broadbent ('Brit. Med. Journ.,' 1869) has insisted on the fact (which Romberg had previously observed) that tickling the palm of the hand or the sole of the foot of a child affected with chorea leads to no increased spasms; on the contrary, it is borne without difficulty, and the excitability to reflex actions sometimes seems to be less than in health. Another point on which he lays still more stress is that the spasms are so often unilateral. An affection of one half of the spinal cord throughout its whole length, from the crus cerebri downwards, without implication of the other half, is, he declares, scarcely conceivable. Moreover the improbability of such a localisation of the disease is increased by the fact that the muscles of the face and trunk are bilaterally affected, these very muscles being liable to be set in action on both sides by stimuli derived from a single corpus striatum, whereas there is no reason to suppose that disturbance of one half of the cord could affect them in a similar manner. Lastly, the fact that the mental faculties are so frequently impaired in severe cases of chorea would naturally incline one to localise the disease as near as possible to the hemispheres.

Dr Dickinson made a series of anatomical investigations in fatal cases of chorea ('Med.-Chir. Trans.,' 1876), from which it would appear that certain morbid changes, consisting of congestion of the vessels, of periarterial degenerations, and of minute spots of sclerosis, are discoverable in the upper regions of the spinal cord, as well as in the lower parts of the brain. Even if the constant occurrence of the appearances which Dr Dickinson has described should be fully established, it would still be a question whether they are not merely secondary effects of the disease. Whatever may be the starting-point of the choreic spasms, one cannot doubt that all the nervous structures which lie between that point and the muscles must have their functional power strained to the utmost during the continuance of such violent movements; and therefore we might expect them to exhibit degenerative changes. It is important to note that Dickinson's cases had all reached an advanced stage. Moreover, it is clear that the persistence of the morbid changes which he describes is compatible with the subsidence of the spasmodic movements and the restoration of health. For in one patient who had twice before had chorea (the last time having been a year previously), the duration of the fatal attack was only thirteen days; and yet changes of old date—periarterial degeneration and scattered spots of sclerosis—were found, besides recent congestion of the cord and basal ganglia.

On the whole, then, the corpora striata seem to be a more probable seat of chorea than the cord—one when the disease is unilateral, and both when all the four limbs are affected.

Another possibility, however, is that the seat of the "discharging lesion" in chorea is the motor region of the cerebral cortex, and many of the objections to the other proposed localities do not apply to this—partly perhaps because its functions are still imperfectly known.

The next question is as to the nature of the anatomical change. Dr Broadbent maintains that the spasmodic movements of chorea indicate that

the functions of a particular part of the brain are deranged. To quote his words, chorea is "a symptom, not a disease. It has been called an insanity of the muscles; it would be better designated a delirium of the sensori-motor ganglia, since it bears the same relation to those parts that the delirium which may occur in a variety of maladies bears to the cerebral hemispheres."

Now, it is possibly true that choreiform spasms may accompany different morbid states of the nervous centres. But it is no less true, and far more significant, that the disease described in the preceding pages as chorea is not met with as an accidental complication of other maladies, but occurs in a particular class of patients, and under conditions peculiar to itself.

Dr Sturges, in his interesting 'Lectures on Chorea' (1877), points out with much acumen how choreic movements find a parallel in the nervous twitchings of mental embarrassment or the restless and purposeless movements of a fidgety child. Yet, admirable as is the critical and negative part of his remarks, it remains true (1) that if a functional disease, the symptoms of chorea must yet depend on functional disturbance (*i. e.* disordered nutrition from irregular supply of blood, or from some poison, or from a molecular change of other origin) which affects one part of the nervous muscular apparatus and not another. Syncope results from anæmia of the cerebrum, not of the liver; tetanus from strychnia affects the cord, not the bulb; and epilepsy depends on some unknown minute derangement in the cells of the cerebral cortex, not in those of the retina. Our business is to fix the "seat" of a disordered function as much as of an impaired structure. (2) It remains true that, while no line can be drawn between health and disease, or between the gravest premonitions of an impending malady and its slightest early symptoms when developed, we must nevertheless recognise certain limits beyond which slight or recoverable deviations become serious enough to threaten comfort or life, or where they lose the accidental character of individual variation and assume the constant and common characters of "recurrent concomitant symptoms" (*Symptomencomplex*). We then know that a disease is before us, according to the only reasonable definition of the word; a condition which has to be understood and treated by a skilled adviser. (3) The fact that an excitable and restless child may often be seen to make movements like those of chorea only proves that choreic spasms are physiological; that the "disease" is an exaggeration and perversion of the action of natural nervo-muscular mechanisms. So the diurnal variations of temperature in health are preserved in pyrexia; so the several primitive layers of the embryo maintain their peculiarities when they give rise to morbid growths; and so the reflex spasmodic neuroses described in the beginning of the present chapter are only perversions of normal excito-motor functions. (4) Like other diseases, chorea has its own natural history, its beginning, middle, and end, its proclivities, and its antipathies. In fact, according to the principles laid down in the first chapter of this book, there is scarcely a member of the whole nosology which better deserves to be called "a disease" than St Vitus's dance.

Sex.—Chorea is much more common in *females* that in males; and (like hysteria) it shows its predilection for the former sex in the case of children before the age of puberty; but, unlike hysteria, it is much more apt to occur in children than in adults.

Tabular statements in regard to these points have been published by the writer in the nineteenth volume of the Third Series of the 'Guy's Hos-

pital Reports.' He found that among a hundred and fifty patients 42 were males and 106 females. These numbers were compiled by Surgeon Manser in 1873. Dr Halstead found the corresponding figures for 1874—1878 were 43 male to 129 female patients. Dr Goodall, our present Medical Registrar, has been good enough to complete these statistics down to the present time. He finds that in the eleven years 1879—1889 we had in our wards 74 male and 188 female patients with chorea. The late Dr Hughes, among 100 cases, found 27 boys and 73 girls (*ibid.*, Second Series, vol. iv, p. 372), and among 209 cases 42 male and 167 female patients (*ibid.*, Third Series, vol. i, p. 245). So that the proportion in a large London hospital would be about one fourth male to three fourths female cases (228 to 663).

Of 531 cases treated in the Hôpital des Enfans Malades at Paris during twenty-two years,* 138 occurred in boys and 393 in girls, *i. e.* rather more than one to three. (We shall presently see that the excess of female patients is less among those under puberty.) Among 422 children under twelve treated for chorea at Great Ormond Street, Dr Hillier found 122 boys to 300 girls, again rather more than one to three. Among 141 children at the Evelina Hospital, Dr Goodhart had 43 boys and 98 girls. A large proportion of boys was also found by Rufz at the Children's Hospital at Paris from 1824 to 1833 (overlapping the first three or four years of Sée's period at the same institution), viz. 51 to 138; and in Wincke's monograph on chorea published at Leipzig in 1844, the proportion was 117 to 210, or more than half the patients were boys, whose ages ranged from four to eighteen. If we put together the statistics mentioned in this paragraph (statistics which, with the partial exception above noticed in the Paris cases, are independent of each other) we find that the total numbers are—of 1610 patients, 471 male to 1139 female, or a proportion of about 2 to 5.

Among 436 cases of the Collective Investigation Committee the proportion of the sexes was 115 to 322, or again rather more than one to three.

Age.—Chorea is characteristically a disease of childhood, seldom or never seen in infancy, and rare after twenty, except in the case of puerperal or pregnant women. Sydenham's lower limit of "the tenth year" is, however, too high. From the fourth or fifth to the fifteenth year for boys, and to the seventeenth for girls, is the choreic period.

Of 216 patients with chorea observed in Guy's Hospital 187 were between six and seventeen years old. Of *first attacks*, among 322 patients recorded there, only two were two years old, and none under that age. The disorder first appeared in five cases between two and five years of age, in 102 between five and ten, in 134 between ten and fifteen, and in 55 between fifteen and twenty. Only two male patients were over twenty when first affected with chorea, one not yet twenty-one, the other thirty-eight; while there were six young women between twenty and twenty-six.

Of Dr Hughes's hundred patients in Guy's Hospital, only one was under eight years old (a boy of five), thirty-two were between eight and ten, forty-five between ten and fifteen, twenty between fifteen and twenty, one (a young man) was twenty-five, and two young women were twenty-two and twenty-eight. In his second series of 198 cases, nine were between four and eight years old, 148 were between eight and sixteen, and above sixteen there were only four men, aged 18, 20, 21, and 43.

In Paris, Sée found that out of 531 cases of chorea, 453 occurred between six and fifteen; in Philadelphia, Hammond found the proportion 67 out of 82;

* Sée, 'De la Chorée et des Affections Nerveuses,' Paris, 1851.

and in Germany, Rufz gives the corresponding numbers as 180 out of 189.

Exceptions.—Chorea is extremely rare over twenty-five, but besides the well-known liability of pregnant *women* (p. 739), we had what was an unexceptionable case in a man of thirty-eight, and several authentic instances have been recorded of the occurrence of chorea at an advanced age. Among the 439 cases of the Collective Investigation Committee there were only ten above twenty-five years old. Five of these were old women between sixty-three and eighty-six. Dr Graves mentions the case of an apothecary in Dublin who was attacked when seventy years old; Romberg saw chorea in an old woman of seventy-six, which had, however, begun when she was six years old; and Trousseau relates in detail an instance which came under the observation of Dr Henri Roger, in a lady, aged eighty-three, who recovered from the disease in five weeks. Charcot has described *senile chorea* as a distinct variety of the disease, but we may perhaps be allowed a doubt whether all the cases would stand criticism. On the other hand, examples are not altogether wanting of the presence of chorea in new-born infants. Thus Richter is quoted by von Ziemssen as having recorded two cases in each of which a female child was affected at birth, the mother having received a fright while advanced in pregnancy; the infant suffered while awake from clonic spasms, which were absent during sleep, and afterwards ceased almost entirely. A somewhat similar instance is related by Dr Long Fox as having occurred congenitally in an infant born six weeks before the proper time.

Still the fact remains, that in the great majority of cases chorea is a disease of childhood between the ages of six and fifteen years, or between the period of the commencement of the second dentition and that of puberty. The preponderance of females among those who are attacked by it is perhaps due to the sensitiveness and mobility of their cerebral organisation. It accords with this supposition that the children most liable to suffer are those who are delicate and excitable. As to the question whether chorea is especially apt to occur in those who inherit a tendency to the neuroses in general, there is some difference of opinion. Trousseau and Anstie answered this question affirmatively, Sée gave a contrary opinion. We must first agree what maladies we will include under the title. Epilepsy, hysteria, and insanity would be admitted by all; but some would take in infantile paralysis, others tubercular meningitis, or hemiplegia, or idiocy, or tetanus neonatorum—diseases which pathologically are widely different.

Among persons more than seventeen years old the preponderance of females is far greater than it is in children. In Dr Hughes's hundred cases there were seventeen women above the age of sixteen to five men. In the writer's first series there were twenty-one women above fifteen to four men, and in the second series twenty-seven women to four men.

Recurrence.—Another important point in the ætiology of chorea is its liability to recur again and again in patients who have once suffered from it. A large proportion of the patients admitted into any hospital have had the disease once or oftener before. Of 262 choreic patients admitted to Guy's Hospital from 1879 to 1889 inclusive, 102 had suffered previously. The writer has recorded the case of one young man who suffered from chorea every autumn from the age of fourteen to that of twenty-two, and of a girl who was attacked each May, from her eighth year to her fourteenth.

The duration of a relapse is generally less than that of the first illness, but Trousseau mentions some exceptions to this rule.

Mental shocks.—The immediate exciting cause of chorea is often a severe nervous impression such as a fright. Many striking instances of this have been placed on record. Thus Romberg relates the case of a girl, aged ten, who was one morning violently alarmed by a dog which jumped at her and barked, and who was seized with chorea the same evening. Again, von Ziemssen speaks of a boy, aged ten, who was terrified by a shot falling unexpectedly close to him on a field, and in whom the disease reached a great pitch of severity within a few hours. Trousseau gave the case of a girl, aged sixteen, who had been caught hold of by a man as she was going downstairs one evening without a light, and who was so frightened that she had a nervous fit, and from that moment became affected with St Vitus's dance. Bright described the case of a boy who had already recovered from an attack of chorea, and who was sleeping with his father when the latter was seized with a fit of apoplexy; the boy was so alarmed that his disease returned. A child, admitted into Guy's Hospital under Dr Pavy, had been frightened by seeing her brother in flames, ran out of the house screaming, scrambled over three walls, took refuge in a neighbour's house, and was brought home and put to bed. Her mother at the time noticed a peculiar twitching movement about her face and irregular motions of her limbs; next morning she was unable to stand, and chorea quickly developed itself.

There is no doubt that much exaggeration has prevailed in regard to the association of chorea with mental impressions, and that parents often jump too hastily at conclusions with respect to the "causes" of this as of most other maladies. But the cases just quoted seem to be beyond dispute, and they afford good grounds for supposing that the same cause has really been in operation in other instances where a longer interval has elapsed. Dr Hughes ('Guy's Hosp. Rep.,' 1856) related a fatal case in which the symptoms were slight when the patient was admitted, but became suddenly aggravated in consequence of a fright.

In other instances chorea is apparently the result of *imitation*. It has long been taught at Guy's Hospital, both by Dr Addison and by the physicians who have succeeded him, that not more than one or two patients affected with this disease should be placed in the same ward with other children, lest they should copy it. But it does not appear that any of the sisters or nurses remember an instance in which this has occurred. Some examples of the occurrence of chorea in an almost epidemic form have been placed on record, of which the most striking appears to be that related by Bricheteau. At the Necker Hospital one afternoon a young girl was admitted, suffering severely from the disease; in the evening a patient already in the ward, who had previously had chorea and was suffering from hysteria, began to exhibit choreic movements, and in twelve hours had to be tied down; next day two other cases occurred, and within the four following days five more, making eight in all: the disease might probably have spread still further had not the patients been from that time isolated. But might not this have been hysteria simulating chorea?

Pregnancy.—Another condition which plays an important part in the causation of chorea in girls after the age of puberty and in young women is *pregnancy*. Dr Barnes has collected ('Obstet. Trans.,' vol. x) a series of fifty-eight cases of this kind. The period of gestation at which the spasmodic movements are most apt to begin is from the first to the third

month, but sometimes it is much later; and two instances have been recorded in which they followed parturition. First pregnancies are much more often accompanied by chorea than subsequent ones. This of itself suggests that one element in the production of the disease may be the emotional excitement which necessarily arises in a woman who finds herself for the first time pregnant, and the suspicion is confirmed by Dr Wilks's observation that a large proportion of those who are attacked are unmarried girls, to whom their condition is one of shame and distress. Thus a connecting link is established between the chorea of pregnancy and that which occurs under other circumstances; and a still stronger one is the fact that in a considerable number of cases the patient has already had the disease on one or more occasions at an earlier period of life. Among the sixty-six cases already referred to there were fourteen in which previous attacks had occurred.*

Rheumatism.—So far the causal relations of chorea differ but little from those of the neuroses in general. It may be added that, according to Trousseau, chlorosis and anæmia are among the conditions which dispose to its development. We now pass to an entirely different predisposition which applies to this, but to no other nervous disease,—rheumatic fever.

As far back as 1811 rheumatism was stated to be one of the causes of chorea, in the Syllabus of Lectures on Medicine delivered at Guy's Hospital, by Drs Babington and James Currie, "It often follows rheumatism, and these two diseases often come on alternately." Dr Bright (whose testimony carries back the tradition nine years earlier) was himself convinced of the relation.†

Beside Bright and his pupils, the younger Babington and Hughes, Dr Copland and Sir George Burrows were early advocates of the belief that there is a real connection between chorea and acute rheumatism, and that the latter predisposes to the former. See introduced the same theory into France, and it is generally acknowledged in Germany and America. The connection is all the more probable because it is quite unexplained, and supports no theory of either disease.‡

The close association between chorea and rheumatism is shown by the fact that these two disorders are the chief and almost the only causes of valvular endocarditis. Out of over eighteen fatal cases of chorea which occurred in Guy's Hospital between 1848 and 1876, in only one was endocarditis absent; and that the frequency of its occurrence was not dependent merely upon the severity of the chorea is evident from the fact that in five of these cases the patient's death was due to some complication or intercurrent disease. The same conclusion is confirmed by the frequency with which chorea and rheumatism occur in the same patient; a child who has had rheumatism falls ill with St Vitus's dance a few years afterwards, or *vice versâ*; or slight choreic movements appear in the course of a rheumatic attack, or rheumatic synovitis recurs in the course of chorea.

* See also Dr Lever's paper ('Guy's Hosp. Rep.,' Second Series, vols. v, p. 3, and vi, p. 223).

† He thought that an intervening link between the two disorders was to be found in the presence of pericarditis, from which he imagined that irritation was transmitted to the nervous centres, just as in other cases it might be communicated from the intestines or the uterus. In support of such a view there certainly is a case recorded by Dr Wilks, in which pericarditis caused by renal disease was accompanied by choreic movements. That case, however, is at present unique; and inflammation of the pericardium is after all present in very few cases of chorea.

‡ The only plausible arguments against it are forcibly put by Dr Sturges ('Chorea and Whooping-cough,' p. 16).

Statistics are as follows:—Among the patients of Guy's Hospital, (*a*) in the late Dr Hughes's first series ('Guy's Hosp. Reports,' 1846), special inquiries were made in fifty-eight cases; there was a cardiac murmur in nine, and a history of rheumatism without bruit in eight more; (*b*) in his second series, compiled by Mr Burton Brown (ibid., 1855*), special inquiries were made in 104 cases, and in eighty-nine of these there was either bruit or rheumatic history; (*c*) in the series of 150 compiled by Mr Manser, and published in 1874 (ibid., Third Series, vol. xix), forty-two had suffered from rheumatism, and in three of these rheumatic fever supervened while they were under treatment for chorea; there was a bruit in fifty-nine, none in eighty; (*d*) in the fourth series, of 163 cases compiled by Mr Halstead, fifty-three had had rheumatic fever or distinct pains in the limbs, believed to be rheumatic, and in thirty-five of them the rheumatism preceded the chorea by less than six months; there was a bruit in sixty-one, none in 111.

Of Prof. Sée's 128 cases, sixty-one had probably had rheumatism.

Of Dr Sturges' 100 cases, only twenty had had rheumatism.

In 104 consecutive cases of chorea at the Westminster Hospital Dr Donkin found twenty-seven cases of previous rheumatism.

Of Dr Goodhart's 130 cases (collected from several sources) eighty-nine were on positive evidence believed to have had rheumatism.

Of Dr Angel Money's 214 cases (also collected) thirty-three had had rheumatic fever, twenty-three had had rheumatism, and nine were doubtful.

In 172 cases of chorea at the London Hospital Dr Stephen Mackenzie found a history of distinct rheumatism in forty-seven ('Trans. Int. Congr.,' 1881, vol. iv, p. 97).

The Collective Investigation gave 116 instances of precedent rheumatism (excluding mere "rheumatic" pains) in 439 cases of chorea.†

Two children in the same family often have chorea, but we rarely find that the parents of a choreic child have themselves suffered from the malady when young. This is the same kind of "transverse" hereditary relation which we observed to obtain in certain other maladies,—Friedreich's ataxia (p. 539), Thomsen's disease (712), and some forms of muscular atrophy.

Embolic theory.—Attempts have been made to explain the connection between chorea, rheumatism, and cardiac disease upon a theory which, although it has had the support of some distinguished observers, does not appear to be well founded. Its author was the late Dr Kirkes. Starting from the frequent association of endocarditis with chorea, he suggested that the cause of the spasmodic movements might be the introduction of inflammatory products and fibrinous particles from the diseased valves into the blood, and the consequent disturbance of the parts supplied. Dr Hughlings Jackson and Dr Broadbent have expressed a similar view in a more definite form, maintaining that the cause of chorea is embolism of minute arteries in one or both of the corpora striata.

A few observations have since been made by different pathologists which have been thought to corroborate Dr Jackson's theory. In a very severe case, attended with maniacal symptoms, Dr Tuckwell ('Med.-Chir. Rev.,' 1867) found at the under and outer aspect of the right hemisphere a large red patch of softening, affecting to some extent the white as well as the

* The first volume of the Third Series was published in 1855, not 1856, as stated by von Ziemssen, Bd. xii B, S. 443.

† See also statistical papers by Dr Herringham and Dr Archibald Garrod in the 'Medico-Chir. Trans.,' vol. lxxii, pp. 117, 145.

grey matter; and a branch of artery which ran straight into it contained a small, white, tough, fibrinous concretion, tightly wedged into an angle of bifurcation, and connected on all sides with long black coagula, extending into the trunk of the vessel and its branches. On the outer aspect of the same hemisphere was a similar patch, but smaller, and limited to the superficial cineritious substance of the convolutions; in this nothing like an embolus was discovered, but the parts had been cut through in various directions before the dissection of the vessels was commenced. In another case of very acute chorea, which terminated fatally by hæmorrhage into the brain, Dr E. L. Fox ('Med. Times and Gaz.,' 1870) believed that he detected microscopic emboli in the vessels of the corpus striatum.

Dr Angel Money has endeavoured to produce chorea in monkeys and cats by injection of starch-granules or insoluble salts into the cerebral arteries, and the results will be found in the 'Medico-Chirurgical Transactions' for 1885; see also 'British Medical Journal,' July 17th, 1886.

But is it possible to determine whether clots in minute cerebral arteries are of *ante-mortem* formation, still more whether they entered the vessels from below, instead of being formed *in situ*? Moreover, the minute, firmly adherent vegetations which are found on the valves in chorea, do not seem at all likely to be detached and carried away by the blood-stream. If they were so liable to be washed off, what could prevent some of them from being carried into the spleen and kidneys, and producing infarctions there? but such appearances have never been discovered in any of our fatal cases of chorea. Again, as Dr Bristowe remarks, it is difficult to understand the limitation of chorea to the limbs of one side on the embolic theory, since a shower of minute emboli must be supposed to enter the arterioles of one corpus striatum only. We are familiar, however, with endocarditis in which vegetations are carried from the diseased valves, and cause embolism in the brain and in other parts; and in these cases chorea never develops itself.

But the strongest argument of all is the above-mentioned relation of chorea to *mental shocks*. Some writers have supposed that the cases in which the disease follows a fright are distinct from those in which it is associated with endocarditis; but this is certainly not the fact. On the contrary, as already stated, vegetations are found on the cardiac valves in all fatal cases, almost without exception; for instance, a child died under Dr Wilks's care who was attacked by chorea after being terrified by the gunpowder explosion at Erith, and *post mortem* the mitral valve was found inflamed. Now, it is obvious that the disease cannot possibly have two different exciting causes in a single case. It cannot be at one and the same time the result of a mental shock and of embolism of several minute cerebral arteries. In such cases we must admit that the endocarditis is an effect of the chorea.

Still it is not to be denied that any view of chorea presents difficulties. It is strange that a disease so closely related to acute rheumatism should be excited by a nervous shock; it is still more strange if, when so produced, it should possess a power like rheumatism itself of setting up inflammation of the cardiac valves. No more striking instance could be found of the complicated working of different predisposing and exciting causes, which concur in the ætiology of the neuroses, and of many other diseases.

Treatment.—This is a very difficult question; for chorea offers peculiar obstacles to the satisfactory investigation of the action of remedies. Many of the severer cases tend naturally to a more than usually rapid recovery;

and in other instances (as Dr Wilks has proved) the being admitted into the ward of a hospital and kept in bed is of itself sufficient to bring the complaint quickly to a termination. Under either alternative, the medicine which may have been prescribed is apt to get undue credit. Lastly, in the great majority of cases, *chorea subsides of itself after eight to twelve weeks*, under whatever treatment. Now, the method usually adopted is to give one drug for three or four weeks; if that fails, to change it for another, which is continued for about an equal period of time; and, if there is still no result, to begin the administration of a third. The result necessarily is that even if all these medicines are really equally inert they gain very different degrees of credit. The uselessness of the one which is first given is sure to be apparent; but the second runs a chance of apparent success; while the last one is almost certain to acquire the reputation of having cured a case in which its competitors had altogether failed. Yet that very drug, if placed first on the list for a succeeding case, would perhaps show itself as impotent as the others. Probably much of the uncertainty as to the value of remedies in chorea is explicable in this way.

One method of avoiding these difficulties would be to treat a considerable number of cases with some one medicine throughout the whole course of the disease, and then to compare the result with the series of cases related by Drs Gray and Tuckwell, which were allowed to terminate without interference. These observers administered arsenic to fifteen choreic patients; and they state that the average duration of these cases was almost the same as if no medicine had been given. The very volume of the 'Lancet' (1871) in which their investigations are recorded contains reports of twelve other cases by Mr Butlin, treated by Drs West and Dickinson with sulphate of zinc. But it is impossible to compare their results with those of the Oxford physicians. Two of the cases had lasted for so great a length of time before they came under observation (thirteen months and four years respectively) that their introduction would swamp the whole series. Yet these two only present in an extreme form difficulties which belong in a less degree to all the other cases, every one of which had lasted for several days, and many of them for some weeks, before the administration of the medicine was commenced. It is not easy to see how one could avoid this source of fallacy, except by confining one's observations to the children in one particular school, where a uniform treatment could be employed from the very beginning of the disease. So that it is almost impossible to obtain statistical proof of the value of medicines in the treatment of chorea.

But there is very strong evidence, of another kind, in favour of at least one remedy. Very protracted cases, which had resisted all other methods of treatment, have sometimes been found to yield in a very short space of time to *arsenic*. Some striking examples of this are recorded by Romberg.

One is that of a girl, aged eleven, who had for eight years suffered from intense chorea, affecting especially the right half of her body. All drugs had been found useless until she began to take Fowler's solution; in about two months there was a marked improvement, and at the end of two months more she had entirely recovered. Another patient, a girl aged ten, had had the disease two years; arsenic was prescribed, and within three weeks the symptoms presented a marked abatement; and ten or eleven weeks afterwards she was discharged cured. A third instance is that of a girl, eight years old, who for six months had been the victim of chorea to such an extent

that she could not walk, nor stand, nor speak articulately. The remedies which had been tried had failed; Fowler's solution, in doses of four drops three times a day, established a cure in eight weeks. So far as appears, the only objection that can be offered to these cases is that it is not distinctly stated whether or not the patients, while the medicine was being given remained otherwise under the same conditions as before.

Another argument in favour of the effect of arsenic and other drugs upon chorea is derived from our experience among out-patients. There the children have none of the great advantages of quiet, careful feeding and nursing which cure many cases of chorea when admitted to a hospital ward. No change in their condition is made, except the administration of a drug; and when under this treatment a complaint which, in some cases, has lasted for weeks disappears, we can scarcely help attributing the cure to the remedy.

Most observers are of opinion that others of the so-called nervine tonics are also useful. Thus the *sulphate of zinc* has for many years been largely used at Guy's Hospital; one-grain doses of it used to be given at first, which were gradually increased until the patient took a scruple or more. It seems more advisable to begin with a larger dose—for instance, with five grains. This seldom causes nausea or sickness more than once or twice. Another drug which once had a great reputation is the *carbonate of iron*.

Salicyl compounds have been lately used, and, in some cases, with seeming success ('Brit. Med. Journ.,' 1887, vol. i, p. 436).

Static electricity, used in the old-fashioned way, was formerly much used at Guy's Hospital by Addison, Golding-Bird, Gull, Hughes, Babington, and Lever ('Guy's Hosp. Reports,' Series I, vols. ii, vi, and vii; Series II, vol. viii), but it has long been given up. Galvanic and faradic electricity have likewise been tried, praised, and abandoned.

There is reason to believe the milder cases of chorea may to some extent be checked by gymnastic exercises, by military drill, or by the use of a skipping-rope. French physicians have laid stress upon this method of treatment, and it has had undoubted successes.

On the other hand, one sometimes has to deal with cases which are so severe that the administration of nervine tonics, requiring time for their operation, is obviously inapplicable, because the patient is in imminent danger of dying within two or three days. There is the greatest difficulty in determining the value of remedies in cases of this kind. Probably death is sometimes inevitable. But even then the inhalation of *chloroform* gives great relief to the patient's sufferings; and if there be a chance of recovery it may do something towards economising his strength. It also saves him from the sores which would form over the bony prominences if the movements were uncontrolled. In cases which are a little less severe, *chloral* appears to be the best medicine. Several writers have related cases in which it seemed to be effectual; not the least striking is one of Dr Gairdner's, of a girl who took a drachm of it by mistake, and was poisoned, but who on her recovery was found to be cured. Again, the muscular depressants—conium and the Calabar bean—have sometimes been employed. At one time the author prescribed the succus conii in considerable doses for several choreic patients, and some of these recovered more quickly than was expected.

A point of great importance in very severe cases is that the patient should be kept well supplied with food; nutrient enemata ought to be administered frequently; and probably it is right to give full doses of alcohol.

CONGENITAL REFLEX SPASMS.*—We have at the present time (April, 1890) lying in Philip Ward a boy about eight years old who "makes faces" much as a child with chorea does; but on pulling down the bedclothes it is plain that the case is not one of ordinary chorea, for the movements of the limbs are slower, and the spasms are tonic as well as clonic. The arms are extended and the hands go through slow twisting movements, not choreiform, but exactly what we have before described under the term "athetosis" (*supra*, p. 701). The legs are thrust out and very stiff, so that the trunk can be lifted on raising them: the adductors and extensors are in tonic spasm. The feet do not show the characteristic attitude of tetany. The spasms are much increased when the visit to the ward brings a number of strange faces round the bed, but they do not disappear when he is quite undisturbed, and even in sleep the child lies always on one side, with his legs and arms thrust out in front of him to their full length. Handling the muscles increases the rhythmic as well as the spastic contractions, and if they are continued, the child sometimes cries, but it does not appear that he suffers much pain even then. He can put out his tongue; there is no squinting or nystagmus; the functions of the bladder and rectum are normal. He is well-grown, rather slender in build, but not ill-nourished; and there is no deformity of the head, no curvature of the spine, and no club-foot. A normal knee-jerk can be detected with some difficulty: there is no clonus. He can talk, but almost unintelligibly; and though he understands what is said, he is obviously behind his years in intelligence. He is docile and good-tempered, but timid. On further inquiry we learn that this condition is congenital, that he "could not move at all" as a baby, and could not talk till he was much older than usual, and that at one period he was "subject to fits." Lastly, Dr Todd, of Brigg, Lincoln, who brought him into the world, informs us that the labour was unusually protracted, and that there was a well-marked caput succedaneum when he at last was born.

The clinical features of this case, while agreeing with those of a reflex spasmodic neurosis, are those which have been described as congenital choreiform spasms; but the presence of a definite anatomical lesion is rendered almost certain by the absence of family predisposition, and the history of the patient's birth.

Such cases were observed by Dr Sarah MacNutt, an American physician in 1885, as occurring in obstetric practice and due to meningeal hæmorrhage. Many prove fatal in infancy, but in others the hæmorrhage is followed by circumscribed atrophy of the cortex, either from pressure of the clot, or perhaps more likely from disturbance of the nutrition of the convolutions. Why such atrophy produces spasm instead of paralysis, why it is a "discharging" instead of a "destroying" lesion, is hard to say. It almost seems as if there were abolition of inhibitory motor centres in the cortex, which allows co-ordinated reflex movements to take place like those of a decapitated frog or pigeon. See Dr Osler's 'Cerebral Palsies of Children.'

The spasms are not always so general in these cases; they may affect one side, or both legs, or one limb only. In some cases clonic spasms predominate, bringing them nearer to "hammer-spasms" and "histrionic spasms;" in others the resemblance to primary spastic paraplegia is very close; in

* *Synonyms.*—Congenital chorea—Cerebral birth-palsy—Birth-spasms—Post-hemiplegic chorea, with ataxia or athetosis—Maladie de Little—Effects of meningeal hæmorrhage at birth—Spastic rigidity of the new-born (Little), see 'Obstet. Trans.,' 1862—Spastic diplegia.

others again, that to chorea. The long duration of the malady, the more or less deficiency of speech and intellect (not more, however, than in certain cases of chorea while they last), and the history of the origin at birth are the diagnostic characters to dwell on.

The prognosis is unfavourable, but some of the patients gradually acquire increased power of grasping objects, of talking, and even of locomotion.

No treatment is promising. We have tried physostigma, bromides, and chloral hydrate without much benefit. Dr Gowers, who has observed more than thirty cases of this curious malady, says that electricity however applied is useless.

Hereditary chorea in adults—Huntingdon's chorea.—In exceptional cases chorea appears to be hereditary in certain families, and passing over the earlier years of life to make its appearance at about the age of thirty. The clonic spasms begin in the face, and gradually spread to the arms and the legs, continuing for several years. The gait acquires a peculiar hasty and hesitating character, and the speech becomes intoned. Not only is the malady long continued, but it has usually ended in mental decay, and at last in death. This variety of spasmodic neurosis is obviously different from true chorea in its course as well as in its pathological relations. Huntingdon's cases were observed in Long Island, New York. Others have been described in this country, and Huber has recorded a remarkable case which occurred in Eichhorst's practice at Zürich.

*Friedreich's spasms.**—Under this name a spasmodic disease in an adult patient was described by Friedreich in 'Virchow's Archiv' (vol. lxxxvi), and since by Hammond. Gowers considers that the malady is perhaps intermediate between chorea and facial spasm or torticollis, and more nearly allied to senile chorea than to any other malady.

Dr Weir Mitchell's "*habit-chorea*" applies to those involuntary tricks, which are truly spasmodic neuroses. They begin in childhood, but often persist in adult life. Blinking with the eyelids, tapping with the foot, suddenly twisting the head or jerking the hand, hiccough, choking or snorting at intervals, sniffing lecturers and smiling preachers—all these are examples.

Dubini's disease.†—This name has been given to an obscure malady consisting of choreiform spasms which has been observed in Italy, and is supposed to depend upon malarial influence. The suddenness of the spasmodic shocks, the fact that most of the patients are of advanced age, and that some cases are fatal, separate it from true chorea. There are, moreover, tonic paroxysms and epileptiform fits. It was first described by the late Dr Dubini, of Milan, about 1850.

* *Synonyms.*—Paramyoclonus multiplex—Myoclonus multiplex—probably identical with the Convulsive tremors described by Pritchard in 1822.

† *Synonyms.*—Electrical chorea—Typhus convulsivo-cerebralis—Myelitis convulsiva.

THE PAROXYSMAL NEUROSES

Quin etiam, subito, vi morbi sæpe coactus,
Ante oculos aliquis nostros, ut fulminis ictu,
Concidit et spumas agit; ingemit et tremit artus,
Desipit, extentat nervos, torquetur, anhelat
Inconstanter, et in jactando membra fatigat.

LUCRETIUS.

Introductory remarks—Relation to other neuroses and to each other.
EPILEPSY—*Definition—E. minor—E. major: aura: fit: sequelæ—Pathology, seat, and causation—Diagnosis—Treatment of the fit and of the intervals.*
Other forms of eclampsia—Puerperal, uræmic, &c.
Paroxysmal vertigo—Auditory vertigo and Menière's disease—Somnambulism—Catalepsy—Night terrors.
MIGRAINE—*Nomenclature—Symptoms—Pathology and causation—Treatment.*

WE have now to consider a remarkable series of affections, which, although they differ widely in their symptoms, nevertheless have close mutual relations, and present many points of resemblance, both in their causes and in their circumstances.

The chief among them are Epilepsy (including both the *petit mal* and the *haut mal*), Catalepsy, Somnambulism, Migraine, and Paroxysmal Vertigo. With these several others might be included:—Tic douloureux, paroxysmal Insanity, Angina pectoris, paroxysmal Gastralgia, spasmodic Asthma, spasmodic Croup, and Laryngismus stridulus. For reasons of convenience, however, these are treated elsewhere.

No modern writer has so clearly pointed out the relations of these various diseases to one another as Dr Edward Liveing, who, in his treatise on 'Megrim or Sick Headache,' shows how in the same patient they are often transformed one into another in the course of time. Most other writers have dealt with them from too narrow a standpoint; they have included too much under a single affection, as epilepsy.

Common features of the group.—The disorders in question all occur in paroxysms, and at more or less regular periods. Most of the patients are in perfect health during the intervals. We may distinguish them as "paroxysmal neuroses;" or, adopting the happy phrase of Dr Liveing, as "nerve-storms."

These diseases for the most part are *innate* and *hereditary;* and in different members of the same family the inherited tendency may show itself in different ways—one child being epileptic, another asthmatic, a third subject to megrim, and so on.

In more than one of the affections now to be described, each attack is made up of a regular *succession* of phenomena. In migraine it often begins with a peculiar affection of sight; afterwards follow in turn numbness of the fingers, vertigo, headache, vomiting, and sleep. In epilepsy there is perhaps first a sensation passing up from one of the limbs to the head, and then successively complete insensibility, a general tonic spasm, a series of clonic

convulsions, and prolonged stupor. We can hardly fail to regard such seizures as dependent upon the gradual extension of some molecular change from one part to another of the nervous centres.

Other points in which most of these affections agree is that the attacks gradually *culminate* in a certain pitch of intensity, and then subside; and again that there is a kind of *compensation* between the frequency and severity of the paroxysms, for a slight one is followed by another at a short interval, while a severe seizure may ensure a long period of repose.

The paroxysms are often traceable to *causes* similar for all the members of this group. In describing the different varieties of "nerve-storm," one has again and again to mention gastric disturbance, irritation of the brain from dentition or disease of the teeth, exhaustion from deficient food or excessive bodily exercise, disorders of the sight, smell or hearing, as giving rise to a first attack, or to the succeeding ones also. Many of these neuroses bear a relation to puberty and the other great epochs of life, to the appearance of the catamenia in women, to pregnancy or to the puerperal state. Most of them are apt to break out for the first time at some particular age—migraine and epilepsy about puberty, laryngismus stridulus and spasmodic croup during infancy, and tic douloureux in later life; while, as we have seen, tetany and chorea are diseases of childhood. Some of the paroxysmal neuroses tend to disappear spontaneously when a certain period of life is reached; thus migraine often ceases to recur after fifty.

Another feature which is common to the paroxysmal neuroses is their tendency to undergo *metamorphosis* in the same patient as time goes on. This is particularly the case with epilepsy; its attacks are liable to be replaced by vertigo, catalepsy, mania, or other forms of nervous disturbance. Indeed, if we had convenient English names for the *petit mal* and the *haut mal*, it would be well that they should be described separately, for their relation to one another is not very much closer than to some of the other affections treated in this chapter. The connection between migraine and epilepsy is certainly much less intimate.

It would, however, be incorrect to represent the paroxysmal neuroses as an isolated group, for there are unmistakable evidences of relation between Epilepsy on the one hand, and Chorea, Hysteria, and Insanity on the other. But, on the whole, Megrim, Vertigo, and Epilepsy may provisionally be grouped together.

EPILEPSY.*—*Definition.*—Convulsions which closely resemble the fits of epilepsy may accompany the onset of variola and other exanthemata in children; they occur shortly before death by hæmorrhage; they result from Bright's disease and from the puerperal state; and we have seen that they are caused by various organic affections of the brain.

But, in remarkable contrast with the various cases just referred to—in all of which the fits are accidental, occasional, or solitary,—there are other cases in which convulsive fits, of precisely the same kind, return again and again for years together, and often without being traceable to any cause, except in some instances an inherited tendency to nervous disorder. One cannot but regard such cases as essentially distinct from the others, and

* *Synonyms.*—Ἐπιλημψία or Ἐπίληψις, a seizure (Hippocrates, Aristotle)—Morbus Comitialis (Seneca and Pliny), so called, according to the grammarian Festus, because the Roman assemblies were broken up if anyone present was attacked by a fit—Morbus sacer (Cœlius Aurelianus).—*Anglice*, Falling Sickness.—*Fr.* Haut Mal.—*Germ.* Fallsucht.

needing a special name; we therefore speak of them as *epileptic*, and call fits which are accidental, secondary, or symptomatic, *epileptiform*; or we may conveniently apply the term "eclampsia" to the latter generally, instead of confining it to the convulsions which are apt to occur after childbirth.

In limiting the use of the word Epilepsy to an idiopathic and recurrent paroxysmal neurosis, one must not imagine an underlying essential disease of which the fits are only symptoms. The fits themselves constitute the disease, the only other element in its definition being the clinical fact that they tend to return at more or less regular intervals for an indefinite length of time. The distinction between *epileptic* and *epileptiform* attacks is not always easily applied. Some cases of very long standing should nevertheless be classed with symptomatic eclampsia rather than with true epilepsy. On the other hand, a single fit, which is never repeated, may belong to epilepsy in the restricted sense of the term, although the tendency to recurrence is overcome by treatment, or is never called into activity.

Epileptic attacks vary greatly in severity. In accordance with the universal practice, we may divide them into two principal groups, the one corresponding to what the French call the *petit mal*, the other to what they call the *haut mal*. These terms have been Latinised as *Epilepsia minor* and *Epilepsia major*, and it would be convenient to adopt similar expressions in English. The *petit mal* is sometimes spoken of as *epileptic vertigo*, but for more than one reason this use of the term is undesirable.

Epilepsia minor.—A paroxysm of the *petit mal*, or minor epilepsy, may be a mere suspension of consciousness, sudden in its onset, and scarcely more than momentary in its duration. For two or three seconds the patient is lost, but he quickly recovers himself, and goes on with what he is doing. Dr Wilks mentions the case of a shopman who often had a seizure of this kind while serving a customer, and who believed that no one noticed it. In some instances such attacks are attended with muscular rigidity. Dr Chambers relates that a well-known lecturer would sometimes be seized while addressing his class, and would stop in the middle of a sentence, remaining perfectly still, with mouth open and arms extended; after a minute or two he would go on just where he left off without knowing that anything had happened. Dr Chambers, indeed, puts this case as one in which the *petit mal* assumed the characters of *catalepsy*, but it does not seem clear that it really deserved that name. At any rate its close relation to epilepsy was shown by the circumstance that this patient afterwards suffered from that disease in its major form.

In other cases, if the patient should happen to be speaking at the time when he is attacked, he loses the thread of his discourse, and afterwards he cannot remember what he was saying. Or he may stagger and lean against something, or grasp at it for support. He may experience a sensation of giddiness and lie down to avoid falling. Dr Wilks mentions one instance in which a little girl would be sitting in her chair stitching, when she would suddenly fall, but before the nurse could pick her up she would be in her seat and again at work. To such cases the name of "epileptic vertigo" would be applicable if there were not another *paroxysmal vertigo*, which does not bear so close a relation to epilepsy (*v. infra*, p. 767). Moreover, some writers call any nervous affection epileptic which recurs in paroxysms.

If we have the opportunity of watching an attack of minor epilepsy we may notice that the patient's pupils become slightly dilated, and that he no

longer seems to be looking at anything. The face often becomes pale, and afterwards slightly flushed. Dr Reynolds says that the pulse may falter and become irregular, and Dr Moxon has observed the same thing; but it is quite the exception.*

In a large majority of cases the so-called fainting fits which are apt to occur in children are really of an epileptic nature.

It is, however, certain that in some instances the circulation in the face and limbs goes on without interruption during an attack of the *petit mal*. Dr Reynolds testifies to this from repeated observations.

Not infrequently an attack, which is in all other respects one of "minor epilepsy," is accompanied by some slight convulsive movement. There is a transient strabismus; the mouth is drawn to one side; the whole head is turned towards one shoulder; or the body generally becomes for an instant rigid. Cases of this kind form links between the two main varieties of the disease, and show how closely they are related to one another. A further proof is the fact that in perhaps the majority of cases in which attacks of the *petit mal* occur again and again, they are after a time replaced by those of the *haut mal*. Or the patient may suffer alternately from the one and from the other.

Prodroma.—Before describing the phenomena which constitute a regular attack of the *haut mal* we must mention certain sensations which some patients experience at the commencement, and which may precede the other symptoms, so as to afford a warning of the approach of the seizure. To all such phenomena the name of *epileptic aura* is commonly given by a convenient extension of its original meaning; it was first applied to a feeling as of a draught of air passing over the surface until it reached the head, whereupon the patient became insensible. Sometimes a creeping or a dragging sensation is experienced, which begins in the fingers of one hand or the toes of one foot, and rapidly moves upwards. Dr Bazire relates a case in which it first affected the wrist, and then extended downwards to the tips of the fingers. Dr Radcliffe had a patient in whom it was a painful sensation, always referred exactly to the foramen cæcum at the base of the tongue. Other cases have been recorded in which an aura has consisted of a painful sensation at the epigastrium, attended with nausea. Or, again, there may be loss of sensation in one side of the face or in one limb. Nothnagel mentions an instance in which numbness always began in the right shoulder and side of the head, and spread downwards into the right arm, the right leg, and the right half of the body. When an aura passes up a limb its path seldom corresponds with the course of any particular nerve. In some rare cases it seems to start from the seat of a former injury. Sir Thomas Watson quotes a case in which it proceeded from an old cicatrix in the side. Dr Wilks states that a girl under his care localised her aura in a sore spot on her face, and that her father assured him that the application of laudanum to that part would sometimes arrest the fit.

* Some years ago, while I was one day listening with the stethoscope to the heart of a man whom I had never seen before, its beats suddenly ceased. I looked up and saw that his face had turned deadly pale. He said that he was going to faint, and reclined back on the chair from which he had risen. I could feel no pulse at his wrist, and for an instant I thought that he might be going to die, but I had hardly time to ring my bell when the colour returned to his face, and I found that his heart was beating again. Presently there was a little twitching of the muscles in one or both hands. In a minute or two he was able to tell me that he often had "fainting fits," and that some years back he had been subject to epileptic seizures. It appeared clear that the attack which I had witnessed was one of minor epilepsy.—C. H. F.

In other instances it would seem that an epileptic aura is due to an affection of the vaso-motor nerves of some part of the body. The patient perhaps experiences a sensation of coldness or weight in a limb; and the part is found on examination to be pale and cold to the touch, and to have its sensibility distinctly blunted. Trousseau says that when an aura occurs in a finger it is sometimes a little swollen, so that the rings on it which before were loose suddenly become tight.

In other cases, again, an epileptic attack is preceded by a profuse secretion of tears, or of saliva, or of sweat, as in several cases that have come under Nothnagel's observation.

What has been termed a "motor aura" may take the form of tremor or of slight spasms. The eyelids may twitch, or some muscles of the face or of a limb; or more complex movements may take place, the patient turning round, or running some distance.

Again, the epileptic aura may be referred to one of the special senses. Joseph Frank is said to have met with an instance in which it always took the form of a sweet taste. Still odder is Dr Gregory's case, quoted by Watson, of a man who always fancied he saw a little old woman in a red cloak; she seemed to come up to him and to strike him a blow on the head, whereupon he lost all recollection and fell down. Sometimes the only warning is a vague sensation of fear. Dr Reynolds was told by a gentleman that what always passed through his mind was, "This is what I had foreseen. I knew it would come on here; I ought to have avoided it by remaining away,"—although in reality he had not suspected that a fit was impending.

An epileptic aura may last for a few seconds or for several minutes, or even longer. Nothnagel mentions that when it has lasted more than ten minutes, he has been able to ascertain that there was distinct loss of sensation in the part affected. Sometimes it occurs and passes away without being followed by a fit. In one of Nothnagel's patients a vaso-motor aura in one leg sometimes occurred six times daily, whereas she had only about one epileptic attack in a week.

*Symptoms of the fit.**—An attack of the *haut mal* begins by the patient suddenly—almost instantaneously—falling unconscious, with strained and rigid muscles. As he is seized, he may utter a sharp shrill cry; or he may be perfectly silent, the respiratory movements being arrested by spasm; or he may emit a smothered groaning sound. In many cases the face becomes pale; but in some its colour remains unchanged. The heart goes on beating, and the carotid arteries continue to throb. But the radial pulse is sometimes imperceptible, this being probably due to the contracted state of the muscles of the arms.

The *tonic spasm* which occurs at this period of an epileptic or other kind of fit is peculiar; it usually affects one side of the body more powerfully than the other; the head is turned round towards one shoulder, and the eyeballs are strained in the same direction. The pupils constantly become dilated; but Dr Reynolds says that in one instance he observed a momentary contraction before dilatation began. They are generally, if not always, insensible to light. Touching the conjunctiva commonly excites no reflex movements, but according to Romberg it is sometimes followed by closure

* "Inter notissimos morbos est etiam is qui *comitialis* vel *major* nominatur. Homo subitò concidit: ex ore spumæ moventur: deinde interposito tempore ad se redit, et per se ipse consurgit. . . . . Modò cum distentione nervorum [*i. e.* convulsions] prolabitur aliquis, modò sine illâ" (Celsus, 'De Med.,' lib. iii, cap. xxiii).

of the lids; this observer also states that sprinkling the surface of the body with cold water may in some cases cause shrinking movements. Soon the face flushes, and acquires a dull red or dusky hue. This appears to be in great measure due to the fact that the large veins of the neck undergo compression, and that the flow of blood through them is interrupted by the spasmodic contraction of the sterno-mastoideus and neighbouring muscles. Spasm of the glottis seems also to occur.

The stage of tonic spasm may last only two or three seconds, or it may be prolonged to thirty or forty seconds. Trousseau says that in some rare cases it extends over two or three minutes—in fact, until the patient dies asphyxiated. It is followed by a stage of *clonic spasms*. These also are generally more marked on one side than the other. The fingers of one hand are alternately flexed and extended; the like movements simultaneously occur in the other joints of the arm, and even in the corresponding lower limb, and the eyes are twitched violently towards the affected side. The opposite limbs may escape entirely, or be affected with less powerful convulsive movements. The pupils sometimes oscillate between a state of contraction and one of dilatation. The jaws are forcibly contracted, and the tongue is often caught between the teeth and bitten on one side. The blood from it is mixed with the saliva that is poured abundantly into the mouth; and a red foam is sputtered through the clenched teeth. The face is now of a livid purple hue. There is often profuse sweating, sometimes urine is passed involuntarily, and emission of semen or defæcation may occur.

This second stage—that of clonic spasm—may last two or three minutes; according to Dr Reynolds, even ten minutes. Trousseau, however, says that it scarcely ever continues more than four or six minutes: as he remarks, one is very apt to be deceived as to its duration unless one reckons the time by a watch. As the convulsive movements pass off they become slightly altered in character. They are no longer wholly meaningless. The patient often draws a deep sigh; he may endeavour to change his position, and may look at those about him with a bewildered or suspicious expression.

When the fit passes off, the patient may at once regain his consciousness; or he may be more or less confused for a time; or, more frequently, he becomes drowsy and passes into a deep sleep or stupor, which may last several hours, and is often attended with guttural stertor. During this time, if he can be roused at all, he is generally irritable and peevish. Slight clonic spasms not infrequently recur.

Even when an epileptic fit occurs during sleep, one can often make out its real nature by the fact that the tongue next morning is found to be sore, or that the urine has been passed involuntarily, or even the fæces. As Trousseau points out, if a patient who had never before had difficulty in retaining the contents of his bladder at night, should now and again find that he has wetted his bed, this mere fact should arouse the fear that he may be an epileptic. Dislocation of the shoulder, discovered on waking in the morning, may signify an epileptic fit during sleep. A similar inference may sometimes be drawn from the presence of minute scattered red petechial spots, like fleabites, which occur chiefly on the forehead, throat, and chest. When these petechial hæmorrhages occur after an epileptic attack in a person who has not before been subject to fits, they may be wrongly regarded as evidence that the patient is suffering from blood-poisoning, or that an exanthem is about to appear. Dr Wilks has recorded a case in which this mistake was made.

Exceptional forms.—An attack of major epilepsy does not always conform strictly to the above description. It is sometimes attended with but slight convulsive movements, or perhaps with none at all. Such cases were formerly described as examples of a form of apoplexy, or as "apoplectiform cerebral congestion" (cf. *supra*, p. 597).

We have seen (p. 612) that tumours and other local organic diseases of the brain frequently give rise to fits which are unattended by loss of consciousness, and consist mainly of convulsive movements, perhaps limited to certain parts. These have been distinguished as Jacksonian convulsions, after Dr Hughlings Jackson (pp. 613, 616). It might be expected that similar fits would sometimes occur in genuine epilepsy. All observers, however, are agreed that this is exceedingly rare. Two instances are recorded, one by Trousseau, the other by Nothnagel. The former occurred in a young man aged eighteen, who was liable to convulsions of the facial muscles, affecting only the left side, and not accompanied by loss of consciousness nor by any other epileptic phenomena. But the clinical history was that the disease had first set in six years previously with violent fits of the *haut mal*, and that these had gradually become milder and passed into those of the *petit mal*, which presumably occurred alternately with the attacks of mere partial clonic spasm. Nothnagel's case was in a boy of sixteen. Dr Reynolds, who gives to this form of the disease the appropriate name of "abortive epilepsy," furnishes a list of references to various writers as having described it. But after looking up most of them we are not sure that these writers took care to exclude cases of cerebral tumour and the like. However this may be, exceptions are very rare to the rule, that whenever attacks of clonic spasm recur paroxysmally without loss of consciousness there is local organic disease of the brain—the disease is symptomatic eclampsia, not idiopathic epilepsy.

Recurrence.—The frequency with which the attacks of epilepsy return varies greatly in different cases. A patient may have one fit without ever having a second; or after an interval of two or three years he may be again attacked. In some cases the paroxysms return once, or twice, or three times a year; in other cases more or less regularly once a month. In women they are, on the whole, more apt to occur at the catamenial periods than at other times. But Dr Reynolds says that monthly recurrence is actually more common in the male than in the female sex; and it is very rare indeed for a woman to be liable to epileptic fits only during menstruation. More frequently the interval at which the attacks recur is less than a month. Lastly, there may be one or more paroxysms every day. It is common for two, three, or more fits to occur on the same day or within two or three days, and then for the patient to be free from them for several weeks. They are then said to recur in *series*. When they return with great frequency during a lengthened period, they almost always belong to the minor form of the disease.

There is, however, a special modification of the disease in which the fits follow one another in rapid succession, so that before the patient has recovered completely from one of them another comes upon him. This has been called by French physicians *état de mal épileptique*, and in England *status epilepticus*. When it is at its height the convulsions follow one another with extraordinary rapidity. Sir Crichton Browne in describing such cases says that the limbs are scarcely laid to rest after one fit before they are tossed and contorted by another, and even in the intervals there are frequent

muscular twitchings. The patient lies perfectly unconscious; his heart beats rapidly and tumultuously; his respiration may either be quick and shallow or slow and laboured. His features are swollen and livid, and his lips purple. His body is bathed in profuse perspiration, and the temperature is raised to 105°, or even higher still. Dr Merson found it in one case 107·8°. This condition often terminates fatally; indeed, it is one of the chief ways in which epileptic patients die. Charcot observes that in such cases bedsores over the sacrum form rapidly. He also mentions that a temperature of 105·8° is sometimes reached without death ensuing.

Complications and sequelæ.—Certain occasional results of an epileptic fit have yet to be described. One is a loss of muscular power in one arm, or in both the arm and leg on one side, generally that side which was the more convulsed. Dr Todd described this under the name of "epileptic hemiplegia;" it may either pass off in a few hours or last several days. One must not forget that diminished mobility of the arm after a fit may be due to a very different cause, namely, to dislocation of the shoulder, produced sometimes by muscular spasm, sometimes by the patient having struck that part in falling.*

At one time a woman who was liable to epileptic fits used frequently to come to the hospital to have her shoulder set. In her case the fact that the same dislocation occurred again and again showed that spasmodic contraction of the muscles was its cause. Still more serious injuries sometimes arise during a paroxysm. The skull may be fractured by the patient dropping down on the pavement; or he may be severely burnt by falling against the bars of the grate; or he may be run over in the street. Persons who are liable to epilepsy cannot be too closely looked after.

Delirium has not hitherto been mentioned as accompanying epileptic fits, but in some cases it is one of the most important features. Indeed, in a person subject to the disease, the attack may be wholly replaced by one of paroxysmal mania (p. 773). Again, the epileptic character may be so far maintained that the patient falls down, but quickly gets up again and attacks those about him with the utmost violence. An instance of this is recorded by Trousseau, who remarks that if there had been no witnesses of the commencement of the attack, the patient would have been liable to a criminal prosecution, since he not only assaulted the passers by in the street, but spat at the soldiers who held him. In other cases the epileptic stupor is succeeded by maniacal delirium, during which suicide may be committed, or murder. Still more frequently a fit is followed by loss of memory, incoherence of ideas, and perversion of intellect, which may last several days. It is not surprising, therefore, that when the paroxysms recur very frequently and at short intervals the mental powers become permanently impaired. Patients who have been long subject to epilepsy acquire a peculiar dull, heavy aspect; and this, with their widely dilated pupils, often enables one to recognise them at the first glance. They are apt to be exceedingly irritable, morose, and gloomy. Sometimes the temper is worse immediately before the epileptic attacks; when a fit occurs it seems to afford temporary relief to the brain, and the patient afterwards feels lighter and more cheerful than for a long time before.

* I myself once nearly overlooked this accident. A patient came with her arm hanging helpless. I was pointing out to the students that a transient paralysis often follows an epileptic attack, when I happened to notice that touching the limb gave pain. I grasped the deltoid muscle, and found that the head of the humerus was out of place.—C. H. F.

Impairment of the intellect is by no means confined to patients who have already suffered for a long time from epilepsy. In children a series of fits, continued for a few successive hours, often produces a permanent state of imbecility, or of mania. A considerable proportion of those who are admitted into asylums for idiots are children who were born with full powers of intelligence, and learned to talk as soon as others; but, having been attacked by epilepsy when perhaps four or five years old, they have since lost sense and intellect, have become dirty in their habits, violent in temper, and unable to recognise their parents. Such cases are frequently brought to our out-patient rooms.

In adults, on the other hand, permanent impairment of intelligence occurs only when epilepsy has been of long standing. Indeed, it is believed that some individuals retain their full vigour of mind after having been liable to fits for years. Julius Cæsar, Mahomet, Petrarch, Peter the Great, and Napoleon are commonly cited as illustrations of the fact that the repeated occurrence of epileptic attacks does not always injure the intellectual powers; but in each of these cases the fits were only occasional, and the evidence of their nature is far from complete. The records of every lunatic asylum afford abundant illustrations that dementia may ultimately overtake those who have been long epileptic before the mind gave way. The severity of the individual attacks has less influence in bringing about such a result than the frequency of their occurrence. Esquirol long ago pointed out that dementia more often occurs in persons who are liable to the *petit mal* than in those who suffer from *le haut mal*.

Pathology.—With regard to the pathology of epilepsy much uncertainty of opinion still prevails. As soon as an attempt was made to distinguish the functions of different parts of the nervous centres it was suggested that in a fit there was a torpor of the brain, associated with excitement of the spinal cord. But the regular order in which one paroxysm succeeds another is inexplicable under such a simple hypothesis; and with the advance of physiology the theory arose that the disease might have its seat in some particular part of the brain as the starting-point of the attacks, or the "epileptic centre." Sir William Gull used to speak of epilepsy as a "function;" and by this he meant that the orderly development of the various symptoms which constitute the seizure must depend upon structural nervous arrangements, like those involved in the more complicated physiological actions. Van der Kolk thought the medulla oblongata was the seat of epilepsy; and recent writers, including Reynolds and Nothnagel, have expressed a similar opinion, but include in the area of disturbance the pons Varolii, or the cervical part of the cord.

We must, however, either confine the supposed "epileptic centre" within very narrow limits, or else include in it the entire length of the spinal cord. We cannot imagine that spasmodic movements of the eyes or of the face bear to such a centre any closer relation than do those of the upper or even of the lower limbs. And if we once give up the idea of fixing the seat of the disease in a definite spot within the medulla oblongata, a little consideration will lead us to include in the affected area the mass of the cerebral hemispheres. There is no other way in which we can so simply explain the facts that consciousness is suspended, and that delirium and excitement are often present. Dr Wilks has always strongly expressed the opinion that epilepsy is an affection of the whole of the brain.

The views entertained by Dr Hughlings Jackson are widely different.

This observer has studied with the greatest care those epileptiform and other convulsions which are caused by local diseases, such as tumours of the surface of the brain. He has laid great stress on the fact that whereas a "destroying lesion," affecting a particular convolution, is capable of causing paralysis, a "discharging lesion" of the same part gives rise to convulsions, which may implicate the opposite face, arm, and leg in a definite order. He supposes that in particular convolutions movements are "represented," which involve the action of many different muscles.

In 1873 Dr Ferrier, following Fritsch and Hitzig, performed a series of experiments on the lower animals, with the express object of throwing light on Dr Jackson's theories of epilepsy (*v. supra*, pp. 615, *et seq.*).

Apart from these experimental results, it would seem, from clinical observations, that when convulsions are set up by local disease of the surface of the brain, the fact that certain parts are especially implicated in the spasms may sometimes enable us to indicate the seat of the lesion. But it is another question whether a similar conclusion is warranted when there is no other evidence of local disease of the brain.

It must also be borne in mind that the spasmodic movements produced by disease—such as are observed in a convulsive attack of whatever kind—are very different in character from the slow and orderly actions to which galvanic stimulation of the brain gave rise in Dr Ferrier's experiments. It is true that spasmodic contractions, and even complete epileptiform fits, were often observed; but these received a separate explanation, as will presently appear. The cases to which reference is now made are those in which, for example, a cat raised the shoulder and adducted the forepaw, as if to strike; or a rabbit munched with its lips and jaws. In an epileptic fit the affected part is violently jerked backwards and forwards, in a way altogether different from what occurs in its natural movements; and surely this suggests that the convulsive movements do not depend merely upon "discharge" of the cortical centres, but are the results of impressions transmitted thence to the corpus striatum, or to some lower plane of the cerebro-spinal centres.

The case is far stronger when, instead of localised spasms, a complete epileptic paroxysm occurs, in which all parts of the body are convulsed, and consciousness is for the time suspended.

Dr Ferrier has discussed this question in his treatise on 'The Functions of the Brain.' His theory is that in the cortex of the brain there are individual centres for each separate muscular action involved in the epileptic convulsion, that they are related to each other in a constant and definite order, and that the attack is due to the discharge of these centres in a tolerably uniform sequence. Much, he says, depends on the primary source of the irritation, but adds that from whatever part of the hemisphere this proceeds, whether from a motor centre, or from the sensory areas towards the back of the brain, the order most commonly observed is that the centres discharge from before backwards, beginning with those of the head and eyes, and ending with those of the leg. He goes on to say that epileptic convulsions can be produced with as great readiness by application of the irritation to the sensory areas as to the motor centres themselves; and that it is doubtful whether consciousness becomes lost when the motor centres of the brain are alone implicated.

These statements of Dr Ferrier seem to deprive Dr Jackson's views, in their most special form, of the support gained from experiment. But Dr

Ferrier's own hypothesis appears less probable than the older opinion that, even when irritation of the surface of the brain causes an epileptic fit, the actual motor impulses start from the basal ganglia or from centres still lower, in the pons, the bulb, or the cord. Surely the orderly development of the phenomena of the paroxysms is more easily explained in this way.

Again, it is well known that there are cases in which epileptic fits are set up by irritation of spinal nerves—by diseased teeth, or (as in Brown-Séquard's experiments) by pulling the hairs of a guinea-pig, when the spinal cord has previously been injured (p. 761). All such cases of reflex eclampsia are much more easily explained on the view that the parts which are the seat of "discharge" are the lower centres, than on the theory that they are the highest centres of all, in the cerebral cortex.*

At the present time a view is widely prevalent, according to which all but the initial phenomena of an epileptic attack result from vaso-motor disturbance. It is supposed that the cerebral arteries undergo spasmodic contraction, and that the consequent anæmia of the brain causes the patient to fall down insensible. It is well known that the face commonly turns pale at the commencement of a seizure. There is ophthalmoscopic evidence that the retina, which derives its blood supply directly from the internal carotid artery, also becomes anæmic. Dr Jackson and Dr Charles Aldridge have proved that the optic disc is pale or even white, and that the arteries which traverse it are much diminished in size. It is to be noted, however, that on the single occasion on which the ophthalmoscope has been used before the clonic convulsions ceased, the disc was at first pink, so as to be undistinguishable from the surrounding choroid; and that it only afterwards became white, and then slowly.

But the most substantial support of the notion that the brain is essentially anæmic during an attack of epilepsy was afforded by the well-known essay of Kussmaul and Tenner. It had before been known that both in animals and in man loss of blood was followed by convulsions. These observers showed that the like result could be brought about by ligature or compression of the four great arteries supplying the encephalon. In reality, however, their experiments only proved that a deficient supply of blood to the brain might be one cause of attacks of an epileptiform kind. They themselves pointed out that ligature of the trachea, rendering the arterial blood rapidly venous, had the same effect; and in either case they referred the convulsions to sudden interruption of the nutrition of the brain. Their theory evidently does not in any way confirm the more modern opinion, that, when an epileptic fit has commenced in the bulb, the patient becomes unconscious as the result of some reflected influence upon the cerebral arteries, which renders the brain anæmic.

After all, the feature of epilepsy most difficult of explanation is the tendency of the fits to recur at more or less definite intervals; and no way of accounting for this seems to be so satisfactory as to refer it to a gradual accumulation of energy in the nervous centres, which is dissipated during the attacks. As van der Kolk points out, it often happens that after a severe fit an epileptic patient remains free longer than usual; but if

* A further argument may be found in the analogy of another paroxysmal neurosis. A tumour in the brain may excite repeated attacks of migraine. One certainly cannot imagine that these are due to "discharge" of the part which is immediately affected by the tumour; they must be due to an influence transmitted downwards to the thalamus or to other sensory ganglia.—C. H. F.

he should have only a slight attack, he soon afterwards has another, perhaps on the following day. Again, in some cases each fit is preceded by a gradually increasing irritability of temper and restlessness, which disappear or are notably diminished after it has taken place. Other patients, as Trousseau remarks, become gay, loquacious, and excited for some hours before an attack; and yet others complain of failure of memory, of torpor, and of physical and mental prostration. Nothnagel mentions the case of a lady, generally a light sleeper, who always knew that she was about to have a paroxysm when she happened to sleep more heavily and longer than usual; nevertheless she would wake up feeling quite well, and would not be attacked until later on in the day.

We are thus brought to regard epilepsy as essentially a paroxysmal neurosis, recurring at more or less regular intervals. In all probability the alterations in the blood supply to the brain, which undoubtedly occur during the epileptic attack, are accidental concomitants rather than essential to the development of any of its symptoms. This view is not inconsistent with the fact that the inhalation of nitrite of amyl is sometimes of service in the treatment of the disease; for when there is an aura the attack can sometimes be overcome by a decided impression on the part to which the aura is referred, and the inhalation may fairly be supposed to act in a similar way, and to cut short the paroxysm by arresting one of its phenomena. Moreover, venesection is sometimes useful, apparently under the same circumstances as those in which the nitrite does good. Yet they must produce contrary effects.

To sum up, then, we must at present be content to adopt the language of some modern writers, and say that epilepsy is "dependent upon an unstable condition of the nerve-tissue in some portion of the nervous system, permitting occasional discharges." This, in reality, is not stating more than that the disease is a "nerve-storm." Just as in migraine, teichopsia may be followed in succession by numbness in the fingers, by headache, by vomiting, and by sleep, so in epilepsy tonic spasms give place to clonic convulsions, and these, again, to stupor or coma. It appears more satisfactory to refer this sequence of phenomena to the gradual extension of some morbid condition from one part of the nervous centres to another, than to ascribe it to modifications in the blood supply.

This account of the pathology of epilepsy can hardly be regarded as complete without some reference to the remarkable experiments of Brown-Séquard, who found that in guinea-pigs and some other animals section of the spinal cord, or even of one or both sciatic nerves, was followed after some weeks by well-marked epileptiform fits, which returned again and again, and could at any time be excited by slight irritation of the cheek (as, for instance, by gently pulling the whisker) or of the side of the neck. The exact bearing of these interesting observations, however, seems to be doubtful, so far as concerns epilepsy in the human subject. They do not afford any support to the idea that the epileptic aura really starts from a distal part of the body and travels along sensory nerves, setting up the paroxysm when it reaches the encephalon. There is every reason to believe that the aura is itself part of the attack, and due to a change in some region of the brain which has close connections with the roots of certain cutaneous nerves; the disturbance is accordingly referred to the peripheral distribution of these nerves.

Ætiology.—In passing on to consider the causes of epilepsy, we have in

the first place to consider *hereditary predisposition*. Dr Reynolds made inquiries which led him to the conclusion that in 12 per cent. of his epileptic patients the same disease had occurred in other members of their families. But this was not all. For in a much larger number of cases other affections of the nervous system could be traced in persons derived from the same stock as the patient. Sometimes it was migraine; sometimes insanity, hysteria, hypochondriasis, or mere nervousness. Thus in a family in whom nervous disorders prevail, one child may be epileptic, another insane or idiotic, and a third hysterical. Several independent observers state the proportion of hereditary nervous influence at about one third of the total cases of epilepsy. Drunken habits in parents are also believed to predispose to epilepsy in their offspring; but the inclination to intemperance may itself be a sign of a neurotic tendency.

There is no reason to suppose that tubercle, rickets, or general malnutrition is concerned in the causation of epilepsy. Nor does intermarriage of blood relations tend to cause this disease in the offspring, provided that neither parent is predisposed to it.

As to predisposition of *sex* and *age*, epilepsy appears to affect males and females in about equal proportions, notwithstanding the remark of Celsus, "*sæpius viros quam fœminas occupat*," and the opposite result of some modern statistics. Dr Reynolds makes some sagacious remarks ('System,' vol. ii, p. 295) on the fallacies of statistics in this respect.

The *time of life* at which epilepsy begins in the great majority of cases is between the tenth and the twentieth years. Dr Reynolds found that in 106 out of 172 cases the first fit occurred between these limits of age, and in most it was within the still narrower period of from thirteen to seventeen years. The more marked the inherited predisposition, the earlier is the average age at which the disease develops itself, and it appears in girls sooner than in boys. Apparently the development and commencing activity of the sexual organs are in some way concerned in the causation of the disease. It has been said that in women the time at which the menses cease to appear is again apt to be attended with the development of epilepsy, but Nothnagel says that this is not the case. According to Dr Reynolds, the period between twenty-five and thirty-five years of age is one at which there is a comparative immunity from first attacks; but they often occur in persons about forty. Exceptional instances are recorded of epilepsy beginning at an advanced age; one, for example, by Trousseau, at about sixty-nine.

In other cases epilepsy is not inherited, it is an acquired condition.

Habits of *intemperance*, *sexual excesses*, and particularly the practice of masturbation are believed to be directly concerned in bringing it about. Indeed, both a first fit and succeeding ones have in some individuals occurred only when they were actually intoxicated, and in others only during coitus. With regard to masturbation, all modern writers speak very guardedly, although there is no doubt that this degrading habit has a most depressing effect upon the health of immature youths, and is capable of causing serious nervous symptoms.* That epilepsy may be produced by alcoholic poisoning, and also by plumbism, there is no doubt.

* I do not believe in the existence of any unfelt irritation starting from the genital organs of modest young women, and giving rise to epilepsy or any similar disease. And I regard as an abomination the operation of clitoridectomy, which was some years ago practised upon a theory of that kind.—C. H. F.

Prolonged anxiety of mind, grief, and destitution have been supposed to lead to epilepsy, but on no sure grounds.

With regard to definite *exciting causes*, there is much difficulty in eliciting the real facts. On the one hand, the parents of children affected with epilepsy are much disposed to conceal a family tendency to that or any other serious disease; and patients themselves are apt to be unacquainted with the real state of health of relatives older than themselves. On the other hand, there is a strong tendency to attribute the disease (and many others, as phthisis) to any accidental circumstance which can by possibility be brought into relation with the first attack. Trousseau says that when a fright was assigned as the cause of epilepsy, he often found on inquiry that it had really occurred months or even years before the fits began, or that it was trifling in character. He himself, however, relates a case in which it appeared clear that the original cause of epilepsy was the terror caused by the sight of a quarrel between two men, one of whom was wounded and fell down dead. In that instance the first attack of *petit mal* occurred within a few days, and subsequently the *haut mal* developed itself.

We have next a very important point to determine, namely, why the first fit should be followed by others. For in some cases the disease has followed attacks which are commonly regarded as accidental in origin.

Thus Dr Hughlings Jackson mentions a case in which a convulsive fit at the onset of scarlet fever proved to be the forerunner of habitual epilepsy. Again, it is said that the simulation of the disease by impostors has ended in their becoming really subject to it; and, if true, this is a still stronger fact in the same direction. Brown-Séquard found that the guinea-pigs in which he artificially set up epilepsy transmitted it to their offspring as an idiopathic disease.

On the other hand, some very remarkable cases are recorded which show that even when epileptiform fits have occurred at intervals for a very long period, as the result of irritation of the nervous centres by some cause acting on a distant part, the liability to their recurrence may cease when the cause in question is removed.

Thus Mr Tomes relates the case of a farm labourer suffering from epilepsy, whose mouth was examined, and the molar *teeth* of the lower jaw were found to be decayed, the fangs of some of them alone remaining. Although he had been treated for the fits during six weeks and complained of no pain, these teeth were removed, and were found to be enlarged from exostosis. During the eighteen months that followed he had not a single fit, although for many weeks before the operation he had had two or three daily. Another case, recorded by Dr Ramskill, is that of a boy who for eighteen months had had epileptic fits, and in whom it was noticed that before the fits he used to rub his left cheek on account of an indefinite uneasiness, not amounting to pain. On examination a molar tooth considerably decayed was found; this was removed, and from that time the boy did not have another fit, although he remained under observation for four months. It must be added, however, that during that period belladonna was administered. A most extraordinary case is one related by Trousseau, of a young clerk, who for several years had been subject to monthly attacks of epilepsy; remedies had been tried in vain at the Hôtel Dieu, when Dr Foville suggested the extraction of some carious teeth which ached constantly. The suggestion was acted on, and from that day the fits disappeared.

Trousseau also relates the case of a man, aged forty, who on several occasions, at very short intervals, was seized with violent epileptic attacks. Dr Monnier found that he had been passing fragments of *tænia*, and gave him large doses of castor oil; a whole tapeworm came away, and from that time the convulsive fits ceased.

Again, an *injury* to the head may be the starting-point of habitual epilepsy. Nothnagel gives the case of a boy who when eight years old fell from a height of twelve feet upon his head. He was stunned for a quarter of an hour, and ten minutes after recovering consciousness he had a characteristic epileptiform fit. There was a slight scalp wound, which healed in a few days. After six weeks he had a second attack, and from that time they recurred at periods which became shorter until he had them at intervals of from four to twelve days. He was twenty-one years old at the time when Nothnagel wrote, and his intellect and memory were already somewhat impaired. A slight scar remained, but this was not painful nor adherent. Nothnagel seems not to have thought that surgical interference would have done any good; and perhaps he was right. But in the 'Lancet' for 1873 two cases will be found recorded, in each of which a piece of the skull was removed by the trephine on account of epileptic fits following an injury to the head. Both of them occurred at Guy's Hospital, the one under Mr Cooper Forster, the other under Mr Bryant. The former patient had received a blow on the head four months before his admission; it left a slight swelling, from which a little pus exuded when it was incised. His first fit occurred the day before he came into the hospital. But a week later he was having four or five fits every hour, and his temperature was 103°. The skull was then trephined, and the piece of bone which was removed was very dense, three eighths of an inch thick in one place, and rough on the outer surface. A fortnight afterwards he could walk the length of the ward, and he never had another fit—at least until the time when the report of the case ceases, which was two months from the date of the operation. In Mr Bryant's case the accident had occurred five years before, and attacks of the *petit mal* had occurred at intervals of about a week during the whole period. The cicatrix was still tender, and occasionally painful. Internal treatment having been tried without any good result, the trephine was applied, and a piece of thickened bone removed. The fits at once became less frequent, and seemed to occur only when he was depressed from want of food in his miserable home, or when he was exhausted by diarrhœa. The same medicines which he had before taken with no benefit were resumed, and after a time he became able to earn his living. When the case was reported sixteen months later, he seemed to be permanently cured.

Relation to other convulsive fits.—One point in favour of a real distinction between true epilepsy and symptomatic eclampsia is the observation of Dr Tyler Smith that puerperal convulsions are by no means of frequent occurrence in women who are habitually subject to epileptic fits. But it is clear from the foregoing paragraphs that there may in practice be great difficulty in applying the distinction in question. Another instance of the same difficulty is afforded by infantile convulsions. On the one hand, it is said that persons subject to epilepsy in adult life are often found on inquiry to have had fits in early childhood. Nothnagel expressly states that this is often the case with children born of epileptic parents, and with those who in later years themselves become epileptic. But, as Dr Jackson points out, there are at present no facts to show what is the proportion of

those who having had infantile convulsions afterwards escape epilepsy; it can hardly be doubted that they form the immense majority. The fits of infants were formerly attributed to irritation of the nervous centres from teething or disorder of the alimentary canal. But the more closely such supposed causes are inquired into the less clear does their relation to the convulsive attacks appear to be. The tendency of modern observation is to associate infantile convulsions and laryngismus stridulus with rickets. Whatever part in their causation one may assign to external sources of irritation, it is certain that another very important part is due to inherited or acquired conditions of the nervous centres, disposing them to convulsive discharge. It may well be that in infancy the brain yields to influences which in after life it successfully resists. The higher centres of the cortex have not yet acquired inhibitory or restraining influence over the lower excito-motor mechanisms of the basal ganglia and the cord. It would be a serious error to say that the occurrence of fits in childhood involved danger of epilepsy in adult life: yet it seems clear that no absolute line of distinction between them can be drawn; nor, in general, between habitually recurrent epilepsy and the various forms of eclampsia, or of epileptiform fits due to external irritation.

The difficulty is evaded if we regard all these affections as members of the same group of paroxysmal neuroses.

Anatomy.—The only morbid changes which are to be found in the brain in epileptic patients must be regarded as *effects*, and not as *causes* of the fits. Perhaps the most important are dilatations of the capillary blood-vessels in the bulb. These were first described by van der Kolk, who maintained that there was a definite relation between their seat and the symptoms observed in the paroxysms, the nucleus of the hypoglossal nerve and the olive (*corpus olivare*) presenting dilated vessels when the tongue had habitually been bitten; whereas in cases in which the tongue escaped he found them chiefly in the nucleus of the vagus. He also described an albuminous exudation into the bulb, causing at first induration of its substance, but afterwards fatty degeneration and softening. Similar conditions have been described in cases of hydrophobia, of tetanus, and of chorea.

The skull is often exceedingly thick and dense in those who have long suffered from epilepsy, the membranes are opaque, and the brain generally is indurated; but these changes seem to bear a closer relation to the impairment of intellect which is so generally present in cases of long standing than to the primary paroxysmal attacks.

Diagnosis.—This involves several different questions, some of which have already been dealt with, or will be considered in other chapters. For example, one may have to distinguish an attack of minor epilepsy from one of *cardiac syncope*, and an attack of major epilepsy from one of *hysteria*.

(*a*) When a person is in a fit, which, if really epileptic, would belong to the major form, we have often first to ask whether it is possible that he is *malingering*. In the first place an impostor is likely to choose for his fits a frequented spot, where he is sure to be seen. When he throws himself upon the ground it is in such a way as not to hurt himself; he may even put out his hands to break his fall. He probably overacts his part, crying out many times instead of only once, throwing his limbs violently about, and making the attack last much longer than a genuine paroxysm of epilepsy. There is, of course, no pallor of his face at the com-

mencement; his skin, instead of being cold, is warm and covered with sweat; his pulse, even if quickened, does not become irregular; his pupils are not dilated, nor insensible to light. He does not keep his eyes wide open, nor are the eyeballs distorted; generally he only separates the lids a little from time to time, so as to watch the bystanders. If an attempt should be made to raise the upper eyelid he resists it, and instead of his conjunctiva being insensible, the slightest contact excites forcible contraction of the orbicularis muscle. On the other hand, he may readily allow the thumbs to be drawn away from the palms, and afterwards close them again. But, according to Dr Marc, in a person really attacked by epilepsy the thumbs require force to loose them; and, when once extended, they remain so until the end of the attack, or at least until a fresh convulsion begins. Again, in a simulated fit the tongue is seldom or never bitten, and there is no foam about the mouth—unless, indeed, this is imitated by a piece of soap in the cheek. The muscular strength of a malingerer is not increased. If a little snuff be blown into his nostrils he cannot help sneezing; in a real fit no such effect is produced. Another test is the absence of sensibility to pain: a test commonly adopted by policemen and others is to press the thumb-nail forcibly beneath that of the person supposed to be in a fit. As already mentioned, there is reason to believe that repeated attempts to feign epilepsy have sometimes ended in the development of a genuine attack.

An educated medical man would naturally succeed in imitating a fit better than one who has no professional knowledge. Trousseau relates that Esquirol, who did not believe that an attack could be simulated so as to deceive, was once talking this very matter over with him and with Calmeil at the asylum at Charenton, when the latter fell on the floor in violent convulsions. Esquirol examined him for a moment, and then said, "Poor fellow, he is epileptic!" upon which Calmeil got up and asked him whether he still retained his opinion.

(*b*) Assuming our patient's attacks to be genuine, one has next to consider whether they are really epileptic or epileptiform. This question is often settled by the clinical history; the case may, perhaps, be of many years' standing, and the fits may have gradually developed from the minor into the major form. But one must bear in mind that attacks may recur at intervals for a great length of time, and present all the characters of idiopathic epilepsy, and yet be symptomatic. Trousseau's case has been already mentioned, of the clerk who had been liable to epileptiform seizures for several years, at monthly intervals, and in whom they ceased when some carious teeth were removed. The same writer relates the case of a lady, aged seventy-one, who for thirty-one years had been subject to attacks recurring with daily increasing frequency, so that she at length had as many as twenty-one in the twenty-four hours. Her forehead and nose presented characteristic signs of former syphilitic disease; and the administration of mercury and iodide of potassium checked the fits.

(*c*) Generally, however, the question of syphilis need hardly be entertained unless the fits are comparatively of recent origin; and the same may be said of Bright's disease, lead-poisoning, chronic alcoholism, and the various organic affections of the nervous centres. The diagnosis between these diseases and true epilepsy must depend mainly upon the presence or absence of the other symptoms which severally characterise them, and upon the condition of the patient between the attacks.

Prognosis.—If we separate the "true" idiopathic disease from sympto-

matic eclampsia due to injury, tumours or other "gross" lesions of the brain, the epileptiform convulsions of uræmia and of the puerperal state, hystero-epilepsy and infantile convulsions—we shall find that epilepsy is no less grave in its prognosis than alarming in its symptoms; and although by treatment we can greatly reduce the number and severity of the attacks, it is only in exceptional cases that we can cure it.

If epilepsy begins in childhood there is good hope that it will disappear about puberty. Eclampsia occurring for the first time after fifty is usually symptomatic.

The frequency of the fits is of worse prognosis than their severity. True *petit mal* commonly ends in *grand mal*.

Hereditary epilepsy is, as a rule, the most difficult to cure.

When no improvement takes place under treatment, and no cause or occasion of the attacks can be discovered and removed, the natural progress of the disease is towards mental imbecility.

Treatment.—In the treatment of epilepsy two things have to be considered: (1) the management of the attacks themselves; and (2) that of the intervening periods, with a view to prevent their recurrence.

(1) So far as concerns *the paroxysm*, the most important point is to ascertain whether the patient has any warning of its approach. If there should be a distinct aura, starting, perhaps, from the hand or foot, one can often, by compressing the limb above, arrest the fit after it has commenced. Some years ago there was, in the Evelina Hospital, a girl who constantly wore round her wrist a piece of cord; this was pulled tight as soon as she felt the sensation which indicated that she was about to have an attack, and not a single one developed itself during several weeks; after a time bromide of potassium was administered, and the aura then ceased to recur. Many instances of a similar kind have been placed on record by different observers. Dr Bazire mentions the case of a woman whose fits were always preceded by spasmodic closure of the left hand; by forcibly extending the fingers, and keeping them open, an impending attack could be warded off. A patient of Dr Reynolds' had jerking of the left leg, which was drawn up behind him, when his attack began; it was arrested by extension of the muscles. Another plan which has been recommended consists in cauterising the surface from which the aura proceeds.

Even when an epileptic fit is not ushered in by any symptoms beyond pallor of the countenance and tonic spasms, it would appear that the prompt inhalation of nitrite of amyl is sometimes capable of arresting it. Sir Crichton Browne has related some instances of this. One is that of a man who started up suddenly in bed, with his eyes fixed and his head turned to one side; these symptoms were known to indicate the approach of an attack; but the nitrite was administered, and the patient at once fell back on his pillow in a half-fainting state, but without the slightest agitation of the muscles. In another case a fit had actually begun in the ordinary way with rigid stretching of the hands by the side, and turning up of the eyeballs, when the nitrite was held to the mouth and nostrils, and in twenty seconds complete recovery had taken place.

After an epileptic attack has fully developed itself, there is evidence that it may still sometimes be cut short by compression of the carotid artery in the neck. This procedure must be supposed to diminish to some extent the blood supply to the brain. It was first suggested by Dr Parry, of Bath, towards the end of the last century. He relates a case in which it proved

successful. A man who had been liable to epilepsy for two years was one day beginning to have a fit; his eyes were assuming a vacant stare, and convulsions were beginning about his throat, when Dr Parry made strong pressure over the right carotid artery; upon this the convulsions ceased, and the attack proceeded no further. He instructed the patient how to compress the vessel, and the latter afterwards assured him that when he had sufficient warning he was often able to prevent the epileptic paroxysm. At Guy's Hospital this practice was adopted by the late Mr Stocker, and sometimes with striking results. But it was most useful in cases with a strong hysterical element. The plan which Mr Stocker used to adopt was to press both thumbs into the neck, one on each side, towards the spine; in doing so he doubtless compressed many other parts beside the carotid arteries, and the pain caused may well be supposed to have been concerned in the rapid restoration of the patient to consciousness, at least when the case was hysterical.

When we are called to a patient in an epileptic seizure we must see that his clothes are loosened, especially about his neck, and we must take precautions to prevent his injuring himself. We may endeavour to keep the tongue from being bitten by putting a piece of india-rubber between the teeth, but this involves the risk of its falling back into the throat and causing suffocation. Epileptics who are liable to attacks in the night should be very careful to remove false teeth from the mouth before going to bed, lest they should become impacted in the pharynx during a paroxysm.

In the *status epilepticus* it would appear that the best remedy is the inhalation of the *nitrite of amyl*. Sir Crichton Browne has recorded ten cases in which he employed it, and eight of them terminated in recovery. The effects of the remedy were of the most striking character. For instance, a man, aged thirty, had from May 6th to the 10th had from twelve to sixteen fits a day; on the 11th he was in a most critical condition; he lay on his back, breathing stertorously, with livid purple features, and streaming with perspiration; the pulse was 140, the temperature 103°. It seemed useless to make trial of the nitrite, but as a forlorn hope he was made to inhale five drops every hour. His breathing at once became less laboured, and he had only three more fits that day, and on the 12th there were gleams of consciousness; the pulse and temperature fell; on the 14th he could answer questions, and by the 17th the fits ceased, and he passed into his usual state of health, and was able to take part in domestic work. Dr Browne adds that he has found no other plan of treatment of nearly the same value in the status epilepticus as inhalation of the nitrite of amyl, although other measures may occasionally be useful.

The withdrawal of a few ounces of blood has sometimes suddenly restored to consciousness patients who were in a state of profound coma. Some years ago a very striking instance of the same kind occurred at Guy's in the practice of Dr Wilks. Perhaps this undoubted benefit of *venesection* in apoplectic epilepsy may explain the reputation of bleeding in cases of cerebral hæmorrhage.

(2) For the *prevention* of epileptic fits in those who are liable to them—in other words, for the cure of the disease epilepsy—the *bromide of potassium* surpasses all other drugs in efficacy. Sir Charles Locock, in 1857, was the first to recommend it, and he spoke of it as being especially serviceable in women in whom the attacks coincided with the menstrual periods. But subsequent observations have shown that there is no such limitation of its curative

power. It is given in doses of from ten to thirty grains three times daily, and it must be continued for months, or even for two or three years. Its effect is sometimes to free the patient for ever from the liability to recurrence of the attacks. In other cases it suspends them for a time, or diminishes their frequency and severity, but when its administration is suspended they soon become as bad as before. In yet other cases it does some good for a time, but seems to lose its power, although the patient may go on taking it without interruption. Lastly, in a very few instances it appears to be altogether useless. No explanation has yet been found for the varying effects of bromide of potassium in different cases.

Most persons can take from ten to twenty grains of bromide of potassium three times daily without suffering any ill effects. But when it is given in larger doses it is apt to cause very striking symptoms after ten days or a fortnight, and the condition so produced has been called *Bromism*. According to Dr Bazire (Syd. Soc. translation of 'Trousseau's Lectures,' vol. i, p. 100) it is characterised by headache, apathy, impairment of the special senses and of common sensations, loss of sexual appetite and vigour, enfeeblement of muscular power in the limbs, tremor of the hands, and impaired action of the heart. A young American lady came under the writer's care, who, after taking bromides for several years to cure epilepsy, had fallen into a dull, stupid, listless condition, sitting for hours without moving, and incapable of joining in society. A very definite symptom of bromism is anæsthesia of the velum palati, uvula, and pharynx, which may be tickled without producing any efforts of deglutition.

Another remarkable effect is the production of a cutaneous eruption. This commonly resembles acne more or less closely; it consists of pustules surrounding hair-follicles, but these are arranged in patches or groups, and dry up into large scabs, beneath which the skin becomes red and thickened. One case of this kind is depicted on the forty-third plate of the Sydenham Society's 'Atlas.' In that instance the scalp and the extensor surfaces of the limbs were especially affected by the eruption, but the face and the legs are more often its principal seat. This unpleasant effect of bromides may be prevented or mitigated by adding arsenic to the medicine.

One is not infrequently consulted by a patient who has just had a first epileptiform attack. The bromide should then be prescribed without delay. The probability of its usefulness may fairly be inferred from its ascertained efficacy in the large proportion of the cases in which its value can be fairly tested. If one were to lay the matter in all its bearings before the patient himself, he would certainly wish to take the medicine regularly for a considerable length of time, even though, in the event of his remaining free from further attacks, it must always remain doubtful whether or not there was a real necessity for treatment.

If the bromide of potassium fails to check the recurrence of attacks of epilepsy, although given in large doses and with the utmost perseverance, we must then have recourse to some other remedy; and perhaps the best is *belladonna*. This was especially recommended by Bretonneau and by Trousseau. At first a quarter of a grain of the extract should be given twice or three times a day; and the dose should be gradually increased. At one time a patient of the author's took two grains three times daily for a great length of time, with marked benefit. Trousseau insists on the necessity that belladonna should be continued for a long period if its value is to be fully tested; a year, he says, is sometimes scarcely sufficient

for the discovery of its influence; and if in the second year there should be some improvement it may be worth while for the patient to go on taking it for three or even four years. He speaks of it as completely curing the disease in some very rare cases; but Dr Reynolds has never known it do more than diminish the frequency of the seizures.

The salts of *zinc* are useful in some cases of epilepsy. Dr Wilks has had under his observation more than one case in which the patient was always better when under their influence. Dr Reynolds speaks favourably of the oxide, but has seen no good result from the sulphate.

The nitrate and the oxide of *silver* have been recommended, but are probably useless. And one must not forget that, taken for a length of time, this drug will stain the skin of a bluish-black colour. It is believed that these preparations may be administered with safety if the course is not allowed to be continued for more than six weeks. Twenty years or more ago an American was to be seen about London who had been castrated and afterwards completely blackened by nitrate of silver in the hope of curing his epilepsy. His skin looked as if it had been polished with black-lead, but the result was negative so far as the disease was concerned.

In some cases the introduction of a *seton* into the nape of the neck has led to the suspension of epileptic fits. A patient of Dr Wilks in Mary Ward, subject to epileptic fits, had a seton put in her neck. The fits ceased entirely. After more than a year the seton was removed and they reappeared. It was again inserted, and again the convulsions ceased.

The application of ice to the spine, as recommended by Dr Chapman, has been fully tried by Dr Reynolds, who reports that it did no good.

Trephining is a very dubious remedy in cases of idiopathic epilepsy, though useful in Jacksonian eclampsia from a cortical lesion.

The food of patients suffering under epilepsy should be digestible, and their meals leisurely and regular. Dr Wilks speaks of having seen cases in which reducing the quantity of meat has been followed by a decline in the number and severity of the fits; but in other instances a generous diet has been advantageous. The writer knew a lady who, by the late Dr Radcliffe's advice, lived on vegetarian diet for some years; and the effect in checking the fits both in frequency and severity was marked. Some definite experiments in regard to the influence of animal food have been made at the West Riding Asylum by Dr Merson, who kept a number of patients for a month on a diet including much meat, and then for the same period on one in which there was none at all; there was no marked difference in the number of fits, but several of the patients were much more dull and languid when taking animal food than when restricted to farinaceous diet.

Both the mind and the body of epileptic patients should be kept in exercise, short of fatigue. The limbs should never be allowed to get cold, particularly at night. Dr Reynolds says that in many cases nocturnal seizures have been prevented by a prop being put under the upper half of the mattress, so as to keep the head and shoulders well raised.

PAROXYSMAL VERTIGO.* — Another affection which may occur in paroxysms is vertigo or giddiness. The term "paroxysmal vertigo" is better than "epileptic vertigo," for the latter has been used as a synonym for the less severe form of epilepsy—the *petit mal*, which is not usually

* Vertigo (*vertere*, to turn) answers to the English words giddiness or swimming of the head.—*Fr.* Vertige.—*Germ.* Schwindel.

attended with giddiness, and it is almost always attended with loss of consciousness (cf. p. 751).

Two forms of paroxysmal vertigo are recognised by systematic writers. In one the patient feels as though he himself were made to turn round and round, or were against his will impelled forwards, or backwards, or to one side. In the other he fancies that objects are revolving round him. In each case he remains perfectly conscious, and all that occurs is duly registered in his memory. If he has had previous attacks, he may be well aware that his sensations are without foundation; yet by the strongest effort of his will he is incapable of overcoming their effect. In a first attack he may be completely deceived. A patient, who happened to be travelling by railway, beheld one side of the carriage suddenly rise four or five feet, and throw him into the opposite corner. He naturally supposed that there was a serious accident; but in reality he had not moved from his seat. Dr Ramskill relates that a patient of his, who was attacked while in the street, felt the pavement uneven, with alternate depressions and elevations over which he seemed to be obliged to lift his feet. At the same time the shop windows seemed to him to be moving forwards, and the passers by to be racing after one another. But he also felt giddy in himself; and as a matter of fact the two forms of vertigo just described cannot be regarded as distinct affections. In most cases both are experienced, either at the same time or successively.

The gait is unsteady or reeling; the patient feels afraid of running against other people or surrounding objects; he catches hold of some support; or he may lose his balance and fall to the ground. Sometimes closing the eyes removes the sensation of vertigo for the time. Nausea commonly accompanies the attacks, or even vomiting.

Paroxysmal vertigo is often connected with impairment of the sense of hearing, the patient being more or less completely deaf on one or both sides, and generally experiencing sensations of buzzing or singing in the ears. In 1861, Ménière recorded in the 'Gazette Médicale' some remarkable instances of this kind; and such cases have been described under the name of Ménière's disease, "auditory," "aural," or *labyrinthine vertigo*; but the prevalent interpretation of their pathology is far from proved.

In the first place, it is certain that affections of the middle, and even of the external ear may give rise to attacks of giddiness, faintness, and sickness. For example, in the 'Archives of Ophthalmology and Otology' for 1871, both Knapp, of New York, and Brunner, of Zürich, mention cases of aural catarrh in which such symptoms appeared; and Toynbee many years ago asserted that cerumen accumulated in the external meatus might by its pressure on the membrana tympani produce similar effects.

In the great majority of cases, however, the more accessible parts of the organ of hearing are free from disease. If the deafness is of one ear, a tuning-fork is not heard on that side, even when placed upon the teeth or upon the top of the head. It is hence inferred that the seat of mischief must be the internal ear.

At this point some very interesting physiological observations appear to find their application. Many years ago Flourens discovered that in pigeons and rabbits section of the semicircular canals causes strange disturbances of equilibrium. More recently Mach, of Vienna, and Crum Brown, of Edinburgh, have shown good evidence that the function of these structures is to

furnish the impressions which form the principal basis of our knowledge as to the relation between our movements and those of surrounding objects. They have even shown what are the several disturbances of equilibrium which irritation or destruction of each canal may be expected to produce. Charcot observed a case of paroxysmal vertigo in which the lesion was chiefly in the left ear, and in which the direction of reeling was principally forwards, but sometimes backwards, while occasionally there was a sense of rotation on a vertical axis, always from left to right. This last would, according to recent writers, be due to irritation of the left horizontal ampulla, while movements forwards and backwards would answer to irritation of the posterior and superior canals respectively. Destruction of the same parts would, however, produce precisely the converse effects, and thus there is no difficulty in accounting for the fact that some patients have shown a tendency to reel towards the side on which they were deaf. In either case the actual movements are supposed to be the reflex results of the impressions conveyed to the co-ordinating centre from the several canals, which under normal conditions balance one another, but which no longer do so when one of them is diseased or injured.

Even when morbid changes in the meatus or tympanum are obviously present, it is commonly supposed that the direct cause of vertigo is disorder of the labyrinth; for pressure upon the fenestra ovalis can easily be conceived to cause increased tension in the semicircular canals. Thus all cases of "auditory vertigo" might come to be regarded as alike examples of Ménière's disease or labyrinthine vertigo.

Ménière himself did not merely wish to draw attention to the fact that vertigo is apt to occur in those who suffer from deafness or from some disease of the internal ear; for this was well known before. What was really new in his paper was that he endeavoured to show that sudden apoplectiform symptoms (including at least a transient loss of consciousness) might occur in a person previously healthy, might be followed by deafness, and that the cause of such attacks might be an affection of the internal ear. He relates several cases of patients who fell down insensible, and who, when they recovered, were found to be deaf; and a similar instance has been recently recorded by Knapp.

The only one of Ménière's cases in which a *post-mortem* examination was made is the tenth and last of his series. A young woman, while menstruating, undertook a night journey outside a coach. She suddenly became completely deaf, and was admitted into Chomel's wards. The principal symptoms were constant vertigo and vomiting. She died on the fifth day. At the autopsy no disease could be discovered in the nervous centres; but the semicircular canals in each ear contained a reddish plastic substance. Surely this observation is very inconclusive. Cases in which an autopsy fails to reveal a satisfactory explanation for cerebral symptoms present during life are, after all, not very rare; and it seems rash to assume that the state of the labyrinths was the real cause of the fatal illness in Chomel's patient. Moreover, as Brunner points out, even if full value were allowed to the case in question, one could hardly take it as demonstrating the nature of those other cases in which cerebral symptoms come on suddenly and rapidly pass off. In these it has been supposed by Knapp that hæmorrhage takes place into the semicircular canals. But since the deafness is often simultaneous in the two ears, the blood must be effused on both sides at or about the same time, and this makes the explanation very improbable.

It is true that hæmorrhage into both retinæ occurs in cases of Bright's disease; but surely not so as to cause sudden and total blindness.*

A different view has been suggested by Dr Wilks; namely, that when there is no affection of the meatus or tympanum, the deafness and the cerebral symptoms are both in some cases due to changes in the nervous centres. It is evident that such an explanation is particularly applicable to cases of Ménière's disease in the stricter sense of that term; the sudden loss of hearing in both ears may fairly be attributed to an affection of the auditory centre; and the giddiness to a similar affection of the centre for equilibrium, which is probably adjacent, since its most important afferent nerves are those which come from the semicircular canals. Whatever peculiarities in the direction of the vertiginous tendencies may be observed, such as have been supposed to depend upon affections of particular ampullæ, they can all be referred to corresponding changes in the centre, for in this the functions of each canal must be fully represented. The analogy of the other paroxysmal neuroses strongly supports Dr Wilks's view. Impairment of sight is a frequent symptom of migraine, and it is certainly due to an affection of the brain and not of the eyes. Indeed, "cloudiness before the eyes" and "obscuration of the visual field" are mentioned as having been present with the vertigo in some of Ménière's and Knapp's cases; and it may be that in these instances the attacks presented a combination of the two neuroses, the nerve-storm spreading beyond its usual limits and encroaching upon the area concerned in migraine. So also in the "apoplectiform" cases described by Ménière, we may account for the loss of consciousness by supposing that the disturbance spread over the hemispheres, as it probably does in epilepsy.

Another strong argument in favour of Dr Wilks's view is afforded by the fact that bromide of potassium may remove both the giddiness and the loss of hearing at the same time; and Mr Hinton recorded under the name of Ménière's disease a case in which paroxysmal vertigo and sickness had been associated with only transient deafness, and in which all these symptoms together were brought back by the administration of quinine.

Knapp has observed that in certain cases the impairment of hearing is particularly marked for certain musical tones, those of the middle octaves being distinctly perceived, while those of the lower, and still more those of the higher octaves are heard very imperfectly. He regards this as a proof that the seat of the affection is in the labyrinth; but the force of the argument is not obvious. Much more weight must be allowed to an observation of Charcot's, that some patients experience sensations of vertigo and buzzing in the ears only so long as the deafness is partial, and lose these symptoms as soon as it becomes complete. But even if we should have to admit that in these instances the internal ear is really the part primarily affected, it would by no means follow that the same thing is true of the "apoplectiform" cases, nor that the vertigo is anything but a neurosis. Why may not the same symptoms be produced in many different ways, and yet be always themselves the immediate result of one particular kind of nervous disturbance? Epilepsy and migraine may be excited by a variety of causes, and so may paroxysmal vertigo.

The late Mr Hinton published nine cases of vertigo, mostly paroxysmal, and associated with deafness, tinnitus, and nausea or vomiting, in the 'Guy's

* The writer once saw retinal hæmorrhage in the course of chronic Bright's disease cause sudden and total blindness; but in that patient the other retina had been long affected without his knowledge.

Hospital Reports' for 1873 (vol. xviii, p. 193). In some of these the hearing was affected on both sides, and in two (Cases 3 and 9) the perception of certain musical sounds was definitely impaired.

A classical instance of vertigo, nausea, deafness and pains in the head, coming on as a paroxysmal disease early in life and persisting until old age and senile imbecility, is that of Dean Swift. Dr Legg and Dr Bucknill have argued in favour of its being a typical case of Ménière's disease; but we have, of course, no knowledge of the condition of the labyrinth. Swift's account of the origin of his attacks in a fit of indigestion while he was living with Sir William Temple would point to *vertigo a stomacho læso.**

Writers describe this "gastric vertigo" as distinct from other varieties of giddiness. It may be that the stomach is not so often concerned in the production of vertigo as the so-called bilious condition which Dr Murchison identified with lithæmia. But without deciding this point the question is whether the giddiness due to disorders of digestion is different in kind from that which depends upon deafness. Dr Wilks has remarked that the vertigo caused by derangement of the liver occurs chiefly when the patient stoops or lays his head upon the pillow, and ceases when he stands upright. But this distinction is not constant; and in many cases there is nothing in the nervous symptoms themselves to show that they depend upon one rather than another of the various causes to which they may be due.

In some cases the ingestion of food which disagrees with the patient leads so quickly to swimming in the head that the connection cannot be overlooked. Dr Murchison speaks of a medical friend of his, who has long suffered from gout, and who, whenever he drinks a cup of tea or a glass of champagne, is seized with sudden giddiness; his head feels empty, and neighbouring objects seem to whirl about him; he would fall did he not lay hold of something to support him; after a few seconds or minutes the attack passes off. In other patients the vertigo lasts longer. Dr Ramskill relates the case of a merchant who was one day quietly walking in the City from one office to another, when he was seized with giddiness, so that he reeled, and had to lay hold of a post which was near at hand. In a few hours, after a free evacuation of the bowels, he became better, but he felt weak and shaken, and complained of a heavy diffused headache. About three hours before the attack he had eaten hastily, and with imperfect mastication, a breakfast of which sausages and Devonshire cream formed a part; and to this the vertigo was ascribed, no doubt with justice. Yet, during the following month, the same patient had five similar attacks, not one of which could be traced to any such cause, he having in the meantime become very particular as to his diet. Dr Ramskill goes so far as to say that in "stomach vertigo" it is the exception if one is able to trace any positive signs of gastric disorder: the proof is that the complaint is cured by treatment directed to the regulation of the digestive organs. Thus, a second medical friend of Dr Murchison's, who had never had gout, and in whose case the only recorded indication of lithæmia was that his urine was often loaded with lithates, was seized with dimness of sight every night while writing. He took iron and quinine and other tonics, but without any benefit. He was advised to give up practice for a time, and try the effect of a change of air; but while he was making up his mind to so serious a step, he took a few

* "I was seized with so cruel a fit of that giddiness" ("and weakness and sickness in my stomach") "which at times hath pursued me from my youth that I was forced to lie down on a bed for two hours" (Dec. 17, 1734).

grains of blue pill, whereupon his symptoms at once disappeared. So, again, Trousseau quotes from Boerhaave the case of a man who, during two years, was seized with symptoms of vertigo whenever he attempted to stand up. In vain had the ablest practitioners attempted to cure him. He had a first attack of gout, and from that time the giddiness ceased.

To complete the chain of evidence which proves that vertigo is one of the paroxysmal neuroses, there remains the fact that it may replace other members of this group. Giddiness is occasionally present during the paroxysms of migraine; and Dr Liveing refers to two cases, in each of which an attack of intense vertigo, of short duration, appeared several times to replace the ordinary sick headache. The connection of vertigo with epilepsy is, perhaps, closer still; for giddiness is one of the symptoms of the *petit mal*.

There are, however, cases in which this neurosis is idiopathic and remains unchanged in type for many years. Dr Ramskill states that he has met with two instances of this "essential" vertigo in which the complaint appeared to be transmitted by inheritance. One of his patients suffering from vertigo had a father living, and then aged seventy-one, who had himself been subject to it for thirty-five years; he also had asthma. Another patient complained for three years of giddiness for which no cause could be discovered, and which resisted all kinds of treatment. It is true that in that case the giddiness after a time became almost continuous; and *persistent* vertigo is probably more often due to anæmia from disease of the arteries of the brain than to any other cause. But it seems probable that in exceptional cases any of the paroxysmal neuroses may at last cease to be interrupted by intervals. For example, a case is recorded by Charcot ('Progrès Méd.,' ii) of a woman, aged fifty-one, who had for six years suffered from continuous vertigo, which did not intermit even at night, and was so severe that she could not walk, nor even stand, for the slightest movement of her head made her clutch at surrounding objects for support. The complaint had lasted twenty-six years; but for a long time it was purely paroxysmal. She had disease of the tympanum on each side.

In the *treatment* of vertigo bromide of potassium is of more service than any other medicine, and its value is sometimes more striking than even in epilepsy itself.

The aural surgeons use chloride of ammonium for those cases which are associated with deafness. We have lately (April, 1890) had a marked case of paroxysmal vertigo, with severe headache, tinnitus, and temporary deafness with vomiting, but with no evidence of organic disease of the brain or the ear, in Philip Ward. The patient, a man of about fifty, had been subject to the attacks for several months, and recovered rapidly under moderate doses of bromide.

A careful inquiry must be made for symptoms of dyspepsia or of lithæmia, and Dr Ramskill recommends that alkalies and vegetable bitters should be used in all cases on the chance of their being of service. It is a good plan to give the bromide with a few grains of carbonate of soda, and with equal parts of the infusions of rhubarb and calumba. Wine is valuable in senile vertigo. Charcot's patient was cured in from two to three months by fifteen grains of quinine daily; but in a very characteristic case of auditory vertigo lately under the writer's care the patient had, by Professor Charcot's advice, taken quinine in full and repeated doses without the least benefit.

PAROXYSMAL MANIA.*—There is a paroxysmal neurosis in which the attacks take the form of transitory mania.

A striking instance is recorded by Dr Maclaren in the 'Medical Times and Gazette' for 1876. The patient was a slight, gentle-looking lady, aged forty-three, with winning manners and a soft quiet voice. She was characterised by exalted religious feelings and morbid sensitiveness. She would be reading her Bible and talking gently to her attendant, when suddenly, without a moment's warning, she would throw the book out of the window, and make a rush to run her head into the fire; or she would, perhaps, turn on the attendant and try to strangle her. She would then struggle on, keeping all the time perfectly silent, or uttering only an occasional word of Scripture, until she was exhausted, or until by a kind of awakening she became restored to her former condition. Sometimes in the attacks she would expose her person. During the intervals she had no recollection whatever of what she had done; at the most she could slightly recall the impulse which led her to attempt some act of violence.

One peculiarity of the paroxysms in this case was that in each of them the patient made efforts to get at one particular picture, which at other times excited no emotion whatever. Thus it seemed that she followed out in successive attacks trains of thought of which she had no knowledge during the intervals; and her condition might so far be termed one of "dual consciousness."

Although Falret described this disorder as *furor epilepticus*, it would appear that a liability to epilepsy is not always present. Dr Maclaren's patient had never had epilepsy, although she was a member of a family in which neurotic affections prevailed; but there is no question that the affection is one which bears a very close relation to epilepsy. As a rule, patients who have seizures of this kind have before suffered from attacks of the *petit mal*, if not of the *haut mal*. Dr Hughlings Jackson, indeed, has expressed the opinion that a transitory epileptic paroxysm really occurs each time before the mental symptoms develop themselves. In other words, he thinks that the affection is identical with that form of mania which sometimes follows an epileptic fit (p. 754). This view rests upon a theoretical basis. He supposes that a necessary condition for the occurrence of the mental disorder is the removal of the control of the highest centres, which are exhausted by having discharged themselves during the fit.

Even when paroxysmal insanity occurs in those who are really subject to frequent epileptic fits, one may be unable to ascertain the fact at the time. It often happens that a patient in this condition is brought to a public hospital by the police, and, as Dr Jackson remarks, it may be impossible for one to say whether he is an epileptic, or drunk, or suffering from meningeal hæmorrhage. He records an interesting case of a woman who was brought to the London Hospital in a maniacal state, with a deep gash in the left arm, by which the elbow-joint was opened, and all the soft parts in front of it were cut through. She accused different people of having inflicted this wound upon her, but it was ascertained beyond doubt that she had done it herself. She had been cutting bread for her children's tea when she suddenly sent them all out of the room. A short time afterwards she was found lying in a pool of blood on the floor. On the following day she was rational, but furious mania returned several times during the next week.

* *Synonyms.*—Epileptiform mania — Paroxysmal insanity — Furor epilepticus.—*Fr.* Folie circulaire.

On inquiry it was ascertained that she had been subject to epilepsy, in both the minor and the graver form. The patient doubtless received the suggestion which led to the infliction of the wound from the circumstance that she had a knife in her hand at the time.

The sufferer from this form of insanity is particularly liable to find himself in the hands of the police for some offence committed during the paroxysm; and although to a skilled medical observer it may be perfectly evident that he is irresponsible, there may be much difficulty in making this clear to others. Dr Jackson has done much towards the elucidation of such cases by studying other instances in which the acts performed are not criminal but absurd; they may be characterised by precisely the same degree of adaptation of means to ends, and yet they leave no trace on the memory. Thus one of his patients had been talking about supper, and it had been agreed that he and his wife should have some cold fowl, and her sister some cocoa. Soon afterwards he felt the symptoms of an attack and sat down on a chair against the wall of the kitchen, where he happened to be. He remembered nothing further, but his sister-in-law came in and found him standing by the table mixing cocoa in a dirty gallipot, which was half filled with bread and milk for the cat, and stirring the mixture with a mustard-spoon, which he could not have obtained except by going to the cupboard for the purpose. If the object fetched had been a knife, and if he had inflicted some injury with it, this purposive action would have seemed a strong point against him.

Dr Jackson, however, himself admits that the form taken by a man's mental automatism during the paroxysm depends very much on his natural disposition. A savage and suspicious man would, when his highest faculties were temporarily in abeyance, be more likely to kill some one than to mix cocoa for his sister-in-law. Indeed, the actions performed during a state of unconsciousness are sometimes exactly those which would have been performed if the individual had been in full possession of his faculties. The patient last referred to had on another occasion ordered dinner at an eating-house, when his mental condition underwent a change, and he remembered nothing more until he found himself at his desk in the office, feeling rather confused. He had to go to the place and ask whether he had had his dinner, and he then found that he had eaten it and paid for it, and that neither landlady nor waiter had noticed any peculiarity about him.

In other cases the patient goes through ordinary actions in an absurd way. Thus another of Dr Jackson's patients, while in an omnibus, was one day observed to blow his nose upon a piece of paper, and when he got out he gave the conductor £2 10*s.* instead of the usual coppers. The same physician had a patient, who one day found the extinguisher of a candle in his waistcoat pocket. For some years he invariably looked at his watch after each fit, and the supposition is that he must, in an attack, have mistaken the extinguisher for his watch.

Falret long ago remarked that an impulsive tendency to wander about is characteristic of this form of insanity. Hence the name used by some French writers of *Folie circulaire.* Trousseau gives some instances. A gentleman was attending a literary meeting at the Hôtel de Ville, when he ran out and walked for some minutes on the quays, avoiding with success both the carriages and the passers by. When he recovered he found that he had neither great-coat nor hat; he returned to the room and resumed, with a perfectly lucid mind, the historical discussion in which he had before

taken an active part. The same gentleman, being a magistrate, was presiding at a provincial tribunal, when he suddenly got up, muttered a few unintelligible words, and went into another room. He was followed by the usher, who saw him make water in one corner, after which he returned to his seat. Another patient of Trousseau's, an architect, used often to have an attack while walking across a narrow plank at a height from the ground. He never met with any accident, although he would run rapidly over the scaffolding, shrieking out his own name in a loud abrupt voice. A moment afterwards he would resume his occupation and give orders to his workmen, without any recollection of what had occurred to him.

In attacks of this kind the state of the patient is often said to be that of a man walking in his sleep. True somnambulism (when it is not a manifestation of hysteria) has every claim to be regarded as a member of the group of paroxysmal neuroses (cf. *infra*, p. 819).

The same is the case, too, with the "night terrors" to which some children are liable, who start up an hour or two after going to sleep, screaming with fright, and unable for some minutes to recognise the parents or nurse. That these various attacks should occur only at night is nothing more than occurs in many cases of epilepsy itself.

MEGRIM.*—*Definition and nomenclature.*—An attack of this disease in its most typical form begins with a peculiar dimness of sight; after a while the fingers of one hand become numb and tingle, or the patient feels some difficulty of speech; presently the power of vision is restored, but pain in the head comes on, which may last for some hours, and, before it ceases, vomiting often takes place. The same succession of symptoms recurs again and again at more or less regular intervals.

Great confusion prevails with regard to the nomenclature of this complaint. Many names for it are in use, but each of them is properly applicable to only one of the various forms it may assume, and several of them would naturally be taken to include other and distinct affections.

The pain is often limited to one side of the head. To such cases the name "hemicrania" has been applied since the days of the old Greek writers; and that word has undergone corruption into the French *migraine* and the English *megrim*. In strictness, therefore, these terms are inapplicable to a case in which both temples ache at the same time; but such cases constitute the majority, and cannot be separated from the others. Still less can those be isolated in which the pain is intense and limited to a single point, as if a nail were driven into the skull—*clavus hystericus*.

Lastly, we have the English terms *bilious headache*, *sick headache*, and *sick giddiness*. But many cases present no indication of gastric or hepatic disorder; and there is a different form of headache far more closely related to dyspepsia and constipation. If we can forget its etymology, the word megrim or migraine is the most suitable general term, and Dr Liveing thus adopted it.

Symptoms.—There is one symptom of megrim which invariably precedes all the others if it occurs at all; this is the *affection of sight*. The patient first notices that he cannot see distinctly some part of what he is looking

* *Synonyms.*—Hemicrania (ἡμικρανία), whence *Fr.* Migraine, and our vernacular term, "the Megrims."—*Germ.* Hemicranie.—Paroxysmal sick headache—Bilious headache (in part)—Clavus hystericus—Hemicrania periodica and Hysteria cephalica refer rather to supra-orbital neuralgia than to Megrim.

at. The portion of the visual field which is thus blotted out is at first very small. It may be at the exact centre of the field, but more generally it is a little to one side of it, so that in reading from a printed page he has to glance slightly away from the word he wishes to see. The dim spot is not black, but seems like a faint cloud, of the same colour as the rest of the surface upon which the eyes are directed. It quickly begins to enlarge, and gradually overspreads more or less of one lateral half of the field. This symptom is known as Hemiopsia (cf. *supra*, pp. 573, 618, 682). Dr Hubert Airy has proposed to call it *teichopsia* (τεῖχος, a city wall; ὄψις, vision), from the peculiar zigzagged outline, with angles like those of a fortification, which often marks the edge of the blind half of the field. The surface within seems to have an undulatory motion which has been compared to that of a boiling liquid; and the angles appear to flicker or to revolve. The form of the cloud is originally oval, but as it grows bigger a gap forms in the side towards the centre of the field, so that it becomes horseshoe-shaped. One part of the curve then seems to touch the point of exact vision; and in this the angles are much smaller and closer together than in the other part, which spreads away into the outer region of the field. Sometimes the cloud is uncoloured; sometimes it presents brilliant gleams of red, blue, and other colours. If the eyes be closed, or if the person should go into a dark room with his eyes open, the whole figure appears to be faintly luminous. As it increases in size, the central part gradually clears up and accurate vision is regained. On a printed page, for instance, a few letters can now be plainly recognised in the midst of the glimmering horseshoe-like curve. Before long the latter likewise disappears, and the patient can see as well as ever. The whole process occupies from ten to twenty minutes, or at most half an hour.

While the oval cloudy patch and its zigzagged border are visible, they are seen in their minutest details by both eyes alike. To this rule a single exception has been recorded by Sir John Herschel, who once satisfied himself that his left eye was alone affected. But, as Dr Airy remarks, everyone is at first inclined to suppose that one eye only is concerned, namely, that of the side on which part of the visual field is blotted out. This fact, that the impairment of vision is referred to both retinæ, proves that the seat of the affection is above the optic chiasma. The limitation to one half of the field is just what we should expect from our knowledge of the anatomy of the nervous centres, the halves of which are in great measure isolated from one another, each communicating with corresponding halves of the two eyes. Sir John Herschel, indeed, has stated that in his own person he once observed "the shadowy pattern of a fortification which passed completely across the field of vision from left to right." If this was so, we must suppose that on that occasion the disturbance which is the cause of the affection extended over to the opposite side of the brain at an early period of the attack. We shall see that at a later stage this is not unusual.

In a few cases, as soon as natural vision is restored, the attack is at an end. But in the great majority a more or less severe headache comes on at this period. It is said that a precisely similar headache may also arise without having been preceded by any disturbance of sight.

A curious circumstance in regard to the visual affection is that some of the best and most careful descriptions of it have been written, not by physicians, but by astronomers and natural philosophers. Wollaston, Arago, Sir David Brewster, Sir John Herschel, Sir Charles Wheatstone, Du Bois

Reymond, Sir George Airy, and Professor Dufour, of Lausanne, were all liable to this paroxysmal defect of sight, and all carefully noted its phenomena; no similar malady has, within the present century, been the subject of two papers admitted into the 'Philosophical Transactions,' as well as of communications to the 'Philosophical Magazine' and other scientific journals at home and abroad. It may be a question whether persons who are not accustomed to employ the eyes for minute observation would notice the dimness of sight, or mention it to their physician. Indeed, when it begins at some distance from the centre of vision it is sure to be overlooked, unless the patient's attention is specially directed to it. This may, perhaps, be the reason why Professor Du Bois Reymond does not mention it in describing megrim from his own experience.

The *pain* of sick headache varies greatly in severity, both in different cases, and in the same case at different times. It commonly begins at some one spot in the brow or temple and gradually spreads over these regions. Sometimes it remains confined to a single point, which is generally over the frontal or parietal bone on one side. It was to these cases that the special name of *clavus* was formerly applied. A strict limitation, however, is very exceptional. According to Dr Liveing, it is not even the rule that the headache should keep to one half of the head. He finds that in the majority of cases it affects the whole forehead and both temples, although with more severity on one side than on the other. It often extends to the orbit, and is referred with special intensity to the back of the eye. More rarely it passes behind the ear to the occipital region. Some writers describe it as of a stabbing, cutting, or boring character; others as throbbing, and increasing with each beat of the heart. It is augmented by every bodily movement that the patient makes, by exposure of his eyes to light, and by every noise that he hears. He therefore lies down, and keeps the room as dark and quiet as possible. But Dr Wilks knows of one patient in whom the recumbent posture aggravates the pain, and who will sit up all night rather than lie down until the attack has passed off; and Dr Liveing speaks of cases in which the pain is so intolerable that the sufferer cannot lie still, but is obliged to get up and move about. It has been said that the patient can sometimes make the visual affection more marked, if not increase the headache, by lying on the side opposite to that on which the dimness of sight is observed; but this seems to be seldom the case.

The headache scarcely ever remains long at the same pitch of intensity. Generally it goes on gradually augmenting in severity until it reaches a culminating point, after which it begins to decline. Its increase is usually steady, but sometimes this takes place by fits and starts.

When the pain becomes intense the patient begins to feel *nausea;* and presently he retches and vomits. Anything that the stomach contains is rejected, including sometimes a considerable quantity of undigested food. If it is empty, the retching is ineffectual, or some mucus at first is brought up, and afterwards a bilious fluid. After the occurrence of free vomiting the pain often quickly passes off, and thus many patients regard it as curative, saying that they get well as soon as they are sick.

In some cases, however severe the headache may be, sickness seldom or never occurs. The pain gradually passes off of its own accord; but often it lasts for the rest of the day; the patient at length becomes worn out and drops off to sleep; and when he wakes on the following morning he finds only a slight soreness of the forehead or temple left. Some persons

lose the pain if they can sleep for a short time, even during the early part of an attack. Dr Liveing mentions a gardener who, if he could at the onset of an attack leave his work and lie down under the shade of a tree, would wake at the end of half an hour as well as ever. Lastly, in some rare cases the complaint terminates by epistaxis, by a copious secretion of tears, by profuse perspiration, or by an abundant flow of pale urine.

Less common symptoms.—An attack of megrim may be attended with exceptional symptoms. In certain cases, which are generally of considerable severity, common *sensation* is impaired in one hand, especially towards the ends of the fingers; and a feeling of tingling, thrilling, or formication may also be experienced. Sometimes the surface of all one arm and of the corresponding leg seems to have gone to sleep, or numbness may extend to the mouth, the lips, tongue, or throat. Dr Liveing says that all these parts are affected bilaterally. Dr Anstie noticed in his own person that, even in the intervals between the attacks of pain, the power of distinguishing impressions was permanently less in the skin round the inner angle of the right eye than on the opposite side; during and soon after the paroxysms the impairment of sensation was greater, and affected a larger area.

Occasionally *muscular power* seems to be more or less distinctly impaired. Ptosis and strabismus from paralysis of one of the recti muscles have each been present in cases which have been regarded by good observers as megrim. Some patients have been known to drop what they were carrying in the hand; but, as Dr Liveing suggests, this may be due to a loss of the sensations which should guide the muscles.

Another symptom which is occasionally present is *giddiness* or vertigo.

Again, in some cases the faculty of *speech* is disordered. The patient has difficulty in finding the expression which he wishes to use, or in constructing a coherent sentence. He may substitute one name for another; and an instance is recorded by Dr Liveing in which not a single word could be uttered. This may occur without any confusion of thought, or the patient may be painfully conscious that his memory is failing him, and that his intellect is embarrassed. Hallucinations are very rare; but there is often much mental depression, with a vague sense of anxiety and dread.

Such serious symptoms are uncommon, and generally commence before the headache, and after the affection of sight has continued for some time.

An oppressive *drowsiness* is sometimes noticed, so that the patient lies half unconscious, not heeding when he is spoken to; but this symptom rather accompanies than precedes the pain in the head.

In some instances there are indications that the vaso-motor nerves are implicated. The temporal artery becomes enlarged, and its tortuosities are much more plainly visible on the affected side of the head; it feels hard and like a cord to the touch. The conjunctivæ may be much reddened. The *pupil* is sometimes altered in size, but observers are not agreed as to the character of the change. Du Bois Reymond says that in himself it is always dilated; Piorry and Latham describe it as being contracted. The eyeball is said to appear retracted in some cases. In one instance Möllendorff found with the ophthalmoscope that the background of the affected eye was of a bright scarlet colour, the optic papilla red and œdematous, the central artery and the veins enlarged and tortuous. These facts seem to indicate that vaso-motor disturbance is concerned in the production of migraine.

After the subsidence of the paroxysms, certain very curious *trophic*

changes are sometimes observed, which can only be ascribed to an interference with their nutrition consequent on the nervous disturbance. One such change is a localised *greyness of the hair*. Anstie relates that when he himself had a severe attack of megrim the eyebrow would show a distinct patch of grey opposite the supra-orbital notch, but that subsequently the individual hairs regained their natural colour. He found that as many as eleven out of twenty-seven patients showed more or less greyness of the hair of the forehead and temple on the side on which they suffered most pain. In other cases the hairs become brittle or fall out. Some years ago the author was asked by a student to examine the hairs from his eyebrows with a microscope, to see if any fungus was present. More than half of each eyebrow, at its outer part, had become denuded of hair; and this condition was more marked on one side than on the opposite. On inquiry he was found to be liable to migraine, and it was more severe on the side on which the eyebrow was more deficient. In a few weeks, under treatment for the neurosis, the hair began to grow again.

According to Anstie, more or less thickening of the tissues occurs in many cases as the result of repeated attacks of migraine. He also speaks of iritis, glaucoma, opacity and ulceration of cornea, as resulting from neuralgia of the fifth nerve; but it is not certain whether such effects occur in cases of true recurrent migraine. An eruption like erysipelas ought, however, to be mentioned; for Anstie relates more than one instance in which a patient suffered from two or three successive attacks of this kind along with neuralgic pain. The upper eyelid is sometimes much swollen.

Another affection which is frequently consecutive to megrim is *xanthelasma* of the eyelids. It always begins near the internal canthus, generally in the upper lid; and (according to Mr Hutchinson) it constantly appears on the left side earlier than on the right. This observer found that most of the patients in whom he noticed xanthelasma of the eyelids had suffered from frequent sick headaches, some of them severely. This cutaneous affection seldom appears before the age of thirty-five or forty years.

The general *circulation* is interfered with in severe attacks of migraine. Möllendorff has found the beats of the heart reduced to fifty-two or even forty-eight per minute. The pulse at the wrist becomes small and contracted. The hands and feet are cold. The face is pale and haggard, and dark borders appear round the margins of the orbits.

The side affected.—It seldom if ever happens that megrim, in all its attacks in the same patient, attacks the same side of the head. As a rule, there is one side rather than the other which is more apt to be affected; but Tissot mentions the case of a lady who had it alternately on each side with great regularity. Wollaston and Sir George Airy may also be mentioned as having been equally liable to the affection on either the right or the left side. Dr Latham describes the headache as beginning, and as more intense, on the side *opposite* to that on which the dimness of sight is noticed, but Dr Liveing deduces from the observations which he has collected that they often both occur on the same side. Both authors say that when the affection of sight and the numbness in the fingers are present together, it is on the same side of the body. Lastly, Dr Liveing has pointed out the fact —interesting in connection with what has been said as regards aphasia from organic lesions—that when the speech is interfered with in migraine there is very generally numbness in the fingers, and that this always affects the right hand, either alone or in association with the left. He has not met

with a single instance in which sensation was impaired in the *left* hand only, together with any affection of the speech.

Pathology.—Megrim is undoubtedly one of the paroxysmal neuroses. As regards its anatomical seat, the disturbance which causes the affection of sight must occur somewhere above the optic chiasma. Indeed, this was long ago pointed out by Wollaston, who thought that the hemiopia which he described would probably be found to arise in the thalamus of one side. As Dr Liveing remarks, the only correction that this statement seems to require at the present time is to add the corpora quadrigemina, and to include within the area of disturbance in a fit of megrim the ganglia of the sensory nerves, down to the nucleus of the vagus. The order in which the symptoms follow one another in the several attacks renders it likely that the affection generally starts in the corpora quadrigemina or geniculata, and passes downwards and backwards along the sensory tract. The numbness and tingling may possibly be due to disturbance in the thalamus, and disorder of speech and impairment of memory seem to show that the "storm" has spread upwards to the convolutions. That it may also extend to the opposite side of the brain appears to follow from the facts that the numbness in the tongue and throat is sometimes bilateral, and the headache frequently so.

As to the exact nature of the change in the sensory tract which gives rise to migraine, nothing definite can at present be stated. The most recent hypotheses refer it to vaso-motor disturbance. Thus the theory of Dr Latham (1872) is that in the early stage the affected side of the brain is anæmic; that the contraction of the blood-vessels of this hemisphere is itself due to a morbid activity of the sympathetic nerve; and that this in its turn results from a defective control or inhibition on the part of the cerebro-spinal system, which he supposes to be enfeebled. In the stage of headache he supposes that there is a secondary hyperæmia, consequent upon exhaustion of the vaso-motor apparatus. Du Bois Reymond had previously (1860) maintained that, at least in his own case, migraine was due to tetanus of the muscular coats of the vessels of the affected side, in the territory of the cervical portion of the sympathetic. On the other hand, Möllendorff (1867) and Wilks (1869) believe that the complaint is caused by paralysis of the very same nerves, with dilatation of the vessels and consequent hyperæmia.* The former writer lays great stress on the fact (which had been pointed out nearly a century ago by Dr Parry, of Bath) that compression of the carotid on the affected side of the head often removes headache as if by magic, though only for a time. But diminishing the blood supply to one side of the brain may very well suspend for a time the disturbance which is felt as pain, and yet that disturbance may not have been caused by an overflow of blood. Moreover, as Dr Liveing points out, the statements of different observers with regard to the condition of the pupil are so diametrically opposed that no other inference seems possible but that it must really differ in different cases; while as for the dilatation of the temporal artery, the flushing of the face, the redness of the conjunctiva, the injection of the fundus of the eye, none of them is constantly present; so that the only possible conclusion seems to be that all these vaso-motor phenomena are only accidental, not essential characters. As Dr Liveing remarks, there is a clear analogy between the paroxysmal neuroses, and certain minor consensual and automatic movements, such as sneezing,

* Dr Eulenburg thinks that both theories are right, and that certain cases are due to spasm, others to paresis of the vaso-motor nerves.

coughing, and gaping, to which may be added ejaculatio seminis—a short convulsion, as it has been termed, and in certain cases accompanied by a true epileptic attack; yet no physiologist thinks of referring any of these to vaso-motor disorder.

Thus it would seem that at present we can form no clearer conception of an attack of migraine than that it is a "nerve-storm," or an "explosive discharge" of nervous irritability.

Ætiology.—The *hereditary* character of megrim is well marked. Dr Liveing found that in twenty-six cases out of fifty-three it was said to be a "family complaint;" and the twenty-six patients in question had among them forty near relations who were liable to it. In many cases it is transmitted without the slightest change of type, and sometimes it passes from a parent to those children only who in other respects resemble him. Sometimes, however, a daughter inherits megrim from an epileptic mother.

As to the relative liability of the sexes, women appear to be more prone to this complaint than men. Eulenburg believes that five women have hemicrania to one man.

The *age* at which it commences is generally about the seventh or eighth year, at the beginning of the second dentition, but sometimes it is the period of puberty, and sometimes that of early adult life. It rarely occurs for the first time in a person over twenty-five or thirty. The more marked the hereditary tendency the greater the probability of its beginning in childhood. At about the age of thirty, persons often suffer from it much more severely than before; and, as Anstie remarks, at this period the attacks cease in many instances to be accompanied by vomiting, so that the complaint is no longer regarded as mere "sick headache," and the patient consequently seeks medical advice, perhaps for the first time. When fifty years of age are reached, or somewhat earlier, the liability to migraine commonly ceases, and old people seldom suffer from it.

In some persons the attacks of migraine recur with regular periodicity. The period is sometimes a fortnight, sometimes a month, sometimes longer still. There are, however, cases in which it is much shorter.

The immediate exciting cause of the paroxysm is often excessive fatigue. Thus, a bank clerk had an attack regularly every week-day, but was free on Sundays; and a governess, under the author's care, had a headache every night. In cases of this kind some of the more characteristic features of the complaint are apt to be missing; but their relation to true migraine can often be established by the account which the patient gives of his previous state of health. Further observation may perhaps show that a headache which is persistent may grow out of the paroxysmal affection. If so, such a case would be strictly parallel to one of epilepsy, followed by the status epilepticus.

In the cases just referred to the complaint may return every day, or every other day, as regularly as the paroxysms of an intermittent fever; and this fact, together with the striking therapeutical influence of quinine, often makes it difficult to exclude the possibility of miasmatic poisoning. But these circumstances, taken by themselves, are far from justifying the conclusion that a case of migraine really deserves the name of "brow ague." It is probable that even in districts where malaria prevails, migraine and other forms of neuralgia are often wrongly ascribed to that cause. But it appears to be certain that it is sometimes really the cause; and in some parts of Spain a miasmatic migraine is said to be endemic. Dr Macculloch has stated that

this kind of headache may occur as a substitute for ague during the whole of one relapse of the disease, and that he has seen a "double tertian" ague, in which the headache and the ague fit occurred regularly on alternate days.

In England, it is universally believed that migraine, instead of being essentially a nervous malady, is the result of "*bilious*" disorder; and when the attack is accompanied by vomiting, this is supposed to expel a "*materies morbi*," in the shape of vitiated bile. Until one has happened to discuss the matter with some patient of intelligence, one can hardly conceive how firmly fixed is the belief in question. The truth is that it is a relic of one of the most ancient doctrines in the history of medicine, that of the four Cardinal Humours, one of which was "yellow," and another "black" bile.

Although it is certain that migraine is never solely due to disorder of the chylopoietic viscera, there is no question that some error of diet is often the direct exciting cause of an attack in a person who is liable to it. No doubt the diffused headache and giddiness which are apt to be more or less present in persons who suffer from dyspepsia or from the so-called congestion of the liver have been often confounded with true migraine. But what is conclusive as to the reality of the influence of improper food is the fact that some persons at least can always bring on an attack of the latter disorder by eating particular articles of diet towards the end of the interval between one paroxysm and another; whereas, for a few days after they might partake freely of the very same things without suffering in any way. Dr Fothergill nearly a century ago stated that he had found nothing more apt to cause "sick headache" than "melted butter, fat meats, spices, meat pies, hot buttered toast, and malt liquors when strong and hoppy." A medical man who had suffered all his life from the complaint told Dr Liveing that he could never take the smallest quantity of wine nor eat the smallest fragment of pastry without bringing on a headache. Many persons speak of butter and pork as particularly frequent exciting causes of migraine; and, making every allowance for the influence of preconceived opinions, it seems likely that such statements are not entirely imaginary.

In women, again, the recurrence of the *catamenia* is often an exciting cause of attacks of migraine, which, perhaps, generally precede the flux, but sometimes accompany or even follow it. Not infrequently each monthly period brings with it a series of more or less distinct paroxysms. Dr Liveing relates the case of a woman who was very liable to the complaint when menstruating, but who throughout repeated pregnancies was always entirely free from it. This writer also mentions an instance in which the headache and the catamenial discharge recurred simultaneously at fortnightly intervals.

But *fatigue* is a far more important exciting cause of migraine. In some persons a straining effort, such as lifting a heavy weight, will bring it on, and in others the exertion of running is apt to have the same effect. Many women are exceedingly liable to be attacked after a hard day's washing, or after a long walk. Another frequent cause is severe mental work; but, above all, anxiety and emotional disturbance. A long railway journey is apt to be followed by a paroxysm in some ladies, and in others merely driving in the streets of London has the same effect. Many persons always have a sick headache after a day's sight-seeing, or after passing an evening in a crowded concert-room or ball-room; and in some susceptible patients an attack may be brought on by glaring lights, loud noises, or strong odours. Dr Airy mentions the case of a person in whom the peculiar affection of sight was

occasionally caused by looking at a striped wall-paper or a striped dress; and Sir John Herschel found that he incurred it as the result of allowing his mind to dwell upon a description of its symptoms.

In several of the conditions already alluded to as exciting causes of migraine, one element is *visual exhaustion*; and this is true not only of over-study, but also of railway travelling and the like. Many years ago, Piorry propounded the theory that the complaint, or at least one variety of it, was the result of irritation of the optic nerves, from straining efforts to see very small objects, or from want of care in regulating the amount of light. This view is quite untenable if applied to all cases of sick headache. But it is perfectly true that when the eyes are structurally imperfect the forced effort to use them may be the immediate cause of attacks of migraine. The defects which lead to this result are chiefly those of the transparent or refracting media of the eyes; their direct effect is the production of spasm of the ciliary muscles, and with this is associated an irritation of nervous filaments, which may diffuse itself over a wide area within the distribution of the fifth nerve. Every practitioner now knows that hypermetropia is a frequent cause of attacks of dimness of sight, headache, and giddiness, which recur when the eyes are used for near work for any length of time, particularly under artificial light. A student suffering from similar symptoms discovered that they were the result of the employment of too powerful concave glasses, which he had chosen without proper advice, in order to correct a moderate degree of myopia, and which he wore even when reading or writing. In this connection, too, astigmatism must not be overlooked.

A less frequent cause of such symptoms is weakness of the internal recti muscles. The author once saw a bank clerk who had previously been compelled to give up work for a period of two or three months on account of cerebral symptoms. These had been thought very serious; but on examination by Mr Higgens it was discovered that the internal recti muscles failed to make the eyes converge properly upon near objects; and when suitable glasses were supplied he soon lost all his complaints. In practice, therefore, one should make it a rule never to prescribe for any kind of frontal headache without eliminating the possibility of its being caused by imperfection of the eyes.

Lastly, affections of the *teeth* must not be overlooked as causes of migraine, at least if clavus be included as a form of it (see p. 388). Mr Salter records the case of a young lady, who for eight years was subject to attacks of headache, confined to a space of about the size of a crown piece, rather to the left of the vertex. They sometimes recurred three or four times a week, beginning after breakfast and lasting all day; they were attended with great prostration. The affected spot became hot, and pressure with the hand gave relief. At length the patient fancied that the left upper canine tooth, which was known to be impacted in the palate, was in some way connected with her sufferings. It was removed, and she never afterwards was attacked by the headache.

Diagnosis.—In its typical form migraine presents little or no difficulty of diagnosis. In one or two cases syphilitic periostitis of the margin of the orbit has produced recurrent pains of somewhat similar character; but this could not escape the notice of any but a careless observer.

When the phenomena of the attacks are ill-developed, however, one may not find it easy to determine whether they belong to megrim, or to some other neurosis.

Several months before his death the late Dr J. J. Phillips had a

severe attack of headache, attended with marked aphasia. When he had recovered, the author one day happened to discuss with him the question whether it could have been of the nature of migraine; but his fatal attack of apoplexy, which doubtless was the result of embolism, began in precisely the same way, and with the same symptoms. It seems doubtful whether Dr Liveing is right in regarding as a mere paroxysmal neurosis a case in which attacks of loss of speech and right hemiplegia recurred, persisting for a week or more at a time.

Relation to other neuroses.—Migraine is so common a complaint that one can hardly attach much importance to the mere fact that some other neurosis occasionally develops itself in those who are subject to it, as indicating that there is any real relation between them. Dr Liveing, however, believes that a transformation sometimes occurs between megrim and epilepsy; and he relates cases in which persons who had suffered from the former afterwards became affected with the latter disease; but it is to be noted that some of them had relations who were epileptic. This writer also refers to an instance in which migraine became replaced after a certain period of asthma; and to another in which a constantly recurring gastralgia disappeared, and was followed by a typical migraine, while this in its turn was succeeded by a kind of spasmodic croup. He also relates a case in which attacks of sick headache were followed after a time by angina pectoris; and another in which insanity developed itself.

Probably no medical man can have suffered again and again from migraine without the thought being forced upon him that such attacks must indicate some serious defect of cerebral organisation; and Dr Liveing quotes Calmeil as having remarked that both the intellectual faculties and the moral disposition of the patient are sometimes impaired by the repeated occurrence of migraine. He also refers to the cases of Parry and Wollaston, both of whom, after having long been subject to this complaint, died of organic cerebral disease. But such results are extremely rare and probably accidental.

Treatment.—This includes: (1) the management of the patient during the intervals between the attacks, so as to prevent their occurrence or diminish their severity; and (2) the treatment of the paroxysm.

(1) Under the first head hygienic measures are of primary importance. One must insist upon the importance of daily exercise, short of fatigue, in the open air; one must prohibit an excess of mental work, and take care that the patient is as far as possible shielded from domestic anxiety. The state of the digestive organs must be carefully inquired into; and due weight must be allowed to any indications of dyspepsia or lithæmia, or to evidence that the attacks are brought on by errors of diet. But in many cases it is a mistake to restrict the patient too closely to what is termed plain food. Rather he should be advised to take a freer quantity of fat, in the form of butter or cream. Alcoholic beverages, however, should generally be avoided, or sparingly indulged in. In severe cases a change of climate is often advisable, and particularly a sea voyage.

One of the good results which may be anticipated from the discussions which have taken place as to the nature of migraine is that those who have the medical charge of young people will look out for the early manifestations of the complaint during childhood or puberty, and insist upon the avoidance of over-study and of undue excitement in those who seem likely to suffer from it. Until recently migraine was almost universally

looked upon as being at once incurable, and almost unworthy of notice on the part of a physician. But the truth is that if systematically taken in hand it is very amenable to treatment; and those who suffer from it know best how serious a matter it is to them, interrupting, as it frequently does, all the engagements, the pleasures, and the duties of life.

Among medicinal agents Dr Liveing says that he has sometimes found the regular administration of belladonna and hyoscyamus of great service. Cannabis indica also may frequently be prescribed with decided advantage; but bromide of potassium is perhaps still more generally useful.

In many cases the preparations of iron (especially the peroxide and the saccharated carbonate) are very serviceable, and this even though there may be no anæmia. Strychnia in small doses (one twentieth of a grain) is said to be another valuable remedy. The author has prescribed arsenic with marked success; it was long ago recommended by Dr Bright and Sir Thomas Watson. Quinine is said to be less serviceable than it is in many forms of neuralgia, except in cases of malarious origin.

There are other medicines of which the good effects are less readily intelligible. One such is iodide of potassium. Dr Todd is said to have found this more successful than anything else; and Dr Liveing mentions a case in which the attacks were so frequent and severe as to render the patient's life a burden to him, and in which five grains of the iodide three times a day set him almost free from them. In other cases chloride of ammonium seems to answer better than anything else. Valerian and valerianate of zinc are said by Dr Liveing to be sometimes of great value.

(2) The treatment of the paroxysms of migraine must necessarily depend on their severity; it is only in very violent attacks that the patient is likely to seek for medical advice. Of his own accord he will keep in a darkened room and maintain absolute quiet. If the feet be cold, they should be wrapped in blankets, and a hot bottle should be placed in contact with them; or they may be immersed in hot water to which some mustard has been added. Hydrate of chloral may then be administered in a dose of from twenty to thirty grains. Dr Anstie speaks of it as being of the greatest possible value in quickly bringing sleep to the patient, who when he wakes up may be free from pain. In some cases, and particularly when connected with neuralgia of dental origin, butyl-trichlor-aldehyde-hydrate ("croton-chloral") is still more decidedly efficacious. Bromide of potassium also is very useful; a scruple, or half a drachm, taken when the sight begins to be affected, sometimes cuts short the attack. At this period a cup of strong tea or coffee is in some persons capable of producing the same effect; even sipping hot water gives relief in some cases. Guarana—prepared in Brazil from *Paullinia sorbilis*—has been recommended by Dr Wilks: half a drachm of it may be taken, mixed with water; or about twenty or thirty minims of the liquid extract. Even this, however, is sometimes altogether useless. Dr Anstie mentions the extract of cannabis indica in a dose of a quarter to half a grain as being very serviceable in the migraine of the young; he says the dose should be repeated in two hours if sleep be not obtained. In some cases a full dose of brandy or a glass of sherry or champagne is very effectual; but there is always danger in allowing a patient to fly to such remedies for the purpose of allaying pain.

Antipyrin, in doses of from two or three to seven or ten grains, has lately been much used for sick headaches, and in some cases it is undoubtedly

efficacious. The patient should take it in hot brandy and water, and lie down for half an hour before taking a second or third dose.

Locally some measure of relief may be afforded by the pressure of a handkerchief tied tightly round the forehead.* Dr Liveing mentions a case in which plunging the head into cold water was often effectual. In some cases it has been found advantageous to apply to the seat of pain a little piece of cotton wool, on which a few drops of ether have been poured, and to cover it with a watch-glass; and bisulphide of carbon has been used in a similar manner. Some patients find great relief from rubbing a stick of menthol into the skin over the seat of pain. Trousseau speaks highly of the application of extract of belladonna to the painful temple; and Anstie says that a diluted ointment of veratria is often serviceable.

Another measure which appears sometimes to be highly successful is the application of the constant galvanic current. Anstie recommends that it should be passed from one mastoid process to the other. Only three or four cells should at first be employed, and never more than ten; it should be applied for but half a minute at a time, and repeated once or twice a day. Giddiness is very apt to be produced unless it be used with great caution.

* "When your head did but ache,
I knit my handkerchief about your brows."
King John.

NEUROSES OF THERMIC AND TOXIC ORIGIN

SUNSTROKE, AND THE EFFECTS OF DRINK AND OPIUM

"And when the child was grown, it fell on a day, that he went out to his father to the reapers. And he said unto his father, 'My head, my head!' And he said to a lad 'Carry him to his mother.' And when he had taken him, and brought him to his mother he sat on her knees till noon, and then died."—2 *Kings*, iv.

"O that men should put an enemy into their mouths to steal away their brains!"
Othello.

"I paused seasonably: but with a difficulty that is past all description. Nothing short of mortal anguish in a physical sense, it seemed, to wean myself from opium; yet, on the other hand, death through overwhelming nervous terrors, death by brain fever, or by lunacy, seemed too certainly to besiege the alternative course."—DE QUINCEY.

HEAT-STROKE.—*Origin and pathology—Symptoms of the cardiac and cerebro-spinal forms—Prognosis—Sequelæ—Diagnosis—Treatment.*

ALCOHOLIC NEUROSES.—*Delirium tremens—its symptoms and course—prognosis and treatment—Chronic alcoholic poisoning—its symptoms and course—antecedents—diagnosis and treatment.*

NARCOTIC NEUROSES.—*Opium-eating—The morphia habit—Symptoms and results—Treatment—Neuroses due to other narcotic drugs.*

BEFORE concluding the long series of functional disorders of the nervous system, we must find room for two which are both acquired, and are both the direct result of external disturbing causes, namely, heat and alcoholic drinks. The ætiologically allied neuroses produced by metallic poisoning have already been noticed (pp. 519—523), but those which attend the abuse of opiates will be most conveniently discussed in the present chapter.

HEAT-STROKE.*—That exposure to intense heat is sometimes followed by alarming or fatal cerebral symptoms is now well known. The affection is not uncommon in the hotter parts of India, and it now and then occurs during the summer in England. It is also frequent during the hot season in New York and in Australia. The common term "Sunstroke" is inaccurate, for the direct rays of the sun are not required to produce it; it often comes on at night, when the temperature is very high, particularly when a number of persons are crowded together, as among soldiers in barracks, or sailors or emigrants on board ship. Sir Joseph Fayrer mentions it as occurring to the stokers in the engine-rooms of the Red Sea steamers. *Heat-stroke* is therefore a better name.

As might be expected, this disease often attacks several men simultaneously or in rapid succession. Dr Maclean speaks of having seen a great many soldiers of the 98th Regiment struck down, of whom about fifteen died on the spot, in taking possession of a steep hill in China in 1842. But the men who suffer from heat-stroke appear to be never more than a minority of those who are exposed at the same time to the sun's rays.

* *Synonyms.*—Solis ictus—Insolatio—Sunstroke—Heat-apoplexy.—*Fr.* Coup de soleil—La calenture (in part).—*Germ.* Hitzschlag, Sonnenstich.

This depends upon the fact that the disease is due to the failure of those natural processes which should enable the human body to resist the action of excessive heat. Dr Maclean remarks that the closely shaven heads of the Chinese bear the hottest sun without ill effects, although it is true that they generally use their fans to keep up a free current of air about their faces. Sportsmen in India expose themselves to very high temperatures with comparative impunity so long as they take care to wear light clothes, to protect the head and the spine, and to abstain from stimulants. Thus it is of the highest importance that soldiers and others who have to bear fatigue in hot climates should be suitably dressed, and should have no accoutrements which can interfere with the play of the lungs. More than one observer has noticed that large and fat men are especially apt to suffer from sunstroke, and that exhaustion from prolonged exertion is a predisposing cause. Other factors which are believed to be often concerned in its development in those who are not directly exposed to the sun are overcrowding and defective ventilation. It would seem to be more likely to occur when the air is loaded with moisture, since this must interfere with evaporation from the skin; but there was extreme dryness at the time of an outbreak which Surgeon-General Longmore saw at Barrackpore. Europeans are supposed to be more likely to suffer from the disease when they have been only a short time in India, but there is good evidence that natives are by no means free from it.

Symptoms.—The phenomena of heat-stroke vary in different cases.

(1) There is a "cardiac" form. In this death may be almost instantaneous by syncope, the man falling down suddenly insensible and making only a few hurried gasping respirations. Dr Morehead also describes milder cases, in which a sense of prostration and faintness is experienced, with vertigo, dimness of vision, dilated pupils, and drowsiness. The patient can be roused by speaking to him, pinching him, or sprinkling his face with cold water. There is constriction of the chest, with sighing respiration, a sense of weight or sinking at the epigastrium, nausea, and sometimes vomiting. The face and lips are pale. The skin is generally cold and clammy, with the exception of the head, which is somewhat hot. The pulse is feeble, and it is generally slow. Under judicious management such cases often recover; but sometimes the pulse sinks, the breathing becomes more sighing and irregular, and death occurs, being perhaps preceded by convulsions. "Cardiac" cases seem to be met with only among those who are attacked in consequence of direct exposure to the sun's rays. When recovery takes place, it is complete and leaves no sequelæ.

(2) There is a "cerebro-spinal" form, of which coma is the principal feature. This often comes on gradually. It may be preceded by nausea and loathing of food, giddiness, congestion of the eyes, extreme debility, and (as more than one observer has noticed) an unusual frequency of micturition. "I cannot hold my water" is said to have been in a large number of cases the first thing complained of. Dr Maclean speaks of the attack beginning with a wild shout of laughter, or with a delirious attempt to escape from an imaginary enemy; headache, he says, is seldom present. When heat-stroke occurs in a man sleeping in barracks, what draws the attention of his comrades to him is often his stertorous breathing. He is then found to be already insensible, with contracted pupils, deeply congested conjunctivæ, and a quick sharp pulse. As far back as 1860 Dr Morehead pointed out that the skin continued pungently hot to the close

of these cases, and even for some time after death. More recently, since the introduction of the thermometer into practice, it has been shown that the condition is really one of hyperpyrexia; and, in consequence, the name of "thermic fever" has been proposed for them. Three instances of this variety of the disease occurred in 1876 at Bristol, and were fully recorded in the 'Lancet' by Dr Shingleton Smith and Dr E. L. Fox. One of them was in a man, aged forty-five, who had been turning a winch on board a steamship, exposed to the full glare of the sun, from 8 a.m. The temperature in the shade was from 92·3° to 96°. At one o'clock he felt unwell, and began to talk incoherently and to throw his arms and legs about. He had been drinking water freely. He was admitted into the hospital half an hour later, and he was then comatose, with pin-point pupils; the temperature was 107°; the pulse 160, weak and intermitting; the breathing laboured; the face not flushed; the skin sweating. At twelve minutes past two the thermometer registered 109°. Under energetic treatment the temperature gradually fell to 100·4°; the pupils became normal and afterwards dilated; but the pulse failed more and more until it could not be counted, and at 7.40 he died. On the very same day, another man, aged thirty-five, was taken into the same hospital in a state of only partial consciousness, with general muscular tremor, soon followed by convulsions and opisthotonos. His temperature was then 110·2°; his pulse was too rapid and too feeble to be counted. He died twenty minutes later, the thermometer at that time registering 111°. Two days afterwards, a third case was admitted at 2.30 p.m. in the person of a man, aged fifty-five, who had been driving a hearse, when he fell backwards, and the reins dropped from his hands. He was comatose; there was tonic spasm of the muscles of the legs; the pupils were contracted, but sensible to light. The temperature at 2.40 was 106·4°; at 2.50 it was 107°. The pulse was 141; the breathing was stertorous and at the rate of 27 in the minute. Under treatment the temperature quickly fell; at 3.25 it was 102°, at 3.40 it was 100°. In this instauce recovery took place. These three cases appear to have presented all the chief symptoms of "thermic fever," as it is described by those who are familiar with sunstroke in India.

Dr Morehead speaks of a "mixed form" of heat-stroke, in which the symptoms are a variable combination of those of the other two forms.

Sir Joseph Fayrer distinguishes syncopal, asphyxial, and hyperpyrexial varieties of heat-stroke as observable in India.

It is well known that *sequelæ* of a very serious kind are not infrequent. Those mentioned by Dr Maclean are persisting headache, either fixed or shifting; a chorea-like affection of the muscles, generally those of the forearms and hands; epilepsy, particularly in those who have inherited a tendency to that disease, or have had fits in youth; and mental weakness.

Fatal event.—When heat-stroke ends in the patient's death, this generally occurs within nine hours, but sometimes not until towards the end of the second day. The average mortality of the disease is estimated at from 45 to 50 per cent. Fatal relapses, after recovery from incomplete coma, are not infrequent; cases which seem to be doing well require to be carefully watched with the thermometer until the skin becomes moist and cool.

Anatomy.—The usual *post-mortem* appearances are those of congestion of the viscera generally. The blood is said always to remain fluid. In twenty-five cases of death, out of forty-eight of heat-stroke, among the troops at Assouan in 1886, Surgeon Douglas Hunter remarked the following condi-

tions after death :—intense lividity of the surface and ecchymosis of the conjunctivæ ; venous engorgement ; muscles of dark colour : in only one case was meningitis observed. When meningitis is produced by the sun's heat (pp. 701–2) the cases seem as a rule to be distinguished by there being an interval between the time of exposure and the onset of symptoms.

In 1856 there was in Guy's Hospital a sailor who four years previously, when crossing the equator, had been attacked by sunstroke in company with another man who died. He himself came to in a few hours, but he could not speak for a month afterwards, and for a time he lost the use of his right arm and leg. From all these symptoms, however, he had perfectly recovered ; and he was now suffering from renal dropsy, which at length proved fatal. At the autopsy Dr Wilks found the arachnoid opaque and marked with white spots, the ependyma granular, and an excess of fluid in the brain. It is to be noted, however, that the vessels at the base were much diseased, so that the other morbid appearances may have been unconnected with heat-stroke.

Diagnosis.—Heat-stroke is no doubt easy to recognise in most cases ; but this often is because the patient is known to have been exposed to a high temperature, rather than because the symptoms are in themselves distinctive. Maclean places reliance on certain characters of the pulse and of the respiration, and on the state of the skin and of the pupils in apoplexy, as serving to exclude that disease from consideration. Probably there need be no doubt as to the real nature of those cases in which there is hyperpyrexia from the very commencement. But this is not always present ; and probably a person attacked on a hot day in India with cerebral hæmorrhage, or embolism, would be exceedingly likely to have his case set down as one of "sunstroke," even by good observers, especially if there were no paralysis.

Apart, however, from the classical coup de soleil or thermic fever of India and the tropics generally, there is a very much milder malady which is probably due to exposure to the sun, and is most often seen in children in England. A boy, after playing in the sun, will come in complaining of feeling sick and faint ; he may perhaps vomit, looks very pale, and has a bad headache. Cool drinks and rest in a dark room generally set him right in a few hours.

It often happens that patients tell one that their complaints all result from a "sunstroke" which they had in England during the previous summer ; but such statements must be received with caution.

Treatment.—The most important remedy is the bold and immediate employment of cold. Ice should be applied to the head ; a stream of iced water may be passed through coils of elastic or leaden tubing (as used by Leiter for the purpose) in contact with the back and the chest ; cold water must be poured over the head and neck and chest for a few minutes at a time. If the patient can swallow he should be allowed to drink freely. In the one successful case at Bristol (which, however, was less severe than the others) Dr Fox, besides applying cold, injected a grain of quinine under the skin in five different places ; and at the end of half an hour the patient took ten grains of quinine by the mouth. Indian practitioners are agreed that venesection is likely to be injurious rather than useful. Dr Hunter recommends a large enema and a dose of calomel.

When, however, the circulation fails, hot-water bottles must be applied instead of, or in succession to, the ice ; and brandy or liquor ammoniæ (♏viij to ʒiss of water) may then be injected subcutaneously. Dr Maclean

says that the application of a blister to the nape of the neck, or to the shaven head, may be of service. Artificial respiration should be resorted to in the asphyxial form of sunstroke. When convulsions set in, the inhalation of chloroform is recommended.

For *sequelæ* of heat-stroke, occurring in a person living in a hot country, removal to a temperate climate appears to be the best treatment. Dr Maclean states that at Netley there are always some cases of this kind, and that they are often very obstinate. He has seen long-continued counter-irritation to the nape of the neck, and a course of iodide of potassium relieve permanently some patients troubled with severe fixed pain in the head; but in other instances these measures have altogether failed. He gives a favourable prognosis for those who come home with epilepsy under such circumstances, as he has usually found the fits subside.

ALCOHOLIC NEUROSES.—Intemperance in drink may affect the nervous system in two ways—acutely as a form of delirium, and in a more insidious but scarcely less dangerous manner.

*Acute alcoholic poisoning.**—In many cases the nervous symptoms of alcoholism take an acute form, and one that may quickly endanger life. They then constitute a disease which has for many years been known under the name of "delirium tremens." The earliest account of it appears to have been published in 1813 by Dr Thomas Sutton, of Greenwich; and the first writer to describe its course, when not disturbed by the administration of medicine, was an American physician, Dr John Ware, of Boston, in 1831; his work, based on the observation of nearly a hundred cases, is perhaps superior to any that has since appeared.

Symptoms.—The patient has for two or three nights been more disturbed by unpleasant dreams than usual, and has by day become more restless and tremulous. At length he is altogether unable to sleep, but as he lies awake his dreams still haunt him. Next morning he may again be rational, but his mind almost always wanders occasionally. Towards evening the delirium returns, and the second night is worse than the first. During the second day there is again a slight amelioration as compared with the night, but the mental disorder is now fully established and persistent.

The delirium in this disease is in many respects peculiar. The patient is not fierce nor violent, nor, on the other hand, is he depressed in spirits and dull in manner. He is loquacious and restlessly anxious to follow his accustomed vocation, but he sets about his business in a blundering manner, and his mind quickly wanders away to something else. If spoken to, he is sufficiently intelligent to answer, and for a minute or two he may converse rationally, but before long he starts off on some fresh topic. His friends probably endeavour to keep him in bed, but he is always wanting to get up and dress himself. Yet, if firmly opposed, he forgets his intention, at least for the time. He commonly has hallucinations of vision. Very often he fancies that rats and mice, or snakes, or cockroaches, are running or crawling over his bed. Or he may address persons who are really absent, but whom he supposes to be in the room. He looks suspiciously behind the curtains or under his pillow, or stretches himself out of bed to see if some one is not concealed beneath it.

* *Synonyms.*—Delirium tremens—Delirium potatorum.—*Fr.* Intoxication alcoolique.—*Germ.* Säuferwahnsinn.

His hands are in constant motion. He picks at the bedclothes, or grasps at imaginary objects. If asked to put out his tongue, it is very tremulous, and is quickly withdrawn; it is commonly moist, and more or less thickly coated. The pulse is quick, soft, and feeble. The skin is moist and often in a state of profuse perspiration. There is an entire absence of appetite and even of desire for drink. As was pointed out by Dr Bence Jones, the amount of phosphates in the urine is very greatly diminished.

Event.—The duration of these symptoms is not absolutely constant, but it is far more regular than is generally supposed. The disease almost always goes on for two and a half days without showing any tendency to subside, and then between the sixtieth and the seventy-second hours it comes to an end. Towards this period the patient is very apt to show signs of exhaustion. His pulse becomes more and more rapid and feeble. His face, at first flushed, is now pale and haggard; his pupils are widely dilated; his tongue sometimes, but rarely, becomes dry and brown.

It is, however, precisely at this time that a favourable change is to be hoped for. Towards the end of the third night the patient commonly falls asleep. At first he is still uneasy and restless, his breathing is irregular, and after an hour or two he may wake up for a little while. He soon goes soundly to sleep again; his breathing is now slow and deep; a profuse sweat breaks out over his body. After six or eight hours he awakes and is relieved. In the next twenty-four or forty-eight hours he sleeps almost continuously, and after this his restoration to health appears complete.

In some cases, particularly in those who have previously been in good health, and in whom the attack has been the direct result of a debauch, the attack terminates earlier—perhaps at the end of twenty-four hours. In other cases it lasts beyond the specified time. Dr Ware speaks of having once known it to extend to nearly six entire days. This is most apt to occur in those who have for a long time been habitually intemperate.

But the end of the disease is by no means always favourable. Sometimes a sudden attack of convulsions occurs, by which the patient is carried off; sometimes he becomes comatose, and sometimes he falls back in his chair and dies unexpectedly in a state of syncope.

Delirium tremens has a marked tendency to attack the same patient over and over again, unless he makes an entire change in his habits.

Ætiology.—So far as is known, delirium tremens never occurs except as the result of alcoholic intemperance; but it is often exceedingly difficult to discover how intoxicating liquors are obtained. Women, in particular, will suborn their servants or attendants, or procure tincture of lavender or sweet spirits of nitre from the chemist, or drink eau de Cologne.

At one time it was generally believed that instead of the immediate exciting cause of delirium tremens being the alcoholic stimulus itself, the disease was most apt to occur in persons who, from whatever cause, had been suddenly deprived of the drink to which they had been accustomed. There can be no question that before the attack the patient has sometimes left off drinking for a few days; and, again, that it often shows itself in those who have been kept for a time without stimulants, in consequence of their having broken a limb or received some other injury. But many patients are attacked by delirium tremens as the direct result of a bout of hard drinking. And the experience of those who have the management of prisons and other institutions where abstinence is enforced, has demonstrated that this does

not in itself bring on an attack, even in the most intemperate. The facts which have been supposed to establish the contrary are otherwise explicable. First, a dislike for stimulants is sometimes an early symptom of the disease, so that the reason why the patient leaves off drinking may be that he is already beginning to suffer from its effects. Secondly, a sudden shock to the system is exceedingly apt to act as a direct exciting cause of delirium tremens in those who are predisposed to it by intemperance. It is this which renders the disease so common in the accident wards of hospitals. Indeed, it often comes on within the first few hours after the patient receives an injury, when there could have been no time for the withdrawal of stimulants to take effect. It is also frequently observed as a complication of acute pneumonia, erysipelas, or some other febrile disease in patients of intemperate habits.

Diagnosis.—As stated above, delirium tremens has been formally recognised only during the present century. Sutton, however, mentions that William Saunders, a former lecturer on medicine at Guy's Hospital, had forty years before (*i. e.* in 1773) described it as distinct from phrenitis.* Sutton had become acquainted with it when practising in East Kent, where spirits brought in by smugglers could be had in great abundance at a cheap rate. Certain of the practitioners in that district, he says, had learnt to treat such cases with opium; and this practice was attended with very marked success in comparison with that of others (including at first Sutton himself), who regarded the disease as an inflammation of the brain, to be combated by venesection, blisters, purging, &c. Sir Thomas Watson, in his classical work on medicine, approaches the subject entirely from this point of view. He speaks of once being summoned to a man who was supposed to be mad, or to have brain fever, and of the necessity that one should be alive to the distinctive symptoms of delirium tremens. But at the present day the opposite mistake is the more likely to be committed, a case being set down as one of this disease when it is really one of acute mania or acute melancholia, diseases which (it must not be forgotten) may also be the result of alcoholic intemperance.

Dr Wilks speaks of having seen general paralysis of the insane mistaken for delirium tremens on two different occasions by the same physician. In the surgical wards of a hospital there is often great difficulty in distinguishing the latter disease from the effects of injury to the head. Again, fever or acute pneumonia may be overlooked, and a case wrongly set down as one of delirium tremens, particularly if the patient should have been intemperate. But in such instances it is perhaps really present as a complication. Moreover, we must remember that pneumonia may be altogether latent in drunkards. Dr Wilks mentions rheumatic fever, when attended with cerebral symptoms, as particularly like delirium tremens in its superficial characters.

Treatment.—The observers who first distinguished delirium tremens from acute phrenitis obtained what seemed to them wonderfully successful results by treating it with opium, and until recently most writers have advocated

* This term, or its equivalents *encephalitis* and *meningitis*, was formerly used as the pathological explanation of delirium or mania with febrile symptoms. But it is now known that the clinical type of disease in question is associated with enteric or other specific fevers, with the hyperpyrexia of rheumatism or erysipelas, or with the toxic effects of alcohol; and in none of these disorders is there inflammation of the brain or its membranes. The symptoms of true meningitis are, as we have seen, very different, and there is no evidence that primary acute inflammation of the encephalon ever occurs.

this practice. Sir Thomas Watson, for example, recommends that three grains of solid opium should be administered as soon as the bowels have been cleared out by a moderate purgative; and that if at the end of two or three hours the patient should show no inclination to sleep, one grain should be given every hour afterwards until the result is attained. Or, he adds, corresponding quantities of laudanum, of Battley's liquor opii sedativus, or of morphia may be prescribed.

But this advice was based upon the supposition that the disease, instead of having a tendency to subside spontaneously, would run on, and perhaps prove fatal unless the patient were made to sleep. Sir Thomas Watson expressly remarks that delirium tremens is not likely to be "healed with a *placebo*, or by waiting upon nature." And it is clear that the physicians who laid such great stress upon the value of opium measured their success by comparison with cases of supposed meningitis or phrenitis treated by venesection, leeches, and blisters. Wilks, Anstie, and most modern writers endorse Dr Ware's statements as to the natural course of the disease.

It is still, however, a question whether the course of the disease may not be abbreviated by hypnotic medicines. Anstie thought that this might be effected by chloral hydrate. He gave thirty grains for the first dose, and repeated it in an hour if the patient did not sleep. He found that the patient almost always got two or three hours of sound repose, and sometimes much more. He was of opinion that in delirium tremens there is a tolerance for chloral, so that as much as ninety grains or even two drachms may be safely given in divided doses within twenty-four hours, at least for a day or two. But Dr Wilks is disposed to share Dr Ware's opinion that the duration of the disease cannot be shortened by giving medicines to send the patient to sleep. He thinks that a certain time is required for the subsidence of the commotion, and that a severe attack must last at least three days.

If this view is correct, it would seem to follow that at the commencement of delirium tremens neither opium nor morphia should be given at all, or at least that their administration should not be repeated so as to cause contraction of the pupils. For, when hypnotic remedies are actively pushed, one is apt to find oneself after two or three days in a difficult position. The patient, instead of sleeping, may be as excited as ever. His pupils are perhaps reduced to the size of pins' points, and he has had as much opium as would kill two or three healthy persons. Under such circumstances—which are precisely those in which a consultation is most likely to be sought—there can be no question that the proper course is to wait. The further administration of hypnotics is dangerous. Dr Wilks says that he has many times seen persons suffering from delirium tremens sent to their last sleep by opium, and the same result has followed the subcutaneous injection of morphia. The fatal symptoms produced by these drugs in patients suffering from delirium tremens seem to differ from those to which they give rise in healthy persons. At least a patient of the author's in these circumstances became collapsed rather than comatose. Indeed, as already mentioned, the disease sometimes terminates by sudden collapse, even when no hypnotics have been given.

Another question, which has sometimes been raised when a patient has died after the administration of a large dose of opium, is whether one can infer that this could not have been the cause of death from the fact that a certain interval of time had elapsed. Thus, in a case that occurred to Sir Thomas Watson, that physician decided that the medicine could not have

been concerned in bringing about the fatal result, because nine hours passed after the last dose (one of three grains) was given before the patient became comatose. This conclusion would be perfectly valid if the case were one of suspected poisoning in a healthy man, but it is scarcely equally safe in delirium tremens. Dr Christison long ago pointed out that in persons actually intoxicated with alcohol the effects of opium are much retarded.

But whatever doubts may fairly be entertained as to the part played by opium in bringing about a fatal result in cases of this kind, there can be none as to the importance of treating delirium tremens so that no such question can be raised. We have seen that most patients do well even if no opium at all is given, and therefore the fact that many recover after having taken heroic doses is no justification for administering them.

This, however, does not settle the problem whether or not it is advisable to give safe and moderate doses of opium in the early period of delirium tremens, at a time when there is no reason to suppose that the case will be one of so grave a kind. To determine that point, one requires to know whether the medicine, if it should fail to give sleep, can do any harm; and, again, whether it is ever needed at a later period, in order that one may not then be debarred from using it by having employed it at first.

Before we can decide the question we must know what is the class of subjects in whom delirium tremens is most apt to prove fatal. Now, as regards *prognosis*, Anstie speaks of old age as especially unfavourable, and one has been always accustomed to think that for young subjects the disease ought to be unattended with danger in a first attack, and even in two or three subsequent ones. Dr Aitken, however, quotes some statistical facts published by Dr Macpherson, and based upon observations made in the General Hospital at Calcutta, and it is remarkable that there the highest average mortality was in persons between twenty-five and thirty-five years old, being from 23 to 24 per cent. of those attacked, whereas the mean mortality at all ages was about 15 per cent. Among fifty-nine cases of fatal delirium tremens which have occurred within the last few years at Guy's Hospital, there were three in persons between twenty and twenty-nine years of age, eight in those between thirty and thirty-nine, ten in those between forty and forty-nine, eight in those between fifty and sixty. What ratios, however, existed between these numbers and those of all patients attacked by the disease during the corresponding periods is uncertain.

The state of the *kidneys* affects the prognosis in delirium tremens very materially, uræmic coma being very apt to occur as a complication. The urine must be repeatedly examined for albumen and for casts; and the quantity passed each day must be noted. If the secretion should begin to fail, Anstie recommends hot fomentations and dry cupping to the loins, hot foot-baths, and the administration of half-ounce doses of infusion of digitalis every three hours. Digitalis was recommended as a remedy for delirium tremens itself by the late Mr Jones, of Jersey, who used to give enormous doses of the tincture, and clearly showed that digitalis is one of the poisonous agents for which there is a special tolerance in this disease.

Of scarcely less importance in reference to the prognosis of delirium tremens is the state of the *heart*. Anstie pointed out that besides feeling the pulse, one should carefully watch the first sound of the heart with the stethoscope. And he laid still more stress upon the use of the sphygmograph. An "irregularly undulating" character of the pulse-wave—such as occurs in the typhoid condition—was found by him to be the most

unfavourable augury. In his article in the 'System of Medicine' a tracing is given which was taken from a man, aged forty, who, after being delirious for nearly a week, fell into a sound sleep of six or seven hours' duration, and when he woke appeared to be so much better that a confident opinion was expressed that he would recover. Anstie's sphygmographic observation, however, led him to augur the worst possible issue, and about twenty-four hours later the case terminated fatally.

The symptoms which indicate approaching failure of the heart's action are chiefly faintness and lividity of the countenance. Under these conditions free stimulation appears to be the only resource. If the stomach will retain ether, this may be given in half-drachm doses. But if not, Anstie recommends port wine, especially if it is old and contains volatile ethereal compounds in abundance. He speaks of giving an ounce and a half of such wine every hour. He also says that hot mustard plasters to the chest may be useful.

Before giving either opiates or stimulants it is most important to see that the bowels are freely open.

Dr Bristowe believes that opium may still be used even in full doses in delirium tremens with good results if given early and watched.

General management.—The liability for the heart to fail in delirium tremens renders it necessary that the patient should be well supported by nourishment from the very commencement of the disease. He may have milk if he will take it; but if not, he must be compelled to swallow strong beef-tea, beef-tea jelly, or soup. He may even have small pieces of underdone chop or steak, if he can be induced to eat them.

The administration of alcohol in moderate doses was formerly recommended in cases of delirium tremens as a routine practice; it was thought to aid in inducing sleep. But all those who have studied the natural course of the disease are now agreed that there is no proof of its acting in this way; and there are great objections to its use, since the patient's future depends entirely upon the effect which his illness may have in leading him to give up stimulants for the rest of his life.

Another matter of the highest importance is that the room should be kept cool and dark and quiet. The patient's friends, who commonly collect around him, must be sent away, one or two intelligent and able-bodied men being alone left to watch him. The gaslights are to be turned down, and in the daytime a dark cloth is to be hung before the window, unless the patient should be terrified by the hallucinations which visit him in the dark. If he should be very troublesome in wanting to get out of bed, the question must be entertained whether he should not be tied down. If an attendant can by persuasion, or by employing a little force from time to time, induce him to lie quietly in his bed, that is no doubt to be preferred. But it is far better that he should be kept in a recumbent position by a sheet folded across his chest and tucked well in, than that he should be violently held down and restrained for any length of time by the hands of those about him.

Chronic alcoholic neuroses.—Until recently the action of alcohol upon the nervous centres was commonly supposed to consist mainly in the production of the acute disease just described. But it is now known that a long-continued state of nervous disorder is of far more frequent occurrence.

Symptoms.—The most important of these neurotic symptoms is muscular tremor. This is more commonly noticed in the hands, which are unsteady

and shake; but Anstie found that in a majority of cases the lower limbs were really affected before the upper. The patient is often able at first to control the movements by an effort of the will. They are generally more troublesome in the morning than at any other part of the day, and they may render him unable to do any work requiring nicety of manual adjustment, until he has taken a dram, or eaten some food, by which for a time they are removed. Even before marked tremor occurs, the action of alcohol is sometimes manifested by a peculiar restlessness; the limbs are apt to start involuntarily, and cannot be kept quiet except by an effort of attention. At the same time there is an irritable condition of the mind; the patient, though he may feel drowsy when he goes to bed, cannot sleep, but keeps turning from side to side.

At a somewhat later stage other cerebral symptoms develop themselves. Anstie speaks of a buzzing or rushing sound in the ears as very common; and with it there is often a dull diffused headache. Muscæ volitantes are often complained of; flashes of light seem to pass before the eyes, especially at night, just when the patient is dropping off to sleep; and there are momentary attacks of vertigo.

The intellectual and moral powers gradually become impaired. All certainty of purpose is lost, and there is mental disquiet which makes it impossible for the patient to settle down to any occupation or to complete the tasks he may begin. He often has a vague feeling of dread for which he cannot account; or he may become the subject of some delusion, such as that an enemy is lying in wait to injure him. Anstie mentions as another symptom that the patient often has a vivid apprehension that he is in danger of falling down a precipice, even when walking on firm ground in broad daylight, and he says that this is of unfavourable augury.

Another occasional effect of chronic alcoholism consists in pains in the limbs, especially around the wrists and ankles, as well as in the shoulders and down the spine. They are somewhat paroxysmal in their character, returning each day at about the same hour, most commonly towards night; and they are greatly aggravated by fatigue, whether of mind or body.

Impairment of sensation is also apt to occur, especially in the upper limbs; and the power of muscular co-ordination may be lost, so that the state of the patient closely resembles that produced by tabes dorsalis. Epileptiform convulsions sometimes show themselves, and they are of the gravest augury. The mental state often passes into mania or melancholia, and ultimately complete dementia may develop itself.

Indications of gastric disorder are present at an early period. A common complaint is that of *nausea* in the early morning, or of actual vomiting, and this may recur day after day as regularly as during early pregnancy. Morning *diarrhœa* is another frequent symptom. There is generally a failure of appetite, particularly at breakfast-time. The tongue is foul, with a thick yellow fur; or, less frequently, it is red and glazed. Moreover, it is tremulous when put out. The breath acquires a peculiar fœtor, which is not that of any alcoholic drink, and which can hardly be described, although when once smelt it is unmistakable. The eyes are red and watery; the conjunctivæ are often slightly jaundiced; the features look flabby and expressionless; the nose, cheeks, and forehead are often reddened, with crimson points and lines, called *stigmata*, which correspond with minute dilated veins.

Sometimes the same parts of the face present a papular or pustular

eruption, such as is commonly known by the name of "acne rosacea," or the nose may be enlarged, with pendulous outgrowths. However, it must not be understood that the appearances last mentioned are seen only in those who have indulged in alcoholic excesses; they are sometimes seen in persons who have been perfectly temperate. Acne rosacea—or gutta rosea, as it is more properly termed—is in fact a symptom of dyspepsia, most often but not always the dyspepsia of drink. Moreover, in women it may be the result of disorder of the menstrual functions.

The limbs of a person suffering from chronic alcoholism are commonly wasted, especially the legs. The abdomen, on the other hand, is generally enlarged or pendulous, and its parietes, as well as the contained viscera, may be loaded with adipose tissue. In those whose chief beverage has been beer, even the limbs may be covered with fat; and since the interstitial connective tissue of the muscles is swollen by fat, there is often apparent bulkiness of the fleshy parts of the arms and legs.

With regard to the chronic action of alcohol upon the nervous centres Anstie remarks that, contrary to what is the case with the digestive organs, the effects depend almost solely upon the quantity imbibed, and not at all upon the form in which it is taken. There are, however, immense differences in the liability of different individuals to suffer from this poison. Some can drink spirits freely for years, and seem to be none the worse for it; others break down in health under comparatively small amounts of drink. Women in particular show little power of resisting its evil effects; perhaps because those who are intemperate commonly pass all their time indoors.

Among the *antecedents* of intemperance must be mentioned, in the first place, those occupations in which persons are brought continually into contact with intoxicating liquors. A large proportion of the patients who present symptoms such as have been described are men employed in breweries or distilleries, public-house keepers and their wives, and travellers for wine and spirit merchants. Cab-drivers and hawkers, and others who are exposed to rough weather, are also very apt to be intemperate; while for some persons, as cooks and shoemakers, it is admitted as an excuse that their occupation is monotonous, deprives them of proper exercise, and keeps them confined in close, ill-ventilated rooms. Depressing mental influences drive others to drink; poverty and despair make them eager for the oblivion of intoxication. Again, there are cases in which stimulants were at first taken for the relief of pain, but in which the habit of indulgence grows upon the patient until it becomes a disease. Anstie has rightly laid great stress on the responsibility of allowing women to take wine or brandy in order to render them less susceptible to neuralgia, or to the sufferings which are so apt to attend on the menstrual period.

Lastly, there is no doubt that proclivity to intemperance is capable of hereditary transmission. This tendency is by some writers regarded as itself a neurosis, to which they give the name suggested by Roesch, "Oinomania," more correctly "Œnomania." The patient is seized every few months with a craving for drink, and for days together he behaves like a madman, taking long journeys without any purpose, or perhaps behaving indecently, eating little and drinking much. But when the affection passes off—at the end of a month or so—he regains his usual health. He then lives soberly and chastely, and manages his affairs with intelligence.

The *morbid changes* that are found in the nervous centres of drunkards are essentially atrophic. The cerebral convolutions are wasted, and the

cerebro-spinal fluid between them is in excess. The arachnoid is opaque and thickened. Even the bones of the cranium are sometimes found denser than normal and without diploë.

The effects of chronic alcoholic poisoning in producing certain forms of spinal paraplegia and the still more characteristic paraplegia due to peripheral neuritis have been already discussed (pp. 423 and 458).

The *diagnosis* of chronic alcoholism may present every degree of difficulty or it may be perfectly easy. Commencing general paralysis, locomotor ataxia, hysteria, and the nervous malaise which may result from dyspepsia, are perhaps the conditions most likely to be confounded with it. Unfortunately one cannot place any confidence in the statements of the patient himself, however guardedly our questions may be put, for persons who indulge in secret drinking are always untruthful. Anstie recommends that one should abruptly but not uncourteously hazard the observation that the diet must be strictly unstimulating; this, he says, will often cause the patient to betray the truth by his manner, or his air of surprised unconcern may prove that the suspicion is groundless.

In the *treatment* of chronic alcoholism the most important point is that the patient should abstain at once and entirely from all intoxicating drinks. It is in such cases above all others that large hydropathic hotels are of service, where regular habits and exercise in the open air are enforced, while an unstimulating diet is provided and all facilities for procuring drink are withdrawn.

The administration of certain medicines may also be of considerable service. Anstie recommends especially quinine, which (if there should be sickness) may be given in a state of effervescence. Dr Marcet's favourite remedy was the oxide of zinc; he says that in doses of from two grains upwards thrice daily it has a powerful effect in inducing sleep. Bromide of potassium often does great good in cases of this kind. To relieve the imperious craving for drink, aromatic spirits of ammonia, tincture of nux vomica and tincture of capsicum in a bitter infusion are extremely useful drugs. According to Anstie, half-drachm doses of ether with the same quantity of tincture of sumbul may do much to restore tranquillity to the nervous system and to make sleep possible. As a more direct hypnotic he recommends from a quarter to half a grain of the extract of Indian hemp. Chloral hydrate and hyoscyamus are also valuable hypnotics. If opiates are absolutely necessary, one should always employ the hypodermic injection of morphia (from one tenth to a quarter of a grain) rather than give any preparation of opium by the mouth, and this only when the urine is free from albumen and the patient has been purged. Even in advanced stages Anstie obtained striking benefit from the administration of cod-liver oil; and he says that when there is much tremor, strychnia is often useful in a dose of $\frac{1}{48}$ to $\frac{1}{32}$ of a grain three times daily.

As to *prognosis*, the symptoms of chronic alcoholism may last for years; and even the hardest drinkers sometimes reach old age. They are indeed apt to become prematurely old, their hair turning grey and their arteries becoming rigid. Gout, chronic disease of the kidneys or liver, or fatty degeneration of the heart may be developed, and cerebral apoplexy may cut them suddenly off. Such diseases as pneumonia or fever are ill borne, and are very likely to prove rapidly fatal, as are also accidental injuries and surgical operations.

NEUROSES FROM MORPHIA.—The evil which arises from the abuse of that invaluable drug, opium, has long been recognised. It is taken as a valuable medicine in malarial districts—in Cambridgeshire, for instance; and probably its abuse began in the same way in China. It produces loss of appetite, constipation, and emaciation, with the deterioration of the character which always follows indulgence in what is known to be an evil habit. Coleridge, after many years of uselessness and misery, emancipated himself from the thraldom. De Quincey has left in his 'Confessions of an Opium-eater' a monument of the pleasures, the pains, and (it must be added) of the incapacity to tell the truth which this habit produces.

Opium-smoking as practised among the Chinese is said to be even more injurious than opium-eating.

The useful and convenient practice of subcutaneous injection of morphia has of late years brought with it, in Western Europe and in America, a more wide-spread abuse than that of opium. The morphia-habit, as it has been termed, seems to be more prevalent in Germany than in England. It usually begins in the legitimate use of the anodyne to relieve pain, but the result is so rapid and effectual that the patient is apt to make the punctures himself, and, once familiar with the practice, he continues it long after the original disorder for which it was prescribed has disappeared.

The patient suffers terribly from insomnia and restlessness, he (or she) loses appetite and becomes pale and sallow, sometimes constipated and sometimes troubled with frequent and irritable action of the bowels. Moreover, sooner or later some of the punctures are sure to suppurate, and the pain of the abscess leads to fresh injections in parts before spared.

The writer has only seen a few cases of this malady. The first was an old gentleman from America, who had long become accustomed to the habit and suffered comparatively little. The next was an elderly lady, who had begun the practice for facial neuralgia and succeeded in breaking it off. The third was a young American, who suffered from obscure pains in the region of the gall-bladder. A fourth was a maiden lady, who began the habit to allay the pain of a renal calculus, and who was cured of both at last. In a fifth case, where the arms were scarred all over with the marks of abscesses, abdominal pain had been the origin. A sixth occurred in a young man, the subject of severe and complicated caries of the bones and joints, and he more than once nearly killed himself with a large dose.

The prognosis is not worse than in cases of alcoholic intemperance. The patient must be encouraged to bear the pain, and when unsupportable it must be relieved by smaller doses of the remedy. Often a single drop of the officinal solution is sufficient, and not infrequently pure water has, by a pious fraud, been substituted for the liquor morphiæ. Moreover, bromides, chloral hydrate, hyoscine, sulphonal, or chloralamide may each be substituted with advantage in its turn.

Neuroses from other drugs.—The *bromides* themselves produce, if long given in large doses, very unfavourable effects on the intelligence, of which an example which came under the writer's care is mentioned at p. 766.

Chloral hydrate produces a still more miserable condition of depression in some patients, and, valuable as it is, should never be used as an habitual medicine.

HYSTERIA AND HYPOCHONDRIASIS

"Orandum est ut sit mens sana in corpore sano."
JUVENAL.

"In nervous diseases he is the best physician who knows best how to inspire hope."
COLERIDGE.

HYSTERIA—*A real and distinct disease—Mental and moral perversions—Hysterical affections of sensation and of movement—Contractures—Globus hystericus—Flatulence—The hysterical fit—Ætiology and Pathology of hysteria—Diagnosis—Prophylaxis—Treatment.*

Catalepsy—Trance—Sleep-walking—Ecstasy—Dancing-convulsions—Mania—Hystero-epilepsy—Anorexia and marasmus—Hysteria in men and boys.

HYPOCHONDRIASIS—*Distinctions from hysteria—The supposition of imaginary diseases, commonly abdominal—Distinction from melancholia—May mask real disease—Treatment—Prognosis.*

HYSTERICAL disorders of function, or pains, or perverted sensations, are seated in the most diverse organs of the body, not only in the head, chest, and abdomen, but also in the joints, the mammæ, the eyes, and the limbs.

But is hysteria a distinct and definite malady? For there are some who refuse to admit its claim.

The name* suggests the erroneous opinion that the organs of generation in the female play the principal part in causing hysterical affections. Those who see little or no connection between them have been tempted not only to do away with the name, but to dispute the clinical association of the symptoms as manifestations of a single malady.

Again, it must be admitted that, more than most other diseases, hysteria is difficult to define. As a neurosis, independent of any appreciable structural change, its definition would naturally be based upon either its causes or its symptoms. But the former are still the subject of discussion, and the latter are singularly variable and inconstant. Moreover, the curability of most hysterical affections, and the fact that they are generally contrasted with organic diseases, have caused a tendency to apply the term vaguely to cases of unascertained nature, but which are regarded as of a trifling character, and likely to be of transient duration. In this way the words hysteria and hysterical have often become meaningless.

Very little study at the bedside, however, is needed to convince one that most of the affections called hysterical are really the expression of a special morbid condition, for which the name of Hysteria is as convenient as any other, besides having the sanction of antiquity and established usage. One finds that two or more of these affections are commonly present in the same patient at the same time, or that a person who has suffered from one is

* Ὑστερία (*i.e.* womb-sickness), from ὑστέρα, the uterus—Passio hysterica.—*Anglicè*, Fits of the mother—The vapours.

exceedingly apt to be afterwards attacked by others. Then, again, there are marked peculiarities in the age, sex, disposition, and other conditions of such patients. Above all, there is in most cases a particular mental state which can easily be recognised apart from other indications of the disease, and which may be regarded as its fundamental character.

Mental condition.—The principal features of the mental state which characterises hysteria are an exaltation of the emotions, a perversion of the will, and a loss of the balancing power of the judgment. The patient seems to have no power of controlling her own feelings. The most trifling occurrences excite her to rapture or plunge her in despair. She may conceive a violent affection for some persons, and an equally irrational aversion from others. She perhaps exhibits great attachment to some pet animal, while other creatures, no less harmless, excite in her repugnance and disgust. She torments all those about her with her caprice and her selfish claims upon their attention; she appears unable to make up her mind to the smallest effort, and lies in bed all day from sheer want of energy to rise. But in some particular direction she will show the most obstinate determination and tenacity of purpose. She has a great craving for sympathy, and is exceedingly anxious for her delicacy of health to be recognised. In pursuance of this object she will apply corrosive acids to her skin, swallow needles day after day, or run them into all parts of her body, and make unsuccessful attempts to poison or drown or hang herself. Her stoical endurance of pain is sufficient to show that a mere defect of volitional power cannot be the essential element of hysteria. Perhaps the best way of expressing the facts is to say with Dr Reynolds ('Syst. of Med.,' vol. ii) that the will is no longer called into exercise by the judgment or reasoning faculties, but only by some one dominant idea or emotion. Thus, as Jolly observes (von Ziemssen's 'Handbuch,' vol. xii), if one can by stimulating her enthusiasm or spirit of emulation supply a hysterical patient with a sufficient motive to undertake any good work, such as nursing, she will often carry it out with more than ordinary perseverance and energy.

Moral perversion.—The moral tone of a hysterical woman is often seriously impaired, particularly in certain directions. Morbid sexual inclinations have often a strong hold upon such a patient, and she will scheme to induce her medical attendant to make a vaginal examination. Sir Thomas Watson relates a case in which a young woman contrived to make a hospital surgeon believe that she had a stone in the bladder; and the imposture was not detected until she had been tied up in the position for lithotomy in an operating theatre full of students. A patient who was supposed to have a hydatid in the liver, and who had been punctured without result, allowed the operation to be repeated, and almost immediately produced a piece of the stomach of a rabbit (or some other small animal), which she declared she had vomited, and which she no doubt thought would be mistaken for a hydatid membrane; a few hours later she sent for her medical man again, to remove from her vagina another piece of the same substance. Others have been known to drink their urine and then to bring it up again, pretending that none was passed in the natural way. The fact that such cases have occurred makes one hesitate to accept as authentic such cases as have been described on the authority of Charcot as hysterical ischuria. There is often great difficulty in detecting impostures of this kind. Charcot himself relates a case of Boyer's in which a woman pretended that urine came from her umbilicus, ears, eyes, and breasts, and that she vomited

fæces; and it was not until she had been placed in a strait waistcoat that little balls of fæces ready prepared for swallowing were found in her bed. Hysterical young women also simulate phthisis and pretend to spit or vomit blood.*

Some of the *factitious affections of the skin* are by no means easy of diagnosis. In the museum of Guy's Hospital there is a model of the right breast of a girl, which is reddened and has on it a number of large raised tubercles, some of them bigger than peas. The first time that patient was an inmate of the hospital the cause of the affection remained undiscovered, although it was evident that some irritant was being intentionally applied. But more than a year afterwards she was again admitted, and Mr Birkett, happening to visit the ward at an unusual hour, found a piece of lint, strewn with powdered cantharides, which she had put on the breast. Sir William Gull related a similar case in which he detected with a lens a glistening fragment of the same powder on the skin itself. Some years ago a girl was admitted into Miriam Ward who had on her chest and breasts a number of whitish gangrenous-looking patches, of irregular sizes and forms; these successively shrivelled up into brownish scabs, which soon became detached, leaving the skin beneath reddened and rather scaly. On one finger, in the groove between the skin and the nail, there was found a bright yellow stain, which affected both structures to about the same extent, and which seemed indisputably to have been caused by a drop of nitric acid. So that perhaps the patches on her chest were likewise produced by the acid, although they did not show the characteristic yellow colour. It happened strangely enough that shortly before she came in there was in the ward another patient affected in exactly the same way, but (there was no reason to doubt) with spontaneous gangrene. She also was a girl, aged eighteen, but apparently free from suspicion. The museum contains models taken from each of these patients.

Other forms in which hysterical patients exhibit their craving for notoriety and their willingness to deceive are illustrated by the cases of the Welsh fasting girl, and Louise Lateau, the Belgian nun.

Physical symptoms.—Among the manifestations of hysteria "the hysterical fit" has always been regarded as especially typical of the disease; and two others, the "globus" and flatulent distension of the abdomen, are more common and scarcely less distinctive. Before describing them, however, it will be convenient to mention some symptoms which are less often seen.

Perversions of sensation.—Not the least curious of these are perversions of the special senses. The patient perhaps complains of intolerance of light, and insists on having the room kept darkened. Here, however, imagination often plays a great part. Dr Reynolds relates the case of a woman who had for weeks been lying with her hands before her eyes to keep out the light of a dull London sky. When he brought a candle close to her in order to examine the pupils she shuddered, knit her brows, and held both hands between it and her eyes. But as soon as her attention was distracted to the state of her front teeth, the brows became relaxed, the hands were removed, and she bore the light without inconvenience. In other cases the patient is distressed by the slightest sound, and will allow no one to speak above a whisper. Yet such a person may herself exclaim in a loud voice or

* A remarkable case of this kind obtained some notoriety about thirty-five years ago by the publication of a book called 'The Female Jesuit in the Family.' The patient, a typically hysterical governess, imposed on the Protestant credulity of the household.

make a great noise in stirring the fire without seeming to mind it. In other cases there appears to be an actual exaltation of perception. A hysterical girl may seem, as Dr Reynolds says, "to hear through stone walls;" but in such matters deception is often practised, for such a patient often pretends that she can see with her eyes shut.

Jolly quotes from Amman the case of a woman who discovered the presence of some cherries in another room by their odour, and who could distinguish one person from another with her nose; and such persons often detect by their taste the presence of the minutest trace of any flavour that they dislike. It is perhaps a perversion of the gustatory sense that leads girls affected with hysteria to eat cinders, sealing-wax, lead pencil, and the like; this perverted appetite used to be called "*pica*." They are sometimes equally fond of repulsive odours or flavours.

The sense of touch, again, may be unduly acute. In almost every case of hysteria there is at some part or other of the body over-sensitiveness to painful impressions—*hyperæsthesia*, or rather, in strict language, *hyperalgesia*. Sometimes the patient complains that it hurts her very much to have the skin over one or more of the spinous processes pressed upon or even touched; sometimes she has extreme tenderness of the breast, or of the edge of the costal cartilages.

Another frequent effect of hysteria is impairment of common sensation, *dysæsthesia* or *anæsthesia*. This may either be limited to ordinary tactile impressions or it may include those of heat and cold; in other instances it concerns only the perception of pain, and is called *analgesia*. Gendrin went so far as to declare that sensation was more or less defective in every case; and other writers have asserted that loss of feeling in some part of the skin is invariably left behind by hysterical fits. Their statements, indeed, are too absolute, but the symptom is undoubtedly often present, and Lasègue and Charcot are probably right in saying that it would be more frequently observed if it were carefully looked for, since the patient may be unaware that she is unable to feel pain in some particular part, until her sensibility is tested. In many cases, however, subjective sensations of tingling, pins and needles, &c., are experienced and complained of by the patient. It is important to note that there is no opposition between hyperæsthesia and anæsthesia, such as might appear from the names. A part of the skin may be exquisitely tender, and the seat of burning pain when touched; and yet at this very part the power of appreciating tactile impressions may be greatly impaired.

Anæsthesia, or defective sensation, is much more frequently observed in some regions than in others; Jolly mentions the backs of the hands and feet, and the parts above the outer malleoli, as its favourite seat. Sometimes it affects exactly one half of the body, leaving the other free. To such cases Charcot has given the name *Hemianæsthesia*. He quotes Briquet as having stated that this occurs on the left side more often than on the right side in the proportion of seventy cases to twenty. The limitation of the parts in which there is loss of feeling is often remarkably sharp, corresponding almost precisely with the median line of the face, neck, and body. It differs from hemiplegia in this character and by the greater implication of sensation than of motion. The mucous membranes are affected in a similar way. Taste may be wanting in one half of the tongue, the sense of smell may be impaired, and there may be a considerable degree of amblyopia, with limitations of the visual field for the several colours—a

symptom which Galezowski calls *Achromatopsia.* According to Jolly, reflex excitability is also wanting; sneezing cannot be induced by irritating the nose, nor retching by tickling the fauces on the affected side. Lastly, Charcot says that the side on which sensation is impaired is cold and pale, and that there may be difficulty in drawing blood from the skin by a needle's prick.

Altogether apart from hemianæsthesia, loss of feeling in the mucous membranes is common. Anstie laid considerable stress on the frequency with which it may be found, if looked for, in the back of the pharynx. According to that writer ('Lancet,' ii, 1872), whenever a person who has not been taking a bromide can without retching let one pass the finger down to the epiglottis, the diagnosis of hysteria is probably correct. Dr Reynolds mentions that he has known several cases, occurring in married women who were still bearing children, in which there was absolute anæsthesia of the vulva and vagina. Impairment of sensation in the bladder is supposed to be sometimes the cause of retention of urine in hysterical patients, but in other cases it appears to be due to a prurient desire to have a catheter introduced. Jolly relates the case of a hysterical patient who burnt herself terribly by taking live coals out of the fire and pressing them with both hands into her vagina, without showing any sign of pain.

Hysterical anæsthesia is probably never permanent. It may last for several weeks and even for months, but sooner or later it always passes off.

Motor paralysis.—Another manifestation of hysteria is paralysis of movement. Aphonia and dysphagia often result from hysterical loss of power in the laryngeal and the pharyngeal muscles respectively; and the former is highly characteristic, so that its presence gives great aid towards the diagnosis of doubtful cases. Not uncommonly hysterical paralysis takes the shape of paraplegia or of hemiplegia. The former affection was described at p. 462. The latter likewise presents characters of its own. It occurs much more frequently on the left side than on the right. The proportion in Briquet's cases was as forty-six to fourteen. An important distinction between it and the paralysis due to a lesion in the opposite side of the brain is that in hysterical hemiplegia the side of the face and tongue remain unaffected. This was long ago remarked by Dr Todd, who also pointed out that the patient in attempting to walk "drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no art of circumduction nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks." Dr Reynolds adds that a paralytic patient looks at her feet; a hysterical patient at the persons who are watching her.

In some cases one limb is alone affected with hysterical paralysis; or, much more rarely, only part of a limb. The only muscle affected singly is the levator palpebræ; and hysterical ptosis is not uncommon.

Hysterical paralysis may be transitory, lasting only a few days, or not longer than a few hours. Sometimes it afterwards reappears either in the same limbs or in others. But there are cases in which it persists for months or even years; and in some cases rigidity sets in, to be mentioned in the next paragraph. The paralysis may disappear suddenly, under the influence of a sudden emotion or shock; or it may pass off very gradually. The removal of the patient to a hospital ward—away from sympathising friends and relations—is often followed by a more or less rapid recovery. In several cases under Dr Wilks's care this result has been brought about rather by moral influence than by any special treatment. The patient is told

that she is expected to begin to move the paralysed limbs; day by day she is encouraged to do more; after a time she is taken out of bed, dressed, and put into a chair; and before long she walks as well as ever.

Contractions.—Another and a very curious effect of hysteria is a chronic spasm or rigidity of one or more of the limbs, associated with a complete loss of voluntary movement. This has recently been studied with much care by Charcot, who has published in his 'Leçons' several capital drawings illustrative of the distortions which it produces. Some English writers have since adopted into our language the French word *contracture* for such cases. The affection often follows immediately upon a severe hysterical fit; and the paralysis and the rigidity then generally develop simultaneously. But in some cases the latter does not come on until there has been loss of power for a considerable time. In certain cases only one leg or one arm becomes contracted; but sometimes both legs are affected, sometimes both the arm and the leg on one side, sometimes three of the limbs, or even all four. When the affection assumes a hemiplegic type, and the rigidity follows the loss of power after some interval, it might be thought that there would be a difficulty in distinguishing it from the "late rigidity" of hemiplegia due to organic disease of the brain (p. 567); but in the hysterical affection the contraction comes on, not gradually, but suddenly, and as the immediate result of a fresh hysterical fit. Moreover, the positions assumed by the individual limbs are peculiar. The arm is semiflexed; but, according to Charcot, the leg always falls into a state of rigid extension, the knee and ankle being straightened and the toes stretched down to the utmost extent possible, while the sole is turned inwards. Thus the condition of the foot resembles that which exists in talipes equino-varus. Charcot also mentions that the adductors of the thighs are forcibly contracted; and Jolly relates a case in which one leg became powerfully flexed, especially at the knee-joint. In a marked case of hysterical paraplegia (which came under the writer's observation many years ago) the girl's legs were forcibly flexed at the knees and hips, so as to resist the utmost power that could be safely used to straighten them, until chloroform was administered. She afterwards recovered completely, and became an excellent nurse.

The rigidity in these cases is not relaxed during sleep; nor does it undergo any variations in degree at different times or periods of the day. The spasm is by no means confined to a single set of muscles, for one is not able to force the patient's arm (which is semiflexed) into a position of complete flexion, any more than to extend it. By making the patient inhale chloroform, however, if its action is carried far enough, one can temporarily remove the spasm, the affected parts becoming perfectly supple in all but very exceptional cases.

Anæsthesia of the affected limbs is generally present. The muscles remain well nourished, and retain their electro-contractility at least for a considerable time. But when a limb has been contracted for some years without intermission, a little general wasting may occur, and the reaction of the muscles to galvanism may be somewhat impaired. The termination of hysterical "contracture" is almost always in recovery; and in many cases this takes place abruptly. One of Charcot's cases is very instructive. A woman was first attacked, at the age of thirty-four, with loss of consciousness after a moral shock; she then fell into the fire and burnt her face severely. After this she had several seizures, some of them hysterical in

character, others rather epileptiform. Four years afterwards she had a very severe fit, attended with convulsions and followed by an apoplectiform stupor with stertorous breathing; this was at once followed by left hemiplegia. Rigidity of the left limbs set in abruptly after an interval. In the following year the right limbs also became contracted; and later still the jaws were fixed, so that an œsophageal tube had to be used. Her right arm, however, became free; and Charcot maintained that recovery was still possible. One evening, six years from the commencement of the contraction of the left arm and leg, she had an attack in which she imagined herself to be about to die. She cried out, became agitated, and with her right arm pushed aside those who held her. Her strong desire to get to the window for air was resisted; she became more and more angry; and first her right leg lost its rigidity, then her left leg, and finally her left arm. She got up and walked; and in eight hours her cure was complete. A slight crackling in the joints was all that remained of the complaint.

Such a case has an obvious bearing upon those in which cures are attributed to miraculous agencies. They are seldom related in such a way as to enable one to speak positively as to the exact form of paralysis which had been present. But Charcot quotes an article by Littré in which detailed histories are given of certain persons who were cured at the end of the thirteenth century by pilgrimages to the tomb of St Denis, where the relics of Louis IX had recently been deposited. Three of these were young women who had been suddenly attacked with contraction of one leg or of the arm and leg on one side, and who also had anæsthesia. They were cured suddenly, under circumstances certainly very likely to act upon the imagination. In fact, the analogy seems to be complete.

In some exceptional cases of hysterical "contracture," however, recovery never takes place. Charcot mentions several instances which he believed to be hopeless. One is that of a woman, aged fifty-five, whose legs had been contracted for sixteen years. Under chloroform her knees could still be relaxed; but her feet remained in a condition of equino-varus. This observer has made an autopsy in one case in which all four limbs had been contracted for ten years; and he discovered sclerosis of the lateral columns in nearly the whole length of the cord. The patient was a hysterical woman, and the rigidity passed off several times and returned again before it became permanent. There seems no reason to doubt that the case originally belonged to the same class as the rest. Charcot accordingly supposes that sclerosis may come on secondarily in such cases. The points on which he would lay stress as indicating the probability of an unfavourable termination to a case of long-standing contraction of a limb are that particular groups of muscles are wasted in an extreme degree and affected with fibrillary tremor like what is seen in progressive muscular atrophy; that there is a great diminution in the contractility, as tested by faradisation; and that the rigidity should remain to a very considerable extent when the patient is under the influence of chloroform. On the other hand, he does not attach any importance to a little general loss of substance in the affected limbs, to a slight impairment of the electrical reaction of the muscles, or to the presence of spasmodic movements, which last are not uncommon in such cases, as in those of ordinary hemiplegia.

Globus.—Among the most common and characteristic symptoms of hysteria is the so-called *globus hystericus*. It is difficult to say whether it is merely a morbid sensation, or depends upon spasmodic contraction in the

œsophagus and pharynx. The former opinion has been maintained by Eulenburg and Reynolds, the latter by Jolly. To the patient herself the globus is a sensation, most commonly a feeling as if there were a ball or lump in the throat, which she cannot swallow or get rid of in any other way. She may even put her finger into the pharynx, and make herself sick, although she knows there is not really anything that can be dislodged; or she may drink mouthful after mouthful of water to wash it down. The exact nature of the sensation varies a good deal in different persons. In some it is likened to that which would be caused by a bean sticking in the throat; others feel as if a worm or some other living animal were creeping about in its interior. With others, again, it is rather a feeling of constriction about the neck as if the collar was too tightly fastened. The relation of such a feeling to the state of emotional excitability which characterises hysteria is shown by the fact that a precisely similar sensation is felt by healthy persons when suddenly seized with grief or terror. Thus, children before a fit of crying experience a sensation of a lump in the throat which is probably identical with the more marked form of globus. In some hysterical women the lump is not merely felt in the throat, but seems to rise up from the chest, or from the abdomen.*

Flatulence.—Another frequent symptom of hysteria is a rapid distension of the intestines with gas. The abdomen may suddenly swell up, so that the patient is obliged to take off her stays and undo all the fastenings round her waist. Jolly remarks that this form of tympanites bears no relation to meals. In several instances he has seen it return each morning regularly and attain its maximum about midday. The gas may ultimately escape either by the mouth or from the rectum. Jolly says that it is commonly quite free from odour; and this, he adds, explains the fact that the distension sometimes subsides without any apparent escape of wind in either direction. How such an enormous quantity of gas can be produced in the stomach and bowels is quite unknown. It is sometimes undoubtedly air which has been swallowed; sometimes probably gases, liberated from the food and expanded in accordance with physical laws by relaxation of the muscular walls of the stomach and bowels. The chemical character of the gas needs more exact determination, but it is believed to consist chiefly of carburetted hydrogen.

In some cases hysterical tympanites persists for years together, the abdomen being as tense as a drum. Bamberger alludes to a case of this kind, in which the patient passed through an attack of cholera without alteration in the size of the abdomen, but in which an abundant flow of saliva, coming on spontaneously, once led to its temporary subsidence.

The hysterical fit.—There remains to be described that which was once regarded as the chief symptom of hysteria, the hysterical attack or "fit of the mother." In the majority of cases no such attacks occur during any part of their course; according to Briquet, in three cases out of four. Moreover, when they do occur they present great variety of symptoms. Sometimes they are of so trifling a character that it is unnecessary to send for medical advice. The patient perhaps has an unusually painful globus, and then begins to cry and sob, or falls into violent laughter. Or she may laugh and cry alternately, until she is exhausted. Such an attack, for which the vernacular name is now "hysterics" and was formerly "vapours," may occur singly, or there may be several in the course of two or three days.

* So Lear:—

"Down, climbing sorrow: thine element is below."

A very constant symptom is that when they pass off the patient voids a large quantity of colourless urine, of low specific gravity.

In many cases a hysterical attack is accompanied by violent movements of the body and limbs which may be distinctly convulsive in character. Perhaps the patient screams out, and then fall into a state of opisthotonos, remaining supported for some minutes together only by the back of her head and by her heels. Or she may throw her limbs in all directions, beating them on the ground, or striking her own chest with her closed fists, tearing her hair, kicking those about her, and struggling with all her might to release herself from restraint. Or, again, she may thrust one arm high in the air; or forcibly bend her arm over the chest, and her thighs across one another; some of the fingers and toes being at the same time stretched apart to the furthest possible extent, while others are as strongly flexed. The respiration is much quickened, and it may be very noisy, but it is not actually stertorous. Her features undergo the strangest contortions and grimaces. Her eyes are generally closed; the pupils are of natural size. The eyelids quiver, and the eyes are often turned upwards, particularly if an attempt is made to separate the lids by force. The state of the eyes thus differs altogether from that which belongs to an epileptic fit, for in it they are commonly fixed wide open, and have their pupils greatly dilated.

There appear to be all degrees of impairment of consciousness in hysterical attacks. At the commencement the patient almost always knows what is happening, so that she is able to place herself out of the way of injury. Sometimes she keeps on screaming during the greater part of the fit, or shouts out "fire," "thieves" or "murder," or pours forth a continuous current of the foulest language. Sometimes she has visual hallucinations, addressing furious objurgations to imaginary persons, or fancying that she is surrounded by wild beasts or spectres. Sometimes she seems to be altogether unconscious; but even then by careful watching one may often make out that she really notices what is going on, casting occasional furtive glances at the bystanders from between the half-closed eyelids or modifying her behaviour under the influence of their remarks.

Ætiology.—In passing on to consider the causes of hysteria, we find ourselves face to face with a question which even now can hardly be said to be settled, although it has been discussed for centuries—the question as to the part played by affections of the generative organs in the ætiology of the disease. The very name *hysteria* involves etymologically the doctrine that it is the result of uterine disorder; and the ancient Greek writers—including Plato and Aristotle—actually supposed that the womb left its place in the pelvis and wandered about the body, setting up disturbances in different parts. From their time to the present there have never been wanting theories which, according to the different standpoints afforded by the science of the age, have referred hysteria to a similar cause. The physicians who have held such views in extreme forms have, indeed, been generally professed gynæcologists rather than men acquainted with the pathology of nervous diseases; but Romberg, the great German authority on these disorders forty years ago, defined hysteria as "the reflex neurosis proceeding from sexual irritation."

By different writers various disorders of the female sexual apparatus have been made the starting-point of hysteria. Some have endeavoured to trace it to unsatisfied desire, citing in support of their views the frequency of its occurrence in young widows and in the wives of impotent husbands;

others have referred it to masturbation or to excessive venereal indulgence, and others again to the common menstrual disorders. Addison published a clinical lecture in 1830 to show that its phenomena depended upon "uterine irritation." By Dr Henry Bennet ulceration and induration of the cervix uteri were represented to be morbid conditions of great potency in causing such effects. More recently Dr Graily Hewitt has assigned the principal place to flexions of the womb.

Charcot has laid great stress on the frequency with which hyperæsthesia of the ovary is present. When lecturing on this subject at the Salpêtrière he showed to his class five women (almost all the hysterical patients who were then in the hospital), each of whom had pain in, or tenderness on pressure over, one or both of the ovaries. According to him the pain is sometimes very acute, and widely diffused over the abdominal surface, with special hyperæsthesia of the skin over an area two or three inches in diameter, corresponding with adjoining parts of one iliac and of the hypogastric regions. In other cases no spontaneous pain manifests itself; indeed, the skin is anæsthetic, and the muscles can be pinched up without pain; but on making deep pressure towards the brim of the pelvis one can feel the ovary as an egg-shaped body, which slips beneath the fingers. Further pressure upon it gives rise, not exactly to pain, but to a peculiar sensation which the patient at once recognises, having felt it many times before as the starting-point of hysterical attacks; and this, passing up to the epigastrium, is followed by pain in that neighbourhood, and sometimes by nausea and vomiting. Next, if the pressure on the ovary be continued, the patient experiences palpitation of the heart with extreme rapidity of the pulse, a sensation of globus in the neck, a hissing noise in the ear of the same side, a feeling as if blows were being struck with a hammer on the temporal region, an impairment of vision, especially in the corresponding eye, and then perturbation of consciousness, ending in a regular hysterical fit. Charcot also says that when there is hemianæsthesia, or paralysis, or contraction of the limbs on one side of the body, it is always the ovary on the same side which is hyperæsthetic; and that if such symptoms are bilateral, both ovaries are affected. Lastly, he has found that when a patient is seized with a hysterical attack, even if it is epileptiform, it may be cut short by energetic compression of the ovary continued for three or four minutes.

Charcot, although he speaks of ovarian hyperæsthesia as "playing a predominant part" in the clinical history, nowhere speaks of it as the *cause* of the disease. There are, in fact, ample proofs that Romberg's definition, already quoted, is inaccurate. Thus hysteria is occasionally met with in males, according to Briquet in as many as 5 per cent. of all cases; and Dr Reynolds says that in them its development seldom bears any relation to the generative functions; he has sometimes observed it in boys at puberty, but the most marked cases which he has seen have been in men from thirty-five to fifty or upwards, who have been mentally overworked or greatly reduced in physical power. So, also, in women, although probably more than one half of those who become affected with hysteria show the earliest indications of it between the twelfth and the twentieth years, about the time when menstruation was first being established, there are yet a considerable number of girls who manifest hysterical symptoms at a much earlier period of life. No fewer than one in five of Briquet's cases occurred in girls under the age of puberty.

It is one of the most important facts in the ætiology of hysteria that,

even among children, it is far more common in girls than in boys. We have seen (p. 738) that this is the case with chorea likewise, and in both diseases the preponderance of cases in females is probably due to the same cause, namely, their innate emotional susceptibility and want of power to resist external influences. It has been correctly remarked that hysteria seldom, if ever, shows itself in such women as would be called "masculine," whereas the men and boys who are attacked by it are those who would be said to be of a "feminine" disposition. One influence which does much to favour the development of the disease is the want of a proper education. If the girl is fanciful and capricious, her parents yield to every whim. If she is morbidly self-conscious, she is allowed to indulge her vanity, and to nurse the belief that she is gifted and misunderstood. If she is studiously disposed, she is left to pore over books, and never gets proper exercise in the open air. If she craves for precocious excitement, she is taken to balls and theatres when still young, she is permitted to sit up late at night, to spend the morning hours in a soft bed, to live on a rich and stimulating diet, and to feed her imagination with romances. The influence of example may also induce a liability to hysteria; the daughter of a hysterical mother is very likely to acquire the disease in her turn; so potent does this cause appear to be, that according to the best authors there is no need to suppose that hysteria is ever transmitted by descent. In comparison with the emotional excitability in a girl, her intellectual capacity has but little influence either in rendering her susceptible of hysteria or in guarding her against it. As regards bodily constitution, some hysterical women are robust, with fresh-coloured cheeks and a vigorous circulation, but by far the larger number are weakly and delicate.

The real relations between hysteria and affections of the generative organs are best elucidated by the aid of the analogy afforded by some of the paroxysmal neuroses. We have seen that in many persons the attacks of megrim are constantly excited by disorders of the visual apparatus, but that in others this is not the case. We have found that vertigo is in some individuals brought on by diseases of the ear, and in some by diseases of the digestive organs, but that in others it is traceable to neither. So also it appears that the neurosis, hysteria, cannot essentially depend upon any one set of external exciting causes. In some cases it perhaps develops itself spontaneously. Very often anæmia or chlorosis appears to play the most important part in its causation, and it has sometimes been observed as the direct result of deprivation of food. In other instances—probably in the immense majority—its manifestations are directly called forth by impressions which reach the nervous centres from without. But these are by no means always derived from the generative organs. Emotional and psychical influences—a sudden alarm, the shock caused by the death of a relative, or the unexpected loss of property—may bring on hysteria in a person who had before seemed perfectly healthy. A similar result may follow a gunshot wound (as has been observed by Weir Mitchell), or it may be brought about by a railway collision. In some cases a spirit of imitation suffices for its production; the disease has been known to spread like an epidemic from one woman to another among the inmates of a hospital or a workshop. In other instances, as was pointed out by the late Dr Anstie, indulgence in alcohol seems to be the occasion of hysteria.

Again, it often happens that several of the conditions which may bring forth manifestations of hysteria are present together. A person who is

attacked after a sudden fright may before have had her health depressed by misery and exhaustion of mind and body, or may have been labouring under religious excitement, jealousy, wounded self-love, or remorse. The liability to hysterical symptoms of all kinds is always increased at the periods of menstruation, and there is reason to believe that it is also augmented in many women at the climacteric age, although this does not appear from statistical data.

But no doubt there are many cases in which disorder of the generative organs is really the main cause of the development of this neurosis, and it is practically wise to accept Addison's teaching that such disorder should be carefully searched for whenever it can possibly be supposed to exist. Fortunately we are able to investigate the state of the ovaries by pressure above the brim of the pelvis without offence to a patient's delicacy; but writers are by no means agreed as to the actual facts in regard to the frequency of ovarian irritation or other morbid conditions of the sexual apparatus in hysterical women. Jolly disputes the accuracy of Charcot's statements as to the proportion of cases in which hyperæsthesia of the ovary is present. He quotes Scanzoni and Amann as having found among their hysterical patients as many as 19 or 20 per cent. in whom the generative organs were perfectly healthy; and, as he remarks, the fact that these observers were professed gynæcologists renders it certain that their figures in regard to this point are too low. Speaking from his own impressions he is disposed to state the proportion of hysterical patients free from these complaints at more than 50 per cent. (Ziemssen's 'Handbuch,' xii, 2, p. 461).

Diagnosis.—That this is often difficult must be sufficiently apparent, not only from what has been said in the present chapter, but from the repeated references to hysteria which have occurred in our discussion of organic diseases of the nervous system.* In all doubtful cases it is important to search for the indications of hysteria which have been described above (p. 803). For example, anæsthesia limited to small portions of the cutaneous surface is probably present in many cases without the patient's knowledge, and its discovery may go far towards clearing up a doubtful case.

Hysteria mimics the symptoms of almost every disease of the nervous system, and also of phthisis and of abdominal complaints.

One must bear in mind that a woman may be hysterical and yet be affected with organic disease, either of the brain or of some other part of the body. This remark is true even of cases in which all the symptoms of hysteria are present in the most marked form, still more if we include its more aberrant or doubtful manifestations. Sydenham went so far as to say that the majority of women were hysterical; and since the psychical characteristics of this neurosis are after all only an exaggeration of those which belong to the whole female sex, it is obvious that no absolute boundary line can be drawn.

Again, it is important to note that there is no kind of proportion in the degree to which the different symptoms of hysteria are developed in the same patient. A woman who has hemianæsthesia, or paralysis, or some local pain may present scarcely any indications of emotional susceptibility, may be quite free from uncomfortable sensations in her throat, and may never have had the slightest approach to a hysterical attack. Yet the whole course of the case may show conclusively that the complaint is of this nature. Indeed, when one has to deal with the more marked affections, such as

* See Dr Buzzard's valuable remarks on the "Simulation of Hysteria by Organic Disease of the Nervous System," 'Brain,' 1890, p. 1.

paralysis or contraction of a limb, which is clearly not due to organic disease, one may be justified in making a diagnosis of hysteria, even in the absence of all corroborative evidence.

So, again, one may safely attribute to the same cause all cases of hemi-anæsthesia if unaccompanied by motor paralysis. And even when the loss of sensation is limited to a single limb, one would probably be seldom wrong in coming to the same conclusion. Sir William Gull, however, has recorded in full detail a case in which he at first made a mistake ('Guy's Hospital Reports,' Third Ser., vol. iv). A nurse in the hospital, aged thirty-eight, complained of numbness in the left arm. Below the elbow sensation was completely wanting; but when the skin of different parts above that joint was carefully tested with the point of a needle there were all kinds of contradictions in her statements. She would one minute say that she could feel the needle at a certain spot, and the next minute that she could not feel it. Such observations led to the conclusion that the case was one either of feigning or of hysteria; but after the anæsthesia had existed for two years the limb became gradually paralysed. Two years later she died from the effects of an accident, and it was then found that the spinal membranes were thickened, especially in the cervical region, and that the posterior columns of the cord and the grey matter had undergone degeneration.

In practice we must distinguish not only between hysteria and organic disease, but also between it and malingering; for the two are far from the same. An hysterical girl is proverbially deceitful, but when cured of her hysteria she is cured of her deceit. A malingerer deceives others, but not himself, and his self-control is unimpaired.

Prognosis.—The rule that hysterical fits are devoid of danger to the patient's life is not altogether without exceptions. Wunderlich met with a case in which a servant girl, aged nineteen, who had for eight weeks suffered from a succession of epileptiform fits of hysteria, unattended with fever, fell suddenly into a state of *collapse*, and died in two days with a temperature of 109·4°. Other cases have been recorded by Meyer in which excitement of a nympho-maniacal character was followed by cramps and spasms in various muscles, and led to collapse and death after an illness of some weeks.

Again, the more chronic forms of hysteria sometimes prove fatal by *marasmus* (cf. p. 821, *infra*). Dr Wilks has related in the 'Guy's Hospital Reports' for 1866 the case of a young lady who lost her sister rather suddenly by heart disease, whereupon she began to experience palpitation and sickness and pain over the heart, would take no food, declared that she had her sister's complaint and should soon follow her to the grave, and did actually die in a sort of hysterical convulsion exactly five weeks afterwards. He also gives the case of another girl who would eat nothing, but merely nibbled a biscuit and drank wine by drops. The bowels ceased to act, but masses of scybala were from time to time removed from the rectum. She became thin, and Dr Wilks at length renounced the opinion that the complaint was merely hysterical, and thought that there must be some partial obstruction in the small intestine. But when, at the end of more than two years, she died rather suddenly, he could find nothing to account for her illness. Her body was then fatter than when he first saw her, and she had in fact taken rather more food towards the last.

Treatment.—We must in the first place consider what degree of success may fairly be aimed at in dealing with hysteria. We can hardly expect to modify by drugs the peculiar morbid temper which underlies it, although

moral influences may strengthen the power of self-control. A complete cure of the hysterical tendency is seldom to be looked for, at least until the patient has passed the climacteric period. The earlier the stage at which the disease began to manifest itself the more unfavourable is said to be the prognosis. It is very important that a child who displays emotional excitability which seems likely to develop into hysteria should be placed under the most favourable hygienic influences possible. Exercise in the open air, a plain nourishing diet, limitation of the hours of study, early rising, sea bathing, and the use of the shower bath, at once suggest themselves as prophylactic measures.

When a hysterical patient is anæmic or chlorotic she should, of course, take one of the preparations of iron for a considerable time. If there be any uterine disorder it is generally advisable that the patient should employ some astringent injection for the vagina, such as the compound alum lotion which Addison recommended so highly, and which contains a drachm of sulphate of zinc and a drachm of alum in half a pint of water.

In dealing with special symptoms, again, one must never overlook the importance of moral treatment. This is often successfully employed in the management of hysterical paraplegia—an affection which is apt to persist for months, and even years, if left to itself. So, also, one can sometimes cure hysterical aphonia by urging the patient to speak and confidently assuring her that she is able. For this affection electricity, too, is very useful. Dr Reynolds recommends that sparks should be taken from the larynx; or that a shock should be administered from a Leyden jar; or that an interrupted current should be passed through the throat. In long-standing cases Sir Morell Mackenzie's instrument may be employed, by which galvanism is applied directly to the vocal cords. Each of these methods often leads to the instantaneous restoration of the voice. Dr Reynolds speaks of a strip of empl. lyttæ applied round the throat as having occasionally been successful when electricity had entirely failed.

Paralysis of the limbs may be treated with advantage by faradisation, and passive movements and frictions should also be diligently employed. But Dr Reynolds says that he has found no remedy so effectual as the application of a narrow piece of blistering plaster completely round the affected parts.

Hysterical anæsthesia is said to be sometimes benefited by the application of a rather powerful faradic current by means of a dry metallic brush. Stimulating liniments may also be rubbed into the affected parts.

The remarkable effect on hysterical anæsthesia of the application of gold coins, metallic bracelets, and magnets, and even of pieces of wood and other indifferent objects, attracted much attention a few years ago at the Salpêtrière Hospital (see the second Report by MM. Charcot, Luys, and Dumontpallier to the Société de Biologie, 'Brit. Med. Journ.,' 1878, vol. ii, p. 548). It is difficult, if not impossible, to unravel the underlying facts from the tangled mass of hysterical perversions, physical and moral; but any good effect which "metallothérapie" may have by its action on the imagination of the patient is more than exceeded by the attendant evils (cf. Dr A. H. Bennett's case, 'Brit. Med. Journ.,' 1878, ii, 563; and Dr Donkin's paper, *ibid.*, p. 613).

At present metalloscopy and metallotherapy have begun to recede whither the once famous "metallic tractors of Perkins" and the odic force of Reichenbach preceded them; and we hear more of "hypnotism,"

a revived mesmerism, and of "suggestion" in this induced hypnotic state.*

For rigid contraction of the limbs, the application of the continuous galvanic current is sometimes useful. But probably benefit more often results from straightening the affected joints under chloroform and placing the limb upon a splint, than from any other plan of treatment. Sir Thomas Watson speaks of cases in which a stream of cold water directed upon the contracted part and continued in spite of the patient's complaints of pain, led at once to the relaxation of the spasm. He also mentions an instance in which Sir Charles Clark cured by the same method a girl who had hysterical trismus and could neither speak nor eat. He began to pour pitchers of water upon her face, but before he had emptied the second pitcher she began to scream and complain, with her mouth open widely enough.

When hysteria manifests itself by producing a chronic and long-standing contraction of a limb, the administration of medicines seems to be altogether useless. But for most of the other effects of the disease drugs are certainly useful. Most writers speak of assafœtida and valerian as owing their virtues chiefly to their disgusting taste. But many hysterical women actually relish assafœtida. Moreover, pills containing valerianate of zinc ought on such a theory to be almost inert, whereas there is no doubt that, in doses of gr. j—ij, they are most effectual in removing aphonia, hemianæsthesia, and hysterical hemiplegia. In 1874 a woman was in the clinical ward of Guy's Hospital who had paralysis of her left arm and leg, the latter being rigidly extended; she took large doses of assafœtida for some days without benefit, but was afterwards quickly cured by the valerianate of zinc, so that she walked out of the hospital within five days from the time when she first began to take it. In other cases we have obtained equally good results with assafœtida, five grains of which were taken every three or four hours. Dr Reynolds has found bromide of potassium altogether ineffectual in any form of hysteria. Both he and Jolly recommend that opium or morphia should be given for the relief of pain and to procure sleep; and they rightly observe that for the former purpose the subcutaneous injection of the alkaloid is especially effective. But one must always keep before one's mind the evil results which may follow from the establishment of a morphia habit (see p. 429). The continued use of choral as a hypnotic is to be objected to on precisely similar grounds. And perhaps more harm is done by alcohol than by either of them. The late Dr Anstie was well justified in the strong protest he uttered against the practice of advising a hysterical woman to take a glass of wine or a teaspoonful of brandy, or even a dose of sal volatile to keep up her spirits whenever they are depressed, or to enable her to bear a frequently recurring pain. One should never sanction the employment of alcohol in any form by such patients, except perhaps in very moderate quantities and at meal-times only.

The system of treatment introduced by Dr Weir Mitchell is in bad cases sometimes useful. It consists in separating the patient from sympathising friends, making her eat, and using *massage* to the limbs.

When one is called to a patient who is actually in a hysterical attack, there are several methods by which one may succeed in cutting it short.

* See the late Mr James Baird's excellent little book, 'Magic, Witchcraft, Animal Magnetism, Hypnotism, and Electro-Biology' (1852); and Professor Heidenhain's 'Animal Magnetism,' translated by Dr Wooldridge, with a preface by Mr Romanes (1880). Mesmer was a Viennese physician who threw impressible patients into a *sommeil critique* by magnetised water. The same thing is now done in Paris by means of a revolving kaleidoscope.

One plan, suggested by Dr Hare, is to keep the patient from breathing for a certain time by forcibly closing her nose and mouth. The vigorous inspiration which she makes, as soon as she is allowed to do so, is said to be often followed by a relaxation of all spasm and a subsidence of the fit. Dr Reynolds says that he has found this mode of treatment notably useful when an attack is sufficiently prolonged to make it worth while to interfere with the natural course of events. Another method, to the value of which many can testify, consists in drawing the patient's head and shoulders over the edge of the bed and pouring cold water upon them from a large jug. Sometimes one has merely to make preparations for carrying out this procedure, by giving the necessary orders to the patient's friends in her hearing, in order to bring the fit to an end. An ancient hospital sister in Esther Ward never failed to cut short an outburst of hysteria among her patients by the same homely method.

Compression of one ovary will sometimes arrest a hysterical fit, even although it may be of an epileptiform kind; Charcot speaks of one patient who learnt to apply the pressure herself. Another procedure, often adopted by the late Mr Stocker, consists in pressing upon the arteries and other structures on each side of the neck, in the same way as has already been described under the treatment of epilepsy (p. 765).

One must always keep in remembrance the fact that, excepting in seizures of a strictly epileptiform character, the patient is perfectly alive to all that is going on, although she may seem to be unconscious. A calm and quiet demeanour, the avoidance of all expressions of sympathy or of uneasiness about the issue of the attack, may do much to shorten its duration; for if one is flurried or alarmed she is sure to notice it.

Catalepsy.—In some instances a hysterical attack is attended with very remarkable symptoms, which have long been regarded as belonging to a special neurosis named catalepsy (*κατάληψις* = a seizure or arrest). Its characteristic features are that the patient, although she may be deprived of sensibility and of voluntary motion, remains fixed in whatever position she occupied at the commencement of the fit, and yet that her muscles offer no such resistance to external force as would prevent the limbs from being easily bent or extended by another person, or the body from being placed in any posture. The condition of a cataleptic patient may be compared with that of a lay figure, such as artists use; if she is sitting up her arms can be put at the most awkward angles with the trunk, and will remain without falling, at least for a time; if she is recumbent her spine may be bent upon the pelvis so as to form an obtuse angle with the thighs, and will retain that posture. The name of *flexibilitas cerea* is sometimes given to this peculiar state of the muscles; they have been found by Rosenthal to have their electric sensibility and contractility either normal or decidedly increased. It is, however, a mistake to suppose that during the cataleptic state the muscles are capable of resisting the force of gravity for an indefinite length of time. On the contrary, the limbs, if extended, slowly fall again to the side. Dr T. K. Chambers quotes a case in which an impostor was detected by the simple plan of attaching a weight to the extended hands. She supported it without moving, and this was taken as showing that she was not labouring under a genuine attack of catalepsy; ultimately she confessed the fraud. The eyelids of a cataleptic patient may either be widely open or shut; in the latter case, if opened, they perhaps very slowly close again. The pupils

may contract under the influence of light. After recovery, all memory of what occurred is said to be lost.

These details of the "cataleptic" paroxysm appear to belong to this place, because it probably never occurs in a well-marked form except in persons who either are obviously hysterical or who at least may be strongly suspected. A case of the minor form of epilepsy was quoted (p. 750) in which it assumed a cataleptic character; but fully developed catalepsy does not appear ever to accompany epilepsy in a person of the male sex, or in a woman with no hysterical tendencies. The attacks seem almost always to be traceable, at least in the first instance, to a fright or to some other powerful emotion. Dr Chambers mentions the case of a girl, a patient in St Mary's Hospital at Paddington, who when Covent Garden Theatre was burnt down was awakened by the light flashing into the ward through the uncurtained windows; on the following morning she was attacked with catalepsy. The most striking instances of catalepsy which stand recorded are to be found in medical works of a time when the modern extended conception of hysteria was not dreamt of, and yet the reports of these cases often contain clear proofs that the patients were in the highest degree hysterical. The celebrated history related by Dr John Jebb nearly a century ago is a case in point. The subject of it was a young lady who suffered from hysterical risings in the throat and flatulence, and who was highly susceptible to every change in the weather. Although she was prepared for Dr Jebb's visit when he first went to see her, she was seized with the disorder as soon as his arrival was announced. "She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form the figure of death-like sleep. . . . The positions of her fingers, hands, and arms were altered with difficulty, but they preserved every form of flexure they acquired, nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it, as firmly as her limbs. . . . About half an hour after, the rigidity and statue-like appearance being yet unaltered, she sang three plaintive songs in a tone of voice elegantly expressive, and with affecting modulation." The seizures sometimes lasted as long as five hours.

Trance.—In other cases similar attacks have been of still longer duration. The condition of the muscles has then been different from the *flexibilitas cerea* of catalepsy proper; either the limbs oppose considerable resistance to all attempts to change their posture, or they are completely flaccid, falling into any position whatever by their mere weight. Such patients may most fairly be said to be in a state of trance. Writers describe this condition as sometimes lasting for days and weeks at a time. The face may then be perfectly pale, the breathing so feeble as to be imperceptible except on the closest examination, and even the heart's pulsations and pulse at the wrist discoverable with great difficulty. These are the cases which have led to the popular belief that death is sometimes only apparent, and that there may be a danger of persons being buried alive; and it cannot be denied that a patient in such a condition might easily be allowed to die by careless or ignorant attendants, or might be buried before death.

Sleep-walking.—In other hysterical persons the attacks assume the form of somnambulism or of ecstasy. The former is a condition which may be compared to an acted dream. It is probably sometimes a transformation of epilepsy or some other paroxysmal neurosis; and when not allied to those

diseases it is almost always a manifestation of hysteria. The somnambulist, without seeming to wake from a state of sleep, walks all over the house and even out of doors. She balances herself without difficulty on narrow planks, and manages to avoid all obstacles in her path. She takes no notice of anyone whom she may meet, there is much difficulty in rousing her, she remains for a time bewildered, and she has no recollection of what she has been doing. Marvellous stories are told of the feats which are accomplished by persons in this strange condition. But one must not forget how untruthful hysterical patients often are, how prone to take a pleasure in mystifying and deceiving those about them, and how apt to be encouraged in such a course by the wonder which their performances call forth.

Ecstasy.—The state of ecstasy is one in which a person becomes regardless of all external circumstances and engrossed with some particular emotion or idea. She remains motionless, with staring eyes and fixed expression, or she may repeat a few words with ceaseless monotony. After she returns to consciousness she remembers the vision she beheld in the state of day-dreaming.

A similar state can be produced by mesmerism (or "hypnotism" or "electro-biology"), and is called by the practitioners at Nancy in 1890, *somnambulisme provoqué*. When this condition frequently recurs we have as a result what is called double consciousness, of which marvellous examples are from time to time published. A careful investigation of such cases might, amid much credulity and imposture, possibly discover some facts of physiological interest.

Chorea major.—Mental excitement naturally finds vent in muscular action, and rhythmical movements in turn increase and exalt emotion.

Religious dancing was common in antiquity, and survives as a minuet before the altar, which may still be seen in Seville Cathedral, and as the solemn gyrations of dancing dervishes at Constantinople.

In the Middle Ages there were epidemics of Dancing Mania, of which an interesting account will be found in 'Hecker's Epidemics of the Middle Ages' (p. 87 of Dr B. G. Babington's translation for the Sydenham Society). This Chorea major was also known as the Dance of St John the Baptist, as the Dance of St Vitus* (cf. p. 728), and in Italy as the Tarantula. It spread over Germany and the Netherlands in 1374, reappeared at Strasburg in 1418,† and was not extinct till the seventeenth century. Pearce, a traveller in Abyssinia, early in the present century, gives an account of a very similar dancing mania in that country, which was called "Tigretier," and Dr Davidson has described an epidemic among the natives in Madagascar which closely answers to the accounts of Chorea major in the Middle Ages. Similar cases in Scotland are described by Sir Walter Scott in his 'Demonology and Witchcraft,' and find a parallel in the religious dances of the "Jumpers" in the United States.

A remarkable instance of this kind was recorded by Mr Kinder Wood ('Med.-Chir. Trans.,' vol. vii). The patient, a young married woman, who had suffered severely with pain in one side of the face,

* Possibly the connection with this saint was through the dancing of the daughter of Herodias, but more probably it was because the old pagan festival of dancing and leaping through the fire took place on St John's Day at Midsummer. St Vitus was the patron and help in need of those afflicted with dancing mania, as St Martin in smallpox, and St Anthony in erysipelas.

† "Vier Hundert fingen zu Strassburg an Zu tanzen und springen, Frau und Mann,
Bis ihnen das Wüthen wieder gelag; Sanct Vit's Tanz ward genannt die Plag."

began to be troubled with involuntary movements in the eyelids, which were opened and shut with excessive rapidity. After a time the hands were beaten rapidly upon the thighs, and the feet upon the ground. Then she became half raised from her chair and seated down again, these movements succeeding one another as quickly as they possibly could. Other modifications of her attacks occurred from time to time, until at last she took to skipping about the room, regulating her movements by a series of strokes on the furniture as she passed, or by movements of her lips, as though beating a tune. Someone thinking he recognised the air as "The Protestant Boys" began to sing it, and she suddenly turned and danced up to him, and continued dancing until she was out of breath. Then a drum and fife were procured, and she immediately danced up as close to the former as possible, and went on till she lost the step, or until the measure was changed, or was made so rapid that she could not keep up with it. It was presently found that a continued roll of the drum put an end to her movements; and thus the attacks were at length prevented. The explanation which the patient gave was that there was always a tune dwelling on her mind which at times irresistibly compelled her to begin the involuntary motions.

Still more extraordinary movements were performed by a lady who came under Dr Abercrombie's observation, and who had suffered for two years from various nervous affections. While lying perfectly quiet, she would suddenly with her whole body make a kind of convulsive spring, by which she was jerked entirely out of bed. Or, if sitting or lying on the floor, she would fling herself into bed, or leap, as a fish might, on to a wardrobe fully five feet high. After a time the muscles of the back and neck became affected with a wonderful semi-rotatory movement, which sometimes went on without interruption night and day for weeks together. If the head or neck was touched, the motion was increased to extraordinary rapidity. She was cupped, and the affection suddenly ceased with a convulsive start of the whole body; but it returned again and again, and finally disappeared only when constipation and menstrual disorder were corrected.

Some years ago a similar case occurred in Guy's Hospital. A little girl, aged nine, had been knocked down by a boy five days before her admission. She was insensible at the time, and seven hours afterwards she had a fit. Subsequently she had nine other fits. They began by her making a low sighing or moaning sound, after which the upper extremity became contracted, the teeth were clenched, and with a sudden bound she threw herself completely out of bed. When she recovered consciousness she had no remembrance of what had occurred. On the day of her admission she had fourteen fits, and at one time she remained insensible for two hours. A day or two later she had two fits during the visit of the physician, Dr Owen Rees; in these she clapped her hands, and her face went through a most extraordinary series of contortions. In one attack she struck a part of her head to which a blister had been applied, whereupon she at once became conscious. After she had been in the wards six days one of the fits was commencing, when the nurse told her that she must be tied down: she immediately began to cry and recovered, saying that she would have no more fits. From that time she remained well.

For cases, in which vertical movements of the arm are incessantly repeated, the special name of *malleation* has been invented (cf. *supra*, p. 701).

Dr Wilks once had a girl under his care, who sat by her bedside and kept thumping with her fists all day long.

Gyration.—Hysterical movements of the head or the body round and round have been dignified with the special name of "*rotation.*" Several instances are related by Watson, of which the most striking, related by Dr Watt ('Med.-Chir. Trans.,' vol. v), occurred in Scotland in 1813. A girl, aged ten, who had before exhibited other nervous symptoms, was seized with a propensity to turn round on her feet like a top, with great velocity, and always in one direction. This continued a month, and then passed off. Afterwards she began to roll over and over on her bed, moving rapidly from one end to the other, for six or seven hours every day. Having been taken into a garden she quickly rolled along the whole length of a gravel walk, and even when she was laid in the shallow part of a river she began to turn round as usual, until she seemed on the point of being drowned. She made little or no use of her arms in performing the rotations. After another month she began an entirely new set of movements. Lying on her back she would, by drawing her head and her heels nearly together, bend herself like a bow, then she would relax her muscles, and fall with considerable force on her seat. This she repeated ten or twelve times a minute for several hours daily. After a fresh interval she began to stand on her head; and then to let herself fall again, her knees striking the bed first. This movement also she continued for fifteen hours a day, at the rate of twelve or fifteen times a minute. Blistering, purging, and leeching were employed without result; but having been taken to Glasgow from her home in the country in an open chaise, and brought back again after three days, she was seized with diarrhœa, and soon afterwards recovered entirely.

Maniacal excitement with hallucinations and delusions may complicate the hysterical attack. Such symptoms may be transitory, the patient becoming perfectly rational as soon as the fit passes off; but in many instances they last for a considerable time, and then it may be very difficult to distinguish the case from one of insanity. Many doubtful cases find their way into lunatic asylums, and the truth seems to be that no actual boundary line between hysteria and insanity exists. Dr Savage, in the twenty-first volume of the 'Guy's Hospital Reports' (1876), says that a large number of the female insane patients admitted into Bethlem Hospital have suffered from severe hysteria at former periods in their lives, and that many cases, at first regarded as examples of hysterical insanity, end in death or dementia. Some observers have made it a sort of test for hysteria that the mental faculties should be found unenfeebled when recovery takes place; and thus many doubtful cases have been at last set down as hysterical because of the suddenness and completeness with which the patients have regained their senses. Dr Savage speaks of one woman who imagined that she had the itch, and who refused to shake hands with anyone; she woke one morning quite free from delusion.

Hitherto we have not mentioned the occurrence, in a hysterical attack, of rhythmical *clonic spasms* at all resembling those which are seen in an ordinary epileptic fit; but spasms of this kind are not infrequently present, the other phenomena of the seizure being such as to leave no doubt as to its nature. In 1876 a girl was in Guy's Hospital whose main symptom was a rhythmical clonic spasm recurring at intervals of a few seconds in the neck, shoulders, and arms, and who had transitory fits in which she seemed to lose consciousness, while these jerking motions became much intensified

and succeeded one another with greatly increased frequency. One feature in that case was the perfectly bilateral character of the spasms, and this accords with the statements of writers to the effect that in hysteria the convulsive movements are rarely limited to one side, or much more severe on one side than on the other, whereas in epilepsy both conditions are frequent.

Epileptiform hysteria.—In some cases of hysteria, however, fits occur which in all respects resemble those of epilepsy. The loss of consciousness is complete; the eyes are widely open and the pupils dilated; a bloody foam escapes from between the lips, and the tongue is bitten; the movements are clonic rhythmical spasms of the simplest kind, and repeated again and again with but little variation. Perhaps this should not surprise us, for there are few diseases of the higher nervous centres (whether functional or organic) which may not be accompanied by similar attacks, of the most typically epileptiform character. But writers on hysteria, having probably in view the necessity of laying down a line of distinction between such fits and the ordinary hysterical seizures, have created for the cases in question the special designation of "hystero-epilepsy;" and there have been many discussions as to whether they are transitional links between the two neuroses, or instances of their co-existence in the same patient. But, as we have seen (p. 750), the disease epilepsy is defined not merely by peculiarities in the fits themselves, but by the fact that they recur again and again without any cause, unless it be an inherited tendency to nervous disorder. If it is theoretically inaccurate to regard as epileptic the fits to which some patients are liable for months or years as the result of the irritation of a carious tooth, it must be equally incorrect to give the name to the fits which occur only while a woman is under the influence of hysteria. And Charcot has recently pointed out that the so-called hystero-epilepsy differs altogether from true epilepsy in some important particulars. However closely the attacks may follow one another, they do not, as in the status epilepticus, entail danger to life with the development of a high bodily temperature. He relates the case of a woman who for more than two months laboured under a constant repetition of epileptiform fits. Once they succeeded one another without a break from 9 a.m. to 8 p.m.; and then again from 9 p.m. onwards for an equal space of time. He estimates that she had from 150 to 200 attacks on that one day alone. As he remarks, a patient suffering under epilepsy would soon have succumbed. The woman never passed urine nor fæces involuntarily, and got out to relieve herself in the brief intervals that occurred between the attacks. Her temperature rose occasionally for a short time to 101·3°; but the mean was 98·6°. As further distinctions between hystero-epilepsy and epilepsy Charcot alleges that the former never assumes the type of the *petit mal;* and lastly, that, however perfectly epileptiform a fit may be, it can always be arrested by compression of the ovary.*

Professor Mendel showed the Medical Society of Berlin in 1887 an apparently typical case of hystero-epilepsy or epileptiform hysteria, in the person of a man fifty years old.

* The extraordinary gestures of these unhappy patients have been carefully studied and named, as phases of "Ecstasie," of "Clownisme," and so on. See a report of a visit to Salpêtrière by Dr Gamgee ('Brit. Med. Journ.,' Oct. 12th, 1878); and details of cases in the same No., p. 561, with Professor Charcot's lecture, *ibid.*, p. 789. Some of the attitudes are shown to coincide remarkably with those of dæmoniacs in works of art.

Anorexia nervosa vel hysterica is a remarkable complaint which was described by Sir William Gull ('Clin. Soc. Trans.,' vol. vii). It is attended with extreme wasting, and is commonly supposed to be the result of some latent tubercular affection. But, remarks the writer, the emaciation is actually too great for this diagnosis to be correct; for persons with organic disease seldom become so thin as this until they are no longer able to get about, whereas it is characteristic of the hysterical anorexia that those affected with it display an excessive restlessness and bodily activity. In this complaint the pulse and the respiration are slow, and the temperature is generally below the normal. Sir William Gull's patients were chiefly young women between the ages of sixteen and twenty-three. It does not appear that any of them showed definite indications of hysteria. Some of his cases terminated fatally,* but in others a complete recovery took place, the main points in the successful management of them being the avoidance of fatigue, the administration of nourishing food at very frequent intervals, and the use of warm clothing.

It is in these cases that Dr Weir Mitchell's treatment by seclusion, with abundant feeding and regular shampooing or massage, is most successful.

Hysteria in male subjects.—As above stated, this remarkable neurosis or combination of neuroses is, in the vast majority of cases, a disorder of the emotions, the will, and the bodily functions of *women*—particularly incident to the periods of puberty, of early married life and of the menopause, but possible at any time after the infant has developed into the girl. Nevertheless undoubted cases of hysteria occur in the male sex, just as undoubted cases of hypochondriasis occur in women. It is extremely rare in full-grown men, but is not very uncommon in boys from the age of eight or ten to puberty and, less frequently, from that period till two or three and twenty.

In Dr Wilks's lectures on 'Nervous Diseases' there are several cases recorded. In one the principal symptom was so-called laryngismus, in another aphonia, in another convulsions which were supposed to be due to spinal meningitis, in another the "malleation" or hysterical hammering referred to above (p. 819). Sometimes the boy can be thrown into spasms like those of tetanus or of a frog poisoned with strychnia, as in a patient of Mr Holden's who was cured by removing a fatty tumour.

Dr Rühle, of Bonn, relates the case of a spoilt boy of fourteen, who had terrible convulsive attacks ending with vomiting, and was cured by "removal from home, cold shower-baths, and the fear of the rod" ('German Clinical Lectures,' p. 449).

The editor was once consulted about a schoolboy, who had twice been laid up at home on account of hæmorrhage from the bowels. He was ruddy and well nourished, and no disease of any organ could be discovered. A specimen of "melæna" was at last procured, and proved on examination to contain no blood, but a silver salt from a solution that he had used in photography. He then became violent and threatened to kill himself, but was soon cured by the wholesome discipline of a public school.

A naval surgeon gave a graphic relation in the 'Lancet' a few years ago, of a cabin boy of sixteen, who completely lost his voice and appeared to

* The writer once saw a fatal case of this form of hysteria. Extreme emaciation preceded death. At the autopsy there was confirmation of suspected vaginal irritation, but the organs were like those of starvation. The cord was normal to the eye, and histological examination revealed no lesion.

suffer from attacks of choking and suffocation. He was carefully treated for several days, until a sudden and unexpected pinch made him swear loudly, and thus revealed the imposture.

In 1888 we had in Philip Ward an apparently healthy young countryman, twenty years old, who, beside dyspepsia and hypochondriasis, suffered from "fits" of a decided neurotic character with well-marked globus hystericus.

HYPOCHONDRIASIS.*—From hysteria we pass to hypochondriasis by what seems a natural transition, for these two neuroses are commonly regarded as closely allied, and do in fact resemble each other in one prominent symptom—namely, their tendency to simulate organic diseases of various parts of the body. Indeed, some writers have gone so far as to declare that hypochondriasis, which chiefly occurs in men, is in the male sex the representative of hysteria in the female; and the uncertainty which still exists as to the relative frequency of hysteria in men and boys arises mainly from the fact that cases which would be at once set down as hysterical, if they occurred in women, are often wrongly attributed to hypochondriasis because the patients are of the other sex.

But hypochondriasis may be seen in women, especially about the time of the menopause, without any admixture of the proper characters of hysteria. Moreover, there is an essential difference between the two diseases. We have seen that the mental state in hysteria is characterised by an exaltation of the emotional faculties, and an increased susceptibility to outward impressions, the will being unable to control the feelings. But in hypochondriasis the morbid sensations which the patient experiences are not dependent upon any external cause; they are created by the mind itself. Thus Romberg was not without justification when he called it the antithesis of hysteria.

Again, hypochondriasis is unattended with any of those nervous vagaries —convulsive attacks, paralytic affections, hyperæsthesiæ, anæsthesiæ—which are so characteristic of hysteria. Its predominant symptom is always of one kind; the patient believes, without cause, that he is the subject of serious bodily disease. It is true that the full development of the complaint is often preceded by a period in which there is merely a general perversion of his feelings. He becomes gloomy, reserved, and wrapped up in himself; or his mental state alternates between a moody silence and high spirits with great loquacity. Presently, however, he becomes conscious of a pain in some particular region of his body, upon which he at once concentrates all his attention; and he is now a "hypochondriac."

The disease is one which has retained the same appellation since the days of Hippocrates and Galen. But they conceived the actual seat of the disease to lie in the viscera which are situated immediately below the diaphragm; and a similar hypothesis was intended to be conveyed by the equivalent English expression, "the spleen."† Indeed, it is the fact that the digestive organs are generally more or less disordered. Dyspepsia is often present; the bowels are constipated; large quantities of gas are apt to accumulate in the alimentary canal, giving rise to flatulent belchings and noisy rumblings in the abdomen. In all probability the starting-point of the morbid hypochondriacal feeling is really an impression

* Ὑποχονδριακὸν πάθος—Passio hypochondriaca—Passio atrabiliosa—Melancholia (in part)—Humor atrabiliosus—Milzsucht—The spleen.

† The first to vindicate the position of hypochondriasis among the neuroses was the celebrated anatomist Thomas Willis.

proceeding from the viscera, for among the symptoms of disease of the stomach and of the liver we shall find depression of spirits and irritability of temper. But what is peculiar to the complaint with which we are now concerned is the marked exaggeration of every local pain. Moreover, the region to which the pain is referred seldom, if ever, remains the same during the whole progress of the case. After having long dwelt upon a gnawing or burning pain at the epigastrium, which he felt sure must indicate cancer of the stomach or liver, the patient all at once loses this, and perhaps begins to suffer from symptoms which, if due to organic disease, would point to the throat as its seat. Or he is attacked with dyspnœa and palpitation of the heart, and feels convinced that he is labouring under serious cardiac disorder; or a slight cold leads to a cough, and he begins to collect the sputa, is certain that he is phthisical, and consults the physicians who have the greatest reputation for pulmonary affections; or he finds himself giddy, experiences a sensation of weight and pressure in the head, and forthwith thinks of nothing but of the apoplectic fit which he believes to be impending. But perhaps the most miserable of all hypochondriacs are those who refer their sufferings to the genital organs. To this class belong by far the larger number of those who consult one for spermatorrhœa, or for impotence. They complain that they have dragging pains in their testes, that these organs always hang too low, that their urine is turbid and must contain semen, that they have emissions at night, during defæcation, and at other times. One sees at once that their whole attention and thoughts are concentrated upon the sexual function; and to make matters worse, they are sure to have studied with avidity a number of books and pamphlets, written for the very purpose of stimulating their apprehensions, and of inducing them to seek relief from the quacks who write them.

If further evidence were required to prove that the disease is a neurosis, it would be afforded by the close relation which can be traced between it and other nervous affections. This was especially insisted on by Anstie, who maintained that the hypochondriac almost invariably belongs to a family of which other members have been of unsound mind. Unlike hysteria, hypochondriasis is markedly *hereditary*. As to whether the disease tends to pass into insanity in the same individual there is much difference of opinion. Melancholia is the form which bears the closest superficial resemblance to it; but Romberg points out that this is characterised by a tendency to self-negation, whereas in hypochondriasis the whole attention is concentrated upon personal feelings and sensations; and he goes on to show how the difference is expressed in all the patient's relations with other persons. A person affected with melancholia treats his medical attendant as an enemy or as an ignorant pretender, and constantly tries to avoid him; but the hypochondriac looks up to him as his guardian and saviour. He is constantly on the search for new plans of treatment, and expects that each fresh one will relieve him of his complaints. As Romberg puts it, "the more physicians, the better he is satisfied; he likes to change them as often as he would change a poultice." Yet, in spite of all, he is not unhopeful, and is never weary of life. A tendency to commit suicide is no part of hypochondriasis; and any doubtful case in which such an attempt is made may be safely set down as one of actual insanity. Probably the same may be said of every case in which the patient has distinct delusions, as (for example) that he is made of glass, or that the devil is playing a barrel organ in his belly, or that the bowels are about to escape through the abdominal

parietes, or that he has swallowed spiders, and that they are multiplying within his body. Griesinger, though so great an authority on mental disorders, seems to have entirely mistaken the boundary line between hypochondriasis and melancholia; the cases which he relates as examples of the former are almost all of them really instances of the latter disease (cf. p. 832). This perhaps explains the circumstance that he found hypochondriasis "extraordinarily frequent in young people," and occurring sometimes in the years of childhood. Gull and Anstie express the general opinion when they say that it is scarcely ever seen under the age of puberty, and very rarely makes its first appearance after fifty.

A point which must always be kept in mind is that a person labouring under well-marked hypochondriasis may also have organic disease—of the stomach, for example, or of the liver, or an aneurysm of the aorta. Many a patient has had his complaints made light of until the signs of one or other of these diseases have become too manifest to be overlooked, or until he has died suddenly, from rupture of a large vessel, or from angina pectoris. Anstie further remarked that in certain cases the possibility of malarial poisoning or of chronic alcoholism must be thought of before one sets down a patient's symptoms as the result of mere hypochondriasis.

The *treatment* of this disease is commonly difficult and unsatisfactory. An essential point is that one should not attempt to make light of the patient's sufferings, but should show oneself to be really interested and anxious to relieve them. Exercise, short of severe fatigue, should be taken daily, and mental occupation and amusement should be cultivated with the greatest care. Sea-bathing, the cold water cure, the warm baths of Gastein, may each be prescribed with advantage; but it is difficult to say how much of the benefit is due to change of air and scene, and alteration in the habits of life. Any disorder of the digestive organs must of course be carefully investigated and corrected. The continual exhibition of laxatives is injurious; it is better to depend on diet and exercise to procure a healthy state of the bowels. But an occasional blue pill and black draught are most valuable in many cases, and may be repeated once or even twice a week. Valerian is said to be sometimes useful, and so, with some patients, is tincture of sumbul. Neither quinine, strychnia, nor phosphorus is generally of service, nor iron, unless there be marked anæmia. Anstie rightly laid stress on the importance of not prescribing alcohol for hypochondriacal patients, even in the form of medicinal tinctures, lest by doing so one should encourage habits of over-indulgence in drink.

In weighing beforehand the probable success of treatment in a case of hypochondriasis, one has to take into consideration not so much the apparent severity of the symptoms as the length of time they have lasted and the circumstances under which they began. The younger the patient the better the prospect of cure; particularly if the family history is good, so far as regards the occurrence of insanity or other nervous diseases in his near relations.

PSYCHOSES

OR

DISORDERS AFFECTING THE MIND

(*By G. H. SAVAGE, M.D.*)

"Mentem sanari, corpus ut ægrum,
Cernimus et flecti medicinâ posse videmus."—LUCRETIUS.

Insanity as a (1) *disorder of functions of brain;* (2) *an expression by the nervous system of bodily disease;* (3) *the expression of disease of the brain—Classification—Causation.*

Acute delirious mania—Acute and other forms of ordinary Mania—Melancholia with bodily complaints—Hypochondriasis, with mental complaints—true Melancholia—Stupor, active and passive—Delusional insanity—Hallucinations of the senses—States of mental weakness—by defect—by instability—Specially named varieties of insanity: puerperal, alcoholic, moral, syphilitic, gouty, plumbic, febrile, phthisical, asthmatic, cardiac, renal and diabetic—Epileptic insanity—Paralytic and tabid insanity.

Testamentary capacity—Certificates and other medico-legal points.

Idiocy and imbecility.

IN approaching the study of insanity, it is first of all important to remember that we have not a definite disease, such as typhoid fever or phthisis, to investigate, but that the condition or state called insanity depends, not only upon the evident symptoms, but upon their bearing on the social environment of the individual. There is, in fact, no absolute standard of sanity, and the divisions of the condition called insanity are to a great extent arbitrary. Insanity will have to be here considered chiefly from its clinical or medical aspect, but yet its social and its legal aspects must not be entirely neglected. Though there is no standard of sanity, yet it so happens that disorder of the nervous system expresses itself along more or less definite lines, and we shall have to point out the pretty regular association of symptoms as they occur in the different groups of insanity.

First we must recognise that while some cases of insanity depend upon cerebral disease or degeneration, others depend upon diseases of the body, which are referred to the nervous system; so that there is in the latter cases a mental aspect to a bodily disease. Thirdly, we shall have to consider the most difficult of all cases—those in which there is disorder of function without any visible pathological change in the nervous system. So, then, we have (1) disease of brain with disorder of its function; (2) disorder of some function or functions of the body with insane or nervous interpretations; and (3) disturbance or disorder among the functions or parts of mind.

While recognising *forms* of insanity, we must remember that these forms merge insensibly into one another, that no symptoms are absolutely characteristic of any one form of mental disorder. The *forms* are, then, arbitrary,

but more or less convenient, groupings of symptoms. The brain and nervous system have their special ways of expressing disease, just as the lungs, the stomach, or the kidneys have their special symptoms or ways of expressing theirs. We must look for the chief symptoms of insanity in either perversions of sensations, perversions of will, perversions of emotions, or loss of control or disarrangement of one or more of these parts of mind. The factors of mind for our present purpose will be considered to consist first of all, and perhaps most necessarily, of the inherited nervous system of the individual—the nervous basis; to which is added the experience gained by the various special senses—*perceptions*; and these bound together and arranged by *memory* form a structure upon which the *will*—balance of motives—and the *emotions* are supposed to play. And in each case of insanity it is well carefully to inquire first into the inheritance, next into the normal or abnormal working of the parts of mind as evidenced by the psychical and muscular expression. In considering the forms in which insanity appears, it will only here be necessary to make a few characteristic divisions, as the following:

Melancholic states, in which there are mental and bodily weakness and excessive self-consciousness.

Maniacal states, in which there is weakness with loss of control.

Dementia, in which there is weakness with more or less evident defect in mental function.

Beside the above, there are states due to the misleading by false sense impressions. These have been grouped under *Delusional insanity*.

The above are the more common groups into which symptoms of insanity form themselves. But these symptoms may occur in two distinctly opposite pathological states. They may occur as symptoms of disease of the brain, as seen in the degenerative process called "general paralysis of the insane," or they may occur in what we have seen to be the second and third groups of insanity. In fact, brain disorder, whether this occurs with brain disease, or bodily disease, or a disorder of mental function, expresses itself very much in the same way. Just as one may meet with cough which is due to disease of the lungs, cough which is due to a spasmodic condition, as in asthma, and cough due to defects in cardiac circulation, so we may have delusion due to organic disease of the brain, to bodily disease, as bad supply of blood, or to disorder of function, as seen in false sensory impressions.

Insanity occurs among the civilized and uncivilized, but in its more marked and serious forms it is specially a disease of the more highly civilized, and it will be seen that the more fatal forms of brain degeneration seem to increase directly in proportion to the high pressure of town life. Insanity is not, however, rapidly increasing in England. The statistics of the Commissioners in Lunacy show that whereas in 1875 there were 24·75 males, 28·43 females per 10,000 of the population in England and Wales insane, there were in 1885 respectively 26·61 males, 30·80 females per 10,000 in England and Wales insane, so that the increase is only about two per 10,000; and when we consider the greater general longevity and the consequent accumulation of chronic cases in asylums, it is pretty clear that there is no such increase of insanity in England and Wales as to cause real alarm. In further evidence of this the Commissioners' reports show that the admissions of fresh cases in 1886 were really fewer than the admissions of fresh cases ten years before. In fact, in 1875, while 5·36 men and

5·18 women per 10,000 inhabitants were admitted as insane patients, ten years later 4·8 and 4·9 respectively only were admitted.

In considering the so-called *causes* of insanity, the student must be warned that it is extremely rare to find one definite cause producing any attack of mental disorder, and it is therefore better to use the term *conditions* rather than *causes* of mental disorder. Again, though it is perfectly true that certain vital conditions, such as race, stage of civilization, climate, and the like, deserve to be considered as conditions predisposing to insanity, yet, in a short chapter such as the present, it is beyond our scope to consider more than a few of the general conditions which give rise to mental disorder. These causes may be divided for convenience into those acting chiefly upon the mind, and those acting chiefly through the body—the so-called *moral* and *physical* causes. These again can be divided into *predisposing* and *exciting;* and though some causes act distinctly as moral causes, yet it will be found that in the majority of cases one cause may be both moral and physical, and may also act as a predisposing and as an exciting cause. Take, for example, alcoholic stimulants, which act primarily as a physical and predisposing cause; but the associations of the drunkard are likely to lead him into moral troubles, which again react, and a fresh outbreak of drink and excitement may be the real exciting cause of the outbreak of insanity. The most common *moral* causes are mental anxiety, worry, overwork, money losses, religious excitement, love affairs, domestic trials, fright, and occasionally other emotions. Among the *physical* causes, intemperance, organic disease of the brain, injury to the head, venereal excesses, masturbation, epilepsy, pregnancy, parturition, and lactation are the more common causes assigned in England, and in addition to these, one of the most important conditions of insanity is *heredity*. It is extremely difficult to be sure of the part played by inheritance, yet it is sufficiently certain that a large proportion of people are insane because of the insanity of their parents. The insanity which is passed on from parent to child has special peculiarities, and will have to be referred to more in detail later on. There seems in such cases to be liability to disorder of function rather than to disease of the brain itself, and there also appears a greater predisposition to recurrence in cases with strong neurotic inheritance. Besides direct inheritance we have to remember that insanity may be developed in families where epilepsy, hysteria, hypochondriasis, some spasmodic nervous affections, and the like occur; and it is probable that if to these conditions in one parent be added any special cause of organic degeneration in the other, the offspring will be rendered more unstable, that is, more liable to fall out of step with the other members of a civilized society.

Causes of insanity act directly, *i. e.* immediately, only in very rare instances. The mischief is much more commonly the result of frequently recurring disturbance; and therefore in considering any case of insanity with reference to causation it is important that changes in temper, disposition, and habit should be carefully investigated. It must be understood that causes in the majority of cases are not easy to be traced, and in many cases the supposed *causes*, such as sleeplessness and dyspepsia and even alcoholism, may really be the earlier *symptoms* of the disease.

It is necessary to refer to a few of the general conditions in more detail. In cities degenerative changes and general paralysis are most common, whereas the latter is almost unknown in peasant races, such as the Celtic population in Ireland, Wales, or Scotland. Education is a cause of insanity if ill suited

to the nature of the person, but over-education alone is very rarely the cause. More women than men become insane, but more men have general paralysis and more women recover from insanity. Puerperal and climacteric conditions affect women. Insanity of any form may attack patients of any age, but in childhood the tendency is to idiocy, in early youth to imbecility, in youth to weak and impulsive mental states; in middle life the greatest amount of acute insanity occurs, as well as most general paralysis, at the climacteric melancholia and delusional insanity are frequent, and as age increases the tendency is to dementia.

Solitude and sedentary occupations followed apart are very dangerous conditions; hence widowhood is of evil influence. Domestic worry chiefly affects women, and business worry and anxiety men. Love affairs and sudden shocks, whether painful or pleasant, affect mostly women and very young people. All causes producing excessive nervous exhaustion affect the young most; as do also the abuse of stimulants and chronic poisoning, such as that by lead.

Injuries are not frequent causes of active insanity, but lead to diseases of degeneration. Tumours and coarse lesions of the brain, syphilis excepted, are also rare causes. Religious and political excitement act mostly as exciting causes. Fevers and any causes of delirium may start insanity.

Bodily diseases causing pain or weakness may set up nervous disorder. Insanity may depend on bodily disease, or on physical disfigurement and its moral effects. Insanity may alternate with other nervous diseases, such as epilepsy or hysteria, or it may alternate or replace other nervous or bodily diseases, such as asthma, gout, rheumatism, eczema.

ACUTE MANIA.—By acute mania we mean a state of mental weakness with marked loss of control. Under this head we shall have to place a variety of disorders extending from delirium to simple hysteria, and it will be convenient to make two distinct groups of the maniacal cases.

There is a well-recognised and well-understood condition—delirium; and formerly it was insisted that there were essential differences between mania and delirium, but it is certain that there are connecting links. There are cases in which there is some delirium but much maniacal excitement, and others in which there is little maniacal excitement and more delirium. It is well, then, to remember that when speaking of patients suffering from mania, we may have to consider either those in whom there is great bodily and mental disturbance or those in whom there is great mental disturbance with little or no bodily disease.

Acute delirious mania is a term used to express the symptoms occurring in the cases of mania in which the delirious element is most marked. Typhomania, brain fever, and other terms have been used, but it seems that we still are in want of some general descriptive term which will include all the cases which deserve to be grouped together, because, though most of these delirious cases are more or less maniacal, yet there are examples in which all the other bodily symptoms are present, but instead of mania there is either melancholia or partial weak-mindedness, or at least confusion of thought. Acute delirious mania is a disease most commonly met with in young people of both sexes. It is common in the highly nervous, the accomplished and educated. It is generally ushered in by a slow process of change in temper, or by general feelings of uneasiness, restlessness, malaise, or hypochondriacal symptoms. It is common to meet with young women

who have had some disappointment in love, some shock, or who have suffered from some physical disorder, producing nervous weakness or anxiety; they complain of sleeplessness and headache, loss of appetite, amenorrhœa, and general inability to apply themselves to ordinary occupations. This may be the early stage of acute delirious mania. Acute delirious mania may also follow shock, fevers, such as scarlet fever, measles, and the like; poisoning, as from belladonna, from intemperance in alcohol or ether, and in my experience it may also follow the inhalation of chloroform. It is not confined to young people. The period of depression may be followed quite suddenly by one of excitement, exhibited by voluble talking, exaltation of ideas, or not uncommonly by a rather sudden and startling development of erotic excitement. Within a few days the delirium has reached its height: the patient then has the appearance of one suffering from typhoid fever, lying on her back with a tongue dry and brown, or thickly coated, with sordes on the lips, the eyes suffused, the cheeks either sallow or (during periods of excitement) brightly flushed, the lips moving without any audible result, the hands twitching or picking the bedclothes, the voice, if heard, harsh and raucous, the skin moist, with but slight increase of temperature; then rapid emaciation, tendency to bedsores, and excitement recurring irregularly, but most marked at night, with little or no reaction to the surroundings. There may be hallucinations of sight and hearing. There may be periods of tranquillity and apparent recovery followed by periods of excitement. During the period of excitement there seems to be no memory. Patients recovering have little or no recollection of what has happened during their illness. It is almost impossible to say how long the period of excitement will last, but from a few weeks to two or three months is the limit, and it must be remembered that patients suffering from acute delirious mania are passing through a process in which exhaustion is the chief characteristic, and the excitement is invariably followed by a more or less prolonged period of depression, the depression not being that of melancholia, but rather that of inability to will or to desire, so that they are in a will-less, childish condition. From one third to one fourth of the young cases suffering from acute delirious mania die, and of the others probably a full third remain permanently weak-minded. Some rapidly gain flesh, but remain dull, apathetic, and unlike their former selves. Others pass into general paralysis.

The chief points in *treatment* are to feed freely and abundantly. It is well to begin with a free purge by means of calomel or croton oil, and then to follow up with beef-tea, mutton broth, milk, brandy, and eggs. It is almost certain that artificial feeding will be required. Feeding by means of a nasal tube—or better, by putting a funnel in one nostril, and, while controlling the other, slowly pouring the fluid food down the nostril—will be found of great service in these cases. The feeding by a stomach-pump, by the rectum, or by the nose, must be repeated every three or four hours, and it is of the utmost importance that stimulants should be given without stint. In old days it was feared that with such apparent excitement of the brain stimulants would have an injurious effect, but all modern experience is that stimulants, instead of causing excitement to these patients, are more likely to produce rest. In many cases the excitement is so extreme that some form of restraint, medical or mechanical, must be used. Chloral hydrate, in doses of from fifteen to thirty grains at night, and repeated in ten-grain doses two or three times a day if necessary,

may be tried, but it is better to do without any narcotic if possible. In strong, young, active people the wet pack, *i. e.* the rolling the naked patient in a sheet wrung out of tepid water, and then rolling one or two blankets outside this sheet, and placing the mummified patient on the bed for from one to three hours, will produce rest and quiet, but this treatment should be carefully watched by a skilled nurse, and during the process food and stimulant should be given. In some cases, in the earlier stages, warm baths with mustard in them will produce a stimulating effect upon the skin and secure a good night's rest. The great point to remember is that patients suffering from acute delirious mania are suffering from a disease which, as a rule, either rapidly kills or rapidly passes off, leaving the patient much exhausted, and as soon as the acute attack has passed it is of the utmost importance that they should have careful nursing, without much change in their surroundings. After the illness rest and quiet in the country or by the seaside is preferable, in the writer's opinion, to travel; and it should be remembered that in such cases intellectual work should not be undertaken for a twelvemonth from the onset of the disease.

Complete or only partial recovery may take place; the patient may be left more or less morally or intellectually crippled, and in some cases not only is there defect of mind, but, following the acute delirium, some more or less permanent wasting with contraction of the lower extremities may occur, and the writer has met with cases of delirious mania in which general paralysis has followed and made rapid progress.

There is no very marked tendency to the recurrence of attacks of delirious mania. In fact, delirious mania differs in that as it does in other respects from many forms of ordinary acute mania.

ORDINARY ACUTE MANIA.—In this condition, unlike the last, we have more organised expression of delusion, and more marked loss of self-control with less bodily disturbance. The conditions under which maniacal excitement appears are endless, depending much upon the inheritance, education, race, and other peculiarities of the individual. We meet with every variety, from simple uncontrolled lust to violent homicidal destructiveness, and no one description can fully cover the whole line. Mania may occur as only part of a morbid mental process; it may follow a melancholic stage, or epilepsy; it may be part of *folie circulaire*, or recurrent attacks of insanity; it may be part of the symptoms in general paralysis of the insane or of dementia due to age or definite brain disease. But in all cases the symptoms are sufficiently alike to deserve one description.

The *causes of mania* are in no way special. Mania may follow from moral or physical causes, may follow injury to the brain, direct or indirect, or may follow upon bodily disease or disorder. A shock may produce either mania, melancholia, or dementia.

Nearly all cases of mania begin with uneasy feelings about the epigastrium, occasionally with feelings either of emptiness or fulness of the head, rarely with headache or photophobia. Sleeplessness and fear of impending ruin are common, anorexia, constipation, restlessness, inability to apply themselves to work, irritability, and emotional weakness. This period may last from a few weeks to several months, and may become extremely marked and profound. After the period of depression the patient may almost suddenly grow more restless, more excited, and markedly loquacious. He may say that he has been ill, but that now he is quite well,

and he may become extravagant, boisterous, and over-generous. He may discover that he does not require to sleep so much or to eat so much as he did, and he may be full of ideas that he will be able to make a fortune by some invention. Any interference by his friends is resented, and in many cases such early interference precipitates the attack of mental excitement. Sleeplessness of a restless type is marked, appetite is variable, a craving for stimulants may occur. The bowels as a rule are confined, the skin is often sallow, but of normal temperature, hallucinations of sight and hearing are not uncommon in the earlier stage, delusions as to property, wealth, position, and also as to the actions of friends, are common. Will as a rule is unstable, so that the patient is incapable of persistent effort. He is often emotional and irritable, but his memory is so far good that he remembers what is said or done, though things appear to him different from what they would in health. Sexual desire is frequently great, but there is probably no increase of sexual power. The general bodily condition is one of weakness, and the general mental condition is one of instability and mobility.

The two things that one has to guard against chiefly are the reduction of strength and the infliction of injury to self or others; the real question of treatment depends almost entirely upon the question of danger to the patient or his friends. Mania may end fatally from sheer exhaustion; this occurs mostly in elderly patients, or in people exhausted from some general bodily disease. A large number of maniacal cases get well, but a certain number remain either permanently weak-minded or permanently unstable, so that they are constantly liable to recurring attacks of insanity. Of those who do not sink in the earlier stage the period of excitement may last for weeks or months; probably four or five months is an average period for acute maniacal excitement to persist. During this period there will almost certainly be intervals during which the patient is more quiet and apparently improving, and it is noteworthy that after a night's sleep, produced either by narcotics or as a result of exhaustion, the patient may appear to be improving; but we must be prepared for remissions and relapses in the course of ordinary acute mania. During the maniacal excitement destruction of clothes and filthiness of habits are very common and distressing. Each individual case exhibits peculiarities in its course, symptoms, and termination. Patients often suffer from some bodily ailment before the mental storm begins to pass; one will complain of headache, another of neuralgia, one of rheumatism, and another of restless sleepless misery connected with some trifling bodily ailment before the disappearance of the excitement. As a rule the symptoms disappear slowly, it being noticed that the patient is getting more self-control, has greater desire to see friends or relations, writes letters home, is anxious to see to his business, and very probably may be discontented with his situation. Discontent in persons suffering from mania is equivalent to the returning consciousness of a man who has been stunned; and just as in the latter case there is for the patient himself a painful consciousness, while for the onlooker there is the satisfaction of sensibility returning, so also discontent in the maniacal patient often means returning consciousness. In proportion as the patient has been excited will there be depression or mental exhaustion; and it is pretty certain that a prolonged attack of mania will be followed by a prolonged attack of mental exhaustion or depression, during which there may be more or less sleeplessness and loss of appetite, with general mental weakness; but in these cases

change of surroundings and as speedily as possible a return home will be found the best treatment.

From a practical point of view, all powerful narcotics and depressants are merely means to keep a patient within control, and not means of really curing our insane patients. It will be necessary in some cases to try chloral, bromide of potassium and the like, and it undoubtedly in a few cases will be found that bromide of potassium alone or combined with chloral will lessen the excitement and give time for the patient to recover. But the most important consideration is that, whatever treatment is followed, we must be sure not to affect the appetite or we shall have cause to regret it. Abundant nourishment, with or without stimulants, associated with exercise in the fresh air and as much freedom as is possible, should be the treatment for such cases. Sulphonal is useful and appears to be harmless.

Before leaving the consideration of this group, we must mention the patients who have suffered from acute mania and who never permanently recover. Some, as already stated, remain ever after liable to recurrence of insanity, others remain weakened in some special way so that they are unable to fulfil their duties as before. Thus, one individual will be morally weak and is called either a kleptomaniac or a drunkard, another becomes emotionally weak and takes to ambitious schemes for reforming his fellows or to spending day and night in religious exercises, a third becomes volitionally weak and allows himself to be looked after and attended to by his friends without any desire to help himself.

It is only necessary to add that, if the antecedent stage of melancholy be long, the maniacal one is likely to be prolonged also.

After epilepsy very violent mania is frequent, and chloral given by the mouth or the rectum may check the mania if administered early after the fit. Cases in which the mania depends on senile changes are less favourable but not hopeless.

Maniacal excitement due to general paralysis generally passes off in a few weeks. Mania due to alcohol is very variable, but may last for weeks or months.

MELANCHOLIA.—By the term *melancholia* we mean a state of unreasonable mental depression; grief without a real cause, depending rather upon a physical than a moral determinant.

Melancholia may be but part of the disordered process or may be the whole of the morbid state. In most cases of acute mania and in most cases of general paralysis of the insane there is a stage of melancholia which ushers in the acute symptoms. Melancholy as a form of mental disorder occurs in all ages, though probably it is most commonly met with in and after middle age. It is most common in the dark complexioned. The symptoms of melancholia may depend on general depression of the vital powers, or it may depend upon some special diseased process, as, for instance, heart disease. The most characteristic symptom at the onset is extreme self-consciousness. This develops into grief which is variously expressed; irritability of temper, sleeplessness, loss of appetite and suicidal tendencies develop in order. Melancholia has been divided into many varieties. The chief methods of division depend upon the outward expression of the morbid grief. In some the symptoms are *active* and in others they are *passive*,—in the former the patient restlessly wringing his hands and openly complaining, while in the latter he is struck dumb by his misery. Melancholia is divided

not only by its expression but by its special aspects: in one the expression of grief is in relationship to the bodily functions, while in the other the mental symptoms predominate. The former may be called *hypochondriacal melancholia*, and the latter *true melancholia*. Every shade of melancholia may be seen in relationship to hypochondriasis, but in an asylum we see the more exaggerated cases which must be secluded in consequence of their desire for death and refusal to take food.

Hypochondriacal melancholy.—There are four distinct groups of cases which come under this head. First, there are those patients whose one complaint is that they are dying and that nothing can be done for them, though they make no definite complaint of any special organ.

Next we have a large group of cases in which the symptoms are referred to the *head*. Thus young patients will tell you that their brains are hot or wasting or hollowed or melted, and women about the climacteric will complain of opening and shutting of their brains, of something coming away when they move their necks, or the like. Older patients will complain of adhesions between the brain and the skull, or of miraculous removal of brain and nerve power.

The third group includes those who believe that some terrible calamity has befallen their *digestive tract*. One patient believes that his throat is stopped: these symptoms are allied to exaggerated hysterical globus. Another is sure that his stomach is ruptured or imperforate, or that some connection exists between the stomach and the circulatory system. A few patients imagine that the rectum is permanently closed. All these cases have to be looked upon as exaggerations of hypochondriasis, cases in which every hypochondriacal symptom is greatly developed.

The last group of insane hypochondriacs contains those who believe that some trouble arises from their *reproductive organs*. Middle-aged men believe that they have been rendered impotent, that their testes have withered, or that by some evil habit or by some course of medicine they have become emasculated. Such cases are more commonly met with among men than among women. It is comparatively rare in asylums to find women with uterine and ovarian hypochondriasis. There are young sexual hypochondriacs whose disorder is partly due to masturbation, and these have varying disorders of the brain.

In all cases of hypochondriasis the one object of *treatment* must be the withdrawal of the attention as much as possible from the sensitive part by occupation, while the general health is being improved. Judicious change of air, change of scene, travelling and mechanical occupation, with withdrawal from old associations and companions, are of the utmost value. Such cases recover in a fair proportion, but if past middle age, or if the development of morbid ideas has been slow, and if there be no definite physical illness associated with the mental disorder, the prognosis is bad. Such patients rarely become weak-minded, but persistently hold to their hypochondriacal delusions, which may remain for many years, the rest of the intellectual life of the patient being normal.

Ordinary melancholy.—Ordinary melancholia consists in emotional depression, in which the explanation of the feelings is moral and not physical. It may be the result of shock, or grief, or bodily illness. In certain predisposed persons any cause which reduces the vital standard below a

certain point seems to be able to produce melancholia. The first symptoms are, as a rule, mere uneasiness, restlessness with inability to take interest in surroundings, tendency to tears and emotional disturbance, dread of some impending calamity, with undue sensitiveness to the conduct and the remarks of others. These symptoms are associated with loss of appetite, sleeplessness, and feeble circulation, as seen in winter by chilblains and cold extremities, together with loss of appetite, constipation, and amenorrhœa in women. These evidences of reduced power are all part of the process, and may rapidly pass into most pronounced mental depression. Up to this time there has been a feeling of misery, but there has probably been little or no explanation as to its cause. Later hallucinations of the senses or delusions may arise, and these depend to a great extent upon the education, age, and immediate surroundings of the individuals, so that the young woman thinks that her virtue is called into question, while the old man thinks that he is going to the workhouse. It is common to hear patients in this condition refer to their past wickedness, and to say their souls are lost, that they are possessed by the devil, that they are "the Scarlet Woman," that they are metamorphosed into beasts, or are unnatural, and that they ought to be dead. At this time they will be sleepless, and probably will refuse to take food voluntarily. There will be complete inaction of the bowels and general apathy and listlessness; suicidal tendencies become marked, and it is important to remember that patients as a rule select a special form of suicide which they prefer, and will wait for an opportunity rather than seek death by any other means. Suicidal attempts are mostly to be feared when patients believe themselves to be impotent, when they believe themselves to be injuring their nearest relations, or when they dread being tortured "to make them confess," or when they believe themselves to be followed and dogged about. Patients who "hear voices" are also very liable to suicide. The melancholic process is, as a rule, longer than the maniacal one, and an average of from six to eight months is required for recovery. The symptoms, after slowly advancing, may slowly recede, the prognosis depending really upon the steadiness of the improvement both in mind and body. In some the cure is sudden after sleep or after some sudden improvement in general health, as improved digestion or the like. Perfect recovery may take place, or, what is more common, there may be a slight excess of excitement after the melancholia has passed away. There is a great tendency to relapse in melancholic patients, especially in those belonging to an insane stock. A certain number of patients die from melancholia, but as a rule the cause of death is secondary to the mental disease, there being some congestion of the lung or extreme exhaustion and emaciation depending upon bad nutrition or insufficient feeding. In cases of chronic melancholia wasting of the brain is well marked, and the pia mater is sodden and easily separable.

The *treatment* of melancholia naturally divides itself into preventing harm and doing good. The first essential is to prevent suicide, and this is only to be done by constant watching, by the utmost care in the selection of nurses, and by insisting on the patient having his rooms on the ground-floor. Rest in bed, especially during the colder months, is valuable, warmth and food, with stimulants, being very important. In some cases of active melancholia morphia may be useful. When patients are treated at home, it is necessary to give narcotics, the rule being not to give them continuously, to avoid giving them in increasing doses, and to vary them as much as possible.

The next most important thing is the method of feeding. Patients

who refuse their food absolutely may be fed by the rectum, by the mouth, or by the nose. As a rule, patients who resist should not be fed by enemata. In feeding by the mouth, if sufficient care and time be taken in nearly all cases food may be given in small quantities by the spoon being introduced by the side of the teeth. But if resistance be great it is better to pass a nose-tube or the stomach-pump. The nose-tube is very easily passed, and nurses can be trained in a few lessons to do it without danger; a large-sized soft catheter fixed on the end of a small glass funnel is all that is required, the tube being passed down one nostril, while the other nostril is compressed by the finger. Three to five pints of milk, six eggs, two pints of beef-tea or broth, and four ounces of brandy, may be given in twenty-four hours.

Among the forms of melancholia the most important are *simple melancholia*, that is, melancholy without delusions; *melancholy with stupor*, to which we shall refer again; and *active* or *resistive melancholia*, in which an active expression of grief occurs. There is no special form of melancholia deserving the terms "religious" or "suicidal."

Simple melancholia is often present in men of middle age and of active intellectual habits. Misery without cause, self-accusation about trifles, sleeplessness and loss of appetite, inability to apply the mind or the attention, are the chief symptoms.

A month's rest under supervision, away from wife and friends, followed by restful and recreative travel, are all that is needed; but probably twelve months' rest from mental labour will be beneficial. This state may occur in much-examined youth, in much-worked or worried manhood, or may appear at the climacteric or decline of life. Suicide is common. There are other varieties of melancholia associated with bodily disease. Thus, undeveloped or suppressed gout may be represented by melancholia, and phthisis may show itself only as progressive weakness and refusal to take food, with suicidal impulses. Melancholia, if of an active type, often benefits if morphia be given, and in young cases shower-baths are of service. In gouty and simple cases a course of Turkish baths will often work wonders.

STUPOR, INCLUDING ACUTE PRIMARY DEMENTIA AND MELANCHOLIA WITH STUPOR.—Under this one head two very distinct groups of cases are included, but in both of these the characteristic symptoms are the stupor, the silence, and the abstracted appearance of the patients. According to some there is no such thing as acute primary dementia without delusions, all these patients owing their mental attitude to some dread or terror. In fact they believe such patients to be in a kind of chronic panic, but it will in any case be necessary to distinguish between two groups occurring under this head, and for convenience we will call them the active and passive groups.

Active Stupor.

Common in adolescents, who have a terrified expression, are wasted and thin, with skin purple and cold, resist or are cataleptic, refuse food, are sleepless, wet, and dirty, have distinct delusions of dread, retain memory through the attack, refuse all food and require abundant forced feeding with stimulants; electricity and massage may be useful.

Passive Stupor.

The passive form occurs more frequently as the result of some cause of exhaustion in young or middle-aged people, who have a silly aspect, muscles are flabby, skin greasy, limbs relaxed; they eat what is given them, sleep well, are clean if watched; there is no evidence of the existence of delusions, little or no memory on recovery; and for treatment they require baths, exercise, and general tonics.

The above groups resemble one another in history and in progress to a great extent; most of them occur in young adolescents. It seems as if in certain weak-minded persons there is energy enough for boyhood or girlhood, but when manhood and womanhood comes on it fails. With such cases shock, grief, masturbation, disappointment, overwork, unhealthy occupations, fevers, intemperance, rapid childbearing, or the like, may suffice to produce the breakdown. When once the condition of stupor has been established, as a rule it persists for several months, though occasionally there are recurrences of stupor between intervals of sanity. Little or no good comes to these cases unless the general health can be markedly improved. The patient, if in active stupor, is probably more or less cataleptic, and has to be forcibly fed. He wastes, and is wet and dirty, is very liable to secondary inflammations, and often has chilblains. He takes no notice of his friends. The stage of stupor is of very variable duration and may end in death, in slow or sudden recovery, or in partial weakness of mind.

This state of mere passive stupor may be uniform and persistent, but commonly it is associated with periods of temporary recovery or of periods of excitement, or with sudden outbreaks of impulsive violence. This passive state frequently results from or follows an acute stage of mania, or develops after some severe and exhausting bodily illness.

Feeding, warmth, and watching are of great service. One danger in such cases is death from some secondary complication; local inflammation or phthisis may develop and terminate rapidly. After recovery there is frequently a period of exaltation during which sexual desire and tendencies to lust and intemperance may arise. The prognosis depends greatly upon the family history. If neuroses are common in the family, and if the patient is very young or badly developed, or if he have a badly shaped head, or any tendency to phthisis, the prognosis becomes absolutely bad. If a patient recover from stupor it is very necessary that for some years he should be carefully watched, and he should not return to any occupation which involves severe strain either of mind or body, so that it is much better after recovery from stupor to send the patient for one or more sea voyages, or, if possible, to let him follow some mechanical or outdoor life.

DELUSIONAL INSANITY.—So far we have considered states in which there have been perversions of the whole mental functions associated with more or less bodily disturbance, but under this head we have to consider cases in which the perversion of the senses is the chief symptom. The expression "out of one's senses" is recognised as meaning madness, but only some patients are definitely out of their senses. Such are the cases now to be described. It can readily be understood that as our intellect depends to a great extent upon the impressions we receive and store up from our senses, if our senses mislead us the intellectual result will differ greatly from that of the ordinary standard. The cases which I have to describe depend chiefly upon hallucinations, which are sense impressions not depending upon any external or objective impressions. Thus a person who in the stillness of the night hears "*voices*" has hallucinations of hearing, and the person who, similarly situated, has *visions* or smells *poisons* or tastes *filth* is suffering from hallucinations of the senses of sight, smell, and taste respectively. Hallucinations of hearing are the most common, next in order we have hallucinations of sight, then in order those of touch, taste, and smell. Nearly all hallucinations may be reckoned as painful nervous impressions. The explanation which is given

of the sensation depends to a great extent upon the education and occupation of the individual.

Patients suffering from *delusional insanity* are generally past middle life, single or widowed, often of insane family, leading solitary lives or fulfilling positions which are uncongenial to them in one way or another. Hallucinations have been frequently traced to solitary confinement in prison, and I have met with several cases in which they have developed in men living isolated lives in the wilder parts of India and other English dependencies. As a rule the earliest symptom complained of is nervousness and nervous weakness. Patients become excessively sensitive, and this leads to a condition in which everything seems to point to or to affect the individual; his mind is like the tender corn to the walker, and just as every movement seems to affect the latter, so every action of others seems to be directed against the former. He becomes suspicious, jealous, bad-tempered, more and more solitary, and this increasing solitude further develops the ideas of *suspicion*. He believes himself to be *persecuted* and *followed about*. He may think that his thoughts are read by others or appear aloud to himself and to others so that they read his secrets, he hears people coughing, jeering, or making allusions to him or to his family. He may hear words such as "devil," or worse, constantly whispered in his ears; he may then take the law into his own hands and assault some one whom he believes to be his persecutor. The ideas of interference may become more organised, so that he is convinced that bodies of men, such as the Jesuits or the Freemasons, are interested in his ruin. Under these circumstances the patient becomes exceedingly dangerous, and, unfortunately, it is very hard to break through the hallucinations and to appeal to him by means of any other ordinary sense impressions which might in time loosen the bands of the false sense impressions. If these symptoms have developed slowly for twelve months the prospect of recovery is very slight. Patients who believe themselves to be followed or persecuted should be most carefully secluded or watched with the utmost care as they are both homicidal and suicidal. They are sometimes so exceedingly suspicious that they will deny their delusions so as to mislead those of whom they are afraid. Besides those who believe themselves to be persecuted there are others who believe that everything refers to them, and others who believe that the whole of their sense impressions are make-believes,—that the whole world is a kind of play which is being acted before them.

The general *treatment* of cases suffering from delusional insanity must depend very greatly upon the nature of the delusions, and the length of time which they have existed. Medical treatment is practically useless, but in some cases persuasion, associated with change of surroundings, works wonders. One young man believed that his relatives jeered at him from the roof, and that others made use of foul terms against him and them. This patient was excessively violent, yet by employing him, amusing him, and bringing distinct irrefutable evidence that these people did not exist on the roof and did not accuse his friends of iniquity, we slowly brought him to be more amenable to discipline, and at the end of twelve months he was discharged so far recovered that he has since emigrated and is now in perfect health.

CHRONIC WEAK-MINDEDNESS.—Loss of faculties, more or less general, may result in such extreme mental weakness that it is unsafe for the patient to be at large, as he would on the one hand be a tool for the wicked

and on the other a prey to the vicious. Chronic weak-mindedness, as seen in an asylum, may result from age, apoplexy, course of brain disease, or it may follow nervous exhaustion, general paralysis, brain tumour, epilepsy, or any other conditions of nervous or mental excitement.

Simple *weak-mindedness* is *gauged* by *loss of memory* and *loss of control*, and every variety may be seen, from the weak-minded imbecile to the lustful and dangerous, demented lunatic. The general course of ordinary weak-mindedness due to age and the like is as follows:—Progressive loss of control of the emotions, loss of memory and tendency to collect articles of no value on the one hand, while there is thoughtless and reckless extravagance on the other, a redevelopment of lust and a tendency to intemperance, which all lead to a wastful expenditure of the reduced nervous capital. Often patients who have led reputable lives up to sixty may pass through every phase of moral degradation before it is discovered that their immorality was the result of arterial degeneration. Such cases require to be recognised and to be rather watched than sent to asylums, if the means are sufficient, because the probable termination of such cases is an apoplectic fit or permanent childishness.

Among the subjects of chronic weak-mindedness we must include a large number of patients who, having suffered from acute attacks of mania or melancholia or stupor, instead of recovering their old mental balance, remain either *permanently crippled* or *permanently unstable*. The permanently crippled may exhibit their weakness by loss of one or more of the higher faculties, so that one becomes impulsive and homicidal, another ceases to respect truth and honesty and is called a kleptomaniac, another disregards decency and becomes altogether brutalized. In some a single faculty, such as memory, may seem to be affected out of all proportion to the others; the most characteristic loss after all is that of the highest social relationships. Patients who have not perfectly recovered from acute attacks of insanity invariably lose their affection for near relations and friends, and pass into a condition of philosophical calm or social indifference. Besides the loss of faculty already described, there are cases of permanent instability. Every asylum contains patients who are subject to recurrent attacks of mania or melancholia, patients who during twenty, thirty, or forty years periodically have recurrent attacks resembling in every particular the previous ones, with the probable exception that each attack is rather longer than the former, and leaves some more or less well-marked scar upon the intellect. Among these we have to recognise cases of chronic recurrent mania and of chronic recurrent melancholia.

Another group remains, and that is of those cases which, after an acute attack of insanity, have a few symptoms surviving, organised as it were, so that the patient becomes automatic, with certain peculiar actions or modes of thought fixed. Thus one patient for sixteen years sobbed out hour by hour, "I don't know what to do," and yet there was no sorrow in the later years, the lamentations having become purely automatic, and the patient eating well, sleeping well, and becoming fat. In other cases with chronic chattering incoherence, there may be persistence of some hallucinations, so that the patient is living in two mental worlds, or perhaps it were better to say between them; hearing faintly the real messages from the one, and more clearly the false impressions from the other. Many such cases, though violent at first, may be treated with sufficient safety at home, as is evidenced by the existence in so many country places of feeble-minded

villagers, ready to run an errand or enter into boyish games. Such weak-minded persons may be trained to perform mechanical tasks sufficiently well, so that the simple drudges of an asylum are often patients suffering from chronic feebleness of intellect.

So much for the permanently crippled. The permanently unstable are those who though fairly reasonable during most of their lives are constantly subject to fresh attacks. In some, the recurrences are at long intervals, and are comparatively harmless to the nervous system; in others the frequency or severity of the attacks causes progressive destruction of mind.

The symptoms may be maniacal or melancholic. Each attack as a rule resembles its predecessors in mode of development, symptoms, and mode of termination, but each attack may further impair the stability.

A patient may have an attack of acute mania and recover, a second attack occurs in four years, a third in three, a fourth in two years, and after that one or more attacks may occur yearly till the patient either passes into chronic mania or into hopeless weak-mindedness. In cases of recurrence, if maniacal symptoms are present, powerful drugs such as hyoscyamine in one tenth of a grain, given subcutaneously, may check the outbreak or the recurring habit, especially if the drug be given when the earliest sign of disturbance is noticed.

In recurrent cases of melancholia with suicidal ideas, with each recurrence suicide must be guarded against. Therefore such cases are always anxious ones if allowed to be at large.

With the unstable cases hereditary tendency is common. Such cases may live to an ordinary age. In some cases a weak-minded state gives place periodically to outbursts of violence.

PUERPERAL INSANITY.—There is no special form of insanity deserving the name "puerperal" yet, as the puerperal conditions are not infrequently causes of insanity, the term Puerperal Insanity is convenient.

Insanity may be developed during pregnancy; it may be but an exaggeration of the longings of pregnancy, and on several occasions a distinct connection has been traced between some absurd or morbid appetite and a neurotic tendency.

Beside the mere longings of pregnancy and their insane exaggeration, one meets with true insanity occurring early in pregnancy. This is most common in women who have already suffered from some form of insanity before. Thus, a woman who has had one or more attacks of puerperal insanity is liable to a development of this disorder with a succeeding pregnancy. Insanity of pregnancy may occur in the earlier months, and may pass off spontaneously about the fourth month, or the insanity may develop during the later months of pregnancy, and in that case will probably not pass off till long after delivery. As a rule, the insanity of pregnancy is of a depressed type, the patient being suspicious, jealous, refusing food, dreading poison, and irritable. Sleeplessness, refusal to take food, and tendency to suicide or infanticide, are the most dangerous symptoms and those which have to be the most guarded against. The induction of premature labour is generally useless; and the best treatment is to improve the general health by change of scene, removal from home, or at least removal of children from home, withdrawal of husband's society, and careful watching.

The second group of cases of insanity with pregnancy contains those that

have become insane after the fourth month. Such cases have frequently suffered much from exhaustion, due to frequent childbearing, repeated lactations, and vomiting of pregnancy. They may be maniacal, but usually suffer from melancholia, with ideas of unworthiness, and are strongly suicidal and often infanticidal; some suffer more from simple nervous exhaustion and stupor. The treatment of these cases resembles that usual for such conditions—rest and food, and removal from home.

It is rare for the insanity to pass off permanently with delivery. In some cases during the pains of labour, and immediately after, there is a temporary improvement, but as a rule the sanity following labour in these cases is temporary.

The next class to be considered contains those who become insane soon after delivery. Puerperal insanity occurs in a very large proportion among women with distinct insane inheritance. The causes are of long standing, rather than sudden; repeated pregnancy, poverty, exhaustion from sickness during the pregnancy, or several of the above causes combined, and though there are few noteworthy symptoms till after delivery, yet we should be prepared to find, on careful investigation, that there have been threatenings and warnings which have been neglected.

Within two or three days of delivery occasionally there is a temporary or transitory maniacal attack allied to the febrile disturbance associated with the oncoming of milk, and, like that, the transient mania may be relieved by a free purge. It is noteworthy that in this transitory mania infanticide or suicide may occur.

Insanity occurring after labour is divided into that which befalls the puerperal woman at once, and that which develops within the first two months after delivery. The former contains the greater number of maniacal cases, and the latter the more melancholic. Puerperal insanity does not follow instrumental labours in any great excess. Quite natural and easy labours are frequently followed by puerperal insanity in predisposed subjects. Puerperal insanity has undoubtedly a great tendency to recur in the same individual, but may not recur unless she become again pregnant. Puerperal insanity may be characterised by either maniacal or melancholic symptoms, or from the first there may be stupor. The onset of the disorder is generally as follows:—Sleeplessness for several nights, with irritability, loss of appetite, and querulousness, especially against the husband and child; then increased irritability or anxiety, with ideas of poisoning and refusal to take food; afterwards appear hallucinations of the senses, violent outbreaks occur, or profound melancholy may develop, in the one case the woman being destructive, and in the other suicidal. During this period the milk and lochia may be natural; on the other hand, one or both may be absent. From a practical point of view it is necessary to attend carefully to the milk, and to follow the old wives' simpler methods of "dispersing the milk" rather than making use of belladonna and other poisonous drugs. Friction with castor-oil or the application of hot salt are useful.

An attack of puerperal insanity usually lasts for several months, and requires most careful watching and care. The patients rapidly emaciate, often neglect themselves, and develop some secondary disorder which often proves fatal. There is no more special danger in the insanity than there is in the puerperal condition, but the two combined require double care in their treatment. Abundant feeding is the rule, with removal from home, and the sparing use of chloral or bromide of potassium.

The period of excitement may be unduly prolonged, so that in some cases patients are maniacal for eight or ten months before there is any sign of abatement of the disorder. Among the most characteristic signs of puerperal insanity in its later stages is the patient's discontent and jealousy of her husband, her tendency to mistake those who are about her, and the appearance of a weak-minded stage which is extremely difficult to treat. Following an acute attack of puerperal insanity we must expect to have a prolonged period of depression; and as soon as the patient has ceased to be actively antagonistic to her husband and home, and especially as soon as she expresses a desire to return to her family, it is desirable to make some movements in that direction. It will be found at this period that the patient gets stout, indolent, sleepy, and sometimes is too contented with an asylum; amenorrhœa also exists, and frequently this amenorrhœa and general condition of apathy are only relieved by return to home and domestic cares.

In such prolonged cases a second pregnancy is particularly hazardous; and in all cases of puerperal insanity it is of the utmost importance to impress upon the husband the danger of another pregnancy occurring within at all events two years.

Seventy-five per cent. of puerperal cases recover, about 5 per cent. die, and 20 per cent. remain chronic. Of those who recover, the majority recover slowly; mental and bodily health improve together. Of those who die, some die from septicæmia, but more from exhaustion due to the weak bodily condition associated with the mental excitement. Of those who are uncured, a large proportion have had many previous attacks, but a certain number of patients do not recover from their first attacks of puerperal insanity, and it is therefore well to be guarded in the prognosis even from the first.

Beside puerperal insanity, we have a form of mental disorder associated with *lactation*. From two to three months after delivery patients become suspicious, sleepless, irritable, and inclined to wander from their homes. In some cases there is amenorrhœa, but more often menorrhagia, due probably to incomplete involution of the uterus. These patients are sallow and anæmic, with bad appetite and constipated bowels; they frequently suffer from hallucinations of smell, taste, and hearing.

As a rule they require merely tonic treatment. They recover in the course of two to six months if removed to healthy surroundings, and away from home cares and worries. They require warmth, food, stimulants, fresh air, and, in the summer, baths, and the seaside.

Alcoholic Insanity.—Although no one special form of insanity depends upon drink, yet there seems to be some relationship between alcoholic intemperance and the variety of insanity. It is therefore well to recognise the chief forms assumed by insanity depending upon drink.

Sudden outbursts of drink in nervous subjects will lead to delirium tremens, and delirium tremens, instead of passing off as it ordinarily does, assumes in such cases more or less the character of a chronic delirium. With some patients acute delirious mania follows an acute alcoholic debauch. In such cases the removal of stimulants will not suffice to cure directly, and the persistent use of opium is rather injurious than otherwise. Hallucinations of sight and hearing similar in all respects to those met with in delirium tremens occur, but are more persistent; refusal to take food is constant,

emaciation takes place rapidly, and, unless the patients are fed abundantly with milk and nutrient soups, a fatal issue may be expected. If the patient be young and strong, a purge by croton oil and an emetic of sulphate of zinc may be found the best treatment to start with. If the excitement seems likely to wear out the patient, chloral alone or with bromide of potassium may be given at night, but it is best not to give these drugs continuously or in too large doses.

Beside the acute mental disturbance produced by alcohol, we meet with every variety of delusional insanity, patients believing that their food is poisoned, that they are being watched, spied upon, and the like. Jealousy with vindictiveness is very often met with. Insane interpretations of morbid cutaneous and muscular feelings also occur; thus patients will complain of being galvanized, mesmerized, or "interfered with." The prognosis in those suffering from delusional insanity due to alcohol is not very hopeful, and in any case they should be kept under observation for many months before they are discharged as *well*. In some cases the morbid sensations persist and the morbid interpretations remain, and yet they cease to be active agents upon the patients' will, so that we meet with chronic cases of alcoholism, with chronic delusions, who are able to be at large without danger to themselves or friends. Chronic alcoholism probably tends in some cases to the production of general paralysis of the insane; it is much more certain that it tends to senile dementia, and in the cases of senile dementia connected with alcohol there is a great tendency to apoplexy, hemiplegia, and paralytic weakness of mind.

It is legal to send a patient suffering from alcoholism into an asylum if he cannot be controlled, or if he be dangerous to himself or others; yet we ought to be extremely cautious in signing certificates for persons suffering from chronic alcoholism with symptoms of insanity, for though seclusion will restore them to their former mental state, that is not a healthy or normal one, and the prospect of legal proceedings is in such cases considerable.

MORAL INSANITY.—It may seem unphilosophical to describe a form of insanity as belonging to the moral apart from the intellectual side of man, but the term will perhaps appear clearer to some readers if moral insanity be defined as a perversion of the nervous system by which the man or woman is rendered "anti-social." To give an idea of what is meant I would say there are two distinct conditions under which moral insanity may exist: first, the children of insane or highly neurotic parents seem in many cases to come into the world unable to grow up into full intellectual and moral capacity; secondly, there are certain diseases of the brain or disorders of the mental functions which, having upset the nervous balance, leave a fresh moral standard far below what was the normal and healthy standard of the individual. Thus there are some patients who are unable to develop into their moral and social places, and there are others who having filled their proper positions are, as the result of disease reduced to a lower moral rank.

First as to the young patients, the offspring of parents who have been drunken, epileptic, or insane, not uncommonly are morally rather than intellectually insane. They are perverse and incapable of being trained into their duties to society. Thus they are frequently precocious and sexually vicious, they are cruel, mischievous, untruthful, thievish, and at times pyromaniacs. At the same time these persons may have one or two special gifts.

Thus, they may be extremely rapid calculators, they may be able to reproduce musical combinations, or they may have wonderful memories for isolated facts. As a rule such patients require isolation. As they grow older they become more dangerous from their sexual passions and from their destructive tendencies. They often pass into eccentric, weak-minded drudges in asylums.

Beside these we have to consider those who are morally weak as the result of acute attacks of insanity. After acute mania some young patients will be left kleptomaniacs, others will have lost their self-control and become amorous and impulsive, others cease to be truthful and in one way or other show marked anti-social qualities. In these cases again there is little or no prospect of recovery,—they are like people who have been scarred by the smallpox, and nothing will ever efface the marks.

Not only after mania but after other forms of mental disorder, moral defects may become manifest; and similar changes may follow fevers. Temporary, and in a few cases permanent, moral changes have been observed to follow an attack of typhoid or rheumatic fever.

Moral perversion may occur as an early symptom of general paralysis of the insane; it is common with alcoholism and is frequently seen in the earlier stages of senile dementia. In some cases the faults or vices of old men are really the result of early degeneration of the brain.

Moral insanity is scarcely capable of medical treatment. In the case of many young and middle-aged persons the best and indeed the only course is to let the delinquent receive the due punishment of his faults. The discipline of the jail has undoubtedly done good in some cases. Such young persons will ruin themselves and their friends, and if steps are taken to send them to asylums, they often retaliate and cause endless legal trouble.

Syphilis may give rise to various forms of insanity. The moral effect of the consciousness of transgression may set up hypochondriasis with syphilophobia, and this may end in general paralysis of the insane. Syphilis may produce local brain lesions with various degrees of mental weakness; or epilepsy, which may likewise lead to the same result. Syphilis may give rise to arterial changes which may lead to dementia, or to paralysis ending in dementia. In a considerable number of cases of general paralysis of the insane there is a history of syphilis.

It is not uncommon to find patients who have suffered from syphilis and who have recovered from some secondary symptoms such as ptosis, external strabismus, or hemiplegia, pass into weak-mindedness or into conditions hard to be distinguished from general paralysis. In a certain number of weak-minded children and idiots, and in some elder patients, congenital syphilis has produced sensory or intellectual weakness. In any case of insanity in which syphilis has previously occurred, anti-syphilitic drugs should be used; but in most cases resembling general paralysis or with marked mental defect no good result will follow such treatment.

Gout may occur in neurotic families and may be of special importance. Before and after gouty attacks there are often marked mental changes in patients, and in some cases of so-called suppressed gout most profound and suicidal melancholia may occur only to pass off when active gout appears. Gout may seem to alternate with insanity as with asthma or eczema. Gouty degeneration of arteries is not uncommonly associated with signs of senile decay and dementia.

Lead-poisoning may produce maniacal excitement or it may end in dementia. The same poison may produce all the symptoms met with in general paralysis, and may almost certainly give origin to that disease. It also leads to epilepsy and its results.

After *fevers* it is not uncommon to meet with more or less mental weakness, which does not depend much upon the severity of the fever. This is not uncommon after typhoid fever, and may range from simple loss of memory to loss of control and mania, with emotional disturbance. In persons of highly nervous temperament, the delirium of any fever may set up mental disorder, which may appear as acute delirious mania or, more commonly, simple acute mania.

Phthisis is related to insanity in many ways. The insane are more than usually liable to phthisis. Insanity in one parent and phthisis in the other gives rise to a very unstable type of mind. Many patients who recover from severe attacks of mania or melancholia die a year or two afterwards of phthisis.

A form of insanity called phthisical insanity has been described. The patients are suspicious, and refuse their food because they believe it to be poisoned; they often have hallucinations of taste and smell, and of hearing; they rapidly waste, without having any cough or expectoration; the temperature is more or less like that of phthisis, and dulness is found if sought for under the clavicles. These patients require forcible feeding with stimulants and cod-liver oil. Hæmoptysis with temporary relief to the mental symptoms is not uncommon.

Spasmodic asthma often occurs in neurotic families, and it may occur in persons who have had attacks of insanity. In some cases the spasmodic attacks disappear and the patient becomes melancholic, only being restored to his right mind when the asthma returns.

Heart disease may act as a cause of insanity; mitral incompetence being most frequently associated with feelings of distress, anxiety and melancholia, while aortic lesions, and at times mitral contraction, have been met with associated with mania and great excitement.

With *renal disease*, the tendency is toward mental depression.

Diabetes is not uncommon in neurotic families nor is it very rare in insane persons; but generally with the onset of insanity the diabetes disappears.

Epilepsy and insanity are allied by origin. Epilepsy occurs in neurotic subjects, and epilepsy in infants causes idiocy or imbecility. Frequent recurrence of epileptic fits, whether in the major or the minor form, leads to weak-mindedness. Hallucinations of the senses may precede the fits; and after them periods of unconsciousness may follow, during which highly complex acts may be performed, the patient being in a state allied to somnambulism (cf. *supra*, p. 820). Epileptic fits may be followed by fury of the most violent kind, during which brutal and bloody deeds may be done (p. 754).

Convulsions may probably be replaced by mental disorder—"l'épilepsie larvée." In this the patient after a sudden arrest of the work in hand goes through more or less highly organised acts unconsciously; these acts in each recurrence are usually alike, and just as each epileptic fit is a repetition of its predecessors, so is each mental attack like its forerunners.

Jacksonian epilepsy is not so frequently followed by mental disorder as is the idiopathic epilepsy which occurs in neurotic subjects.

Paralytic insanity.—After apoplexy there is a great tendency to weak-mindedness. This is not always enough to deprive the patient completely of self-control, but it often leads to loss of memory and to a certain want of self-control manifested in erotic lust, in emotional weakness, and in a tendency to be easily influenced by others. It commonly passes from the slighter to the graver forms of dementia, especially if fits recur.

Tumours of the brain generally tend more or less certainly to loss of some of the functions of the mind.

In *locomotor ataxy* we frequently meet with a neurotic history, and in many cases ataxy is an early symptom of general paralysis. But besides, with locomotor ataxy there may be outbreaks of mania, generally of a suspicious, jealous nature; or the symptoms of the disease may be falsely interpreted by the patient: he may say that his legs are electrified and his sexual power removed by his enemies, or he may accuse people of twisting his bowels or destroying his sight.

If the symptoms are part of general paralysis, the prognosis is bad; but if due to locomotor ataxy, the prognosis is much better as far as length of life is concerned.

ADMINISTRATIVE CAPACITY.—One of the most important duties of the medical man is to be able to judge of the mental capacity of a patient who may have had some attack of insanity or other nervous disturbance. First, it must be remembered that some persons who have been insane recover completely, and may be able to transact business as well as ever; that the prospect of recovery and of mental capacity is lessened with the increasing number of attacks and with increasing years. Wills may be made by persons who are chronic lunatics, and yet the wills may be so reasonable that judge and jury will support them. It is important to remember that to upset testamentary capacity it must be shown that the patient had *no* knowledge of what he was doing, or that he had mistaken ideas about what he was effecting, or that his judgment was biassed by delusions of one kind or other, or that, being weak, he was unduly influenced. Defective memory is a thing to be specially noted. If it can be shown that a man when making a will did not remember the number of his children or whether they were living or dead, there would be ground for disputing that will. If, on the other hand, it can be shown that he was emotional and easily led by others, even though the memory was not very defective, yet it might be shown that he was too readily influenced at the time he made his will by those who were near him. If it can be shown that he had a causeless antipathy —the result of a delusion—to his direct heirs, it is only necessary to prove the nature of those delusions and their existence about the time the will was made. Apoplexy probably gives rise to weak-mindedness more frequently than any other disease of the brain, and the mental weakness produced by apoplexy certainly gives rise to the greatest number of trials in the probate courts. A man may have one or more attacks of apoplexy and yet may remain of "disposing" mind; but it is well to remember that after fits of apoplexy memory is very frequently affected, emotional disturbance is readily raised, and other signs of weakness and change of character are generally present.

There may be complete testamentary capacity without capacity for

speech, in fact aphasia and weak-mindedness are not necessarily related; but it may be found difficult to get a jury to understand that a person unable to give the name to the simplest object may yet be able to dispose of his property. This difficulty will be greatly increased if agraphia also is present.

THE CONFINEMENT AND RESTRAINT OF INSANE PATIENTS.—One of the most difficult points for a medical man to decide is as to whether a patient should or should not be sent to an asylum; and having decided on sending a patient away from home, the next difficulty arises as to where the patient should be placed.

For the reception of persons of insane mind there are houses receiving one patient only; this is called *single care*. These houses are without any license, and no certificate is necessary for many patients so placed. But it must be understood that, even though a patient be placed in "single care," it will be necessary to have the ordinary medical and other certificates in due and proper form, if he is so insane as to need his actions to be controlled.

Beside houses which receive only one patient there are others, *licensed houses*, for the reception of two or more, and these merge insensibly into the private *asylums* which are to be found in most counties of England. Next there are registered *hospitals* into which patients are received at various rates, the principle of the hospital being that while some patients are received free or for small sums, others pay more than their actual cost, and thus help to pay for the treatment of the poorer ones. There are also the large borough and county *asylums*, expressly intended for the reception of those who are not sufficiently wealthy to pay for private asylums, and who, from one cause or another, are ineligible for hospitals. Lastly, a certain number of quiet harmless lunatics are confined in the *workhouses* and workhouse infirmaries.

If a person of unsound mind is to be removed to the borough or county asylum, it is necessary that notice should be given to the relieving officer of the parish, who in due course reports to the medical officer of the same parish or some other medical man; and the patient being brought before a magistrate, and the medical man having certified that he is of unsound mind and unfit to be at large, the magistrate signs an order for the transference of the patient to the county asylum. If a person of unsound mind be found wandering about the streets, he may be given in charge of the police, and, being taken before the magistrate, a doctor being called by the magistrate, may give evidence which satisfies the justice of the peace that the person is of unsound mind and requires detention; and then he may, on the magistrate's order and the medical man's certificate, be sent to the asylum. There are special provisions for cases in which no magistrate is available, or when the magistrates decline to act; thus two medical men signing a certificate can practically enforce the removal of a patient to a county asylum. No patient can be kept or retained in a medical man's house for profit except under full legal certificates, unless he be a near relative, it being manifest that no one's liberty of action must be restrained to the advantage of another without legal authority.

There must now be *two medical certificates* on separate sheets of paper (one of these being by the regular medical attendant), a *statement* of particulars in the form given, and a *petition* to a magistrate or judge specially named for the duty, which must be accompanied by the other papers, viz. the medical certificates and the statement; the magistrate then satisfies

himself of the correctness of forms and the necessity for the detention of the patient, and signs *the order* for his reception into any hospital or private asylum or private house. A patient may be sent to any asylum on one medical certificate and the statement, provided that at once steps are taken to get the petition and other certificates after his admission (see *Urgency Form*). Patients may place themselves in asylums voluntarily with the consent of the Commissioners.

FORMS 1 AND 2.

Form of Urgency Order for the Reception of a Private Patient.

I, the undersigned, being a Person Twenty-one years of age, hereby authorise you to receive as a Patient into your (a) _____

(a) House *or* hospital, *or* asylum, *or* as a single patient.

(b) _____

(b) Name of patient.

(c) Lunatic, *or* an idiot, *or* a person of unsound mind.

as a (c) _____ whom I last saw at _____

(d) Some day within two days before the date of the order.

on the (d) _____ day of _____ 189 .

I am not related to or connected with the Person signing the Certificate which accompanies this Order in any of the ways mentioned in the Margin (e). Subjoined [*or* annexed] hereto is a Statement of Particulars relating to the said _____ .

(e) Husband, wife, father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law, partner, *or* assistant.

(Signed)

[*If not the husband or wife, or a relative of the patient, the person signing to state as briefly as possible*:—1. Why the order is not signed by the husband *or* wife, *or* a relative of the patient. 2. His *or* her connection with the patient, and the circumstances under which he *or* she signs.]

Name and Christian Name at length _____

Rank, Profession, or Occupation (if any) . . . . . _____

Full Postal Address . . . . _____

How related to or connected with the Patient . . . . _____

Dated this _____ day of _____ 189 .

(f) Proprietor *or* superintendent of —— house, *or* hospital, *or* asylum (describing house *or* hospital *or* asylum by situation and name).

To (f) _____

_____

[In cases of urgency a lunatic person may be placed under care and treatment upon an Urgency Order made (if possible) by the husband or wife, or by a relative (*i.e.* a lineal ancestor or lineal descendant, or a lineal descendant of an ancestor not more remote than great-grandfather or great-grandmother), accompanied by a Statement of Particulars and one Medical Certificate. If a Petition for an Order for Reception of the patient has been already presented to a Judge, Magistrate, or Justice, a copy of the Urgency Order must be sent forthwith to such Judge, Magistrate, or Justice. An Urgency Order will remain in force for seven days from its date, or if a Petition for a Reception Order is pending, then until such Petition is finally disposed of.

LUNACY 4 and 2.

(52 and 53 Vict. c. 41.)

The Urgency Order, Statement of Particulars, and Medical Certificate must be sent to the Superintendent or Proprietor of the Asylum, Hospital, or House where the patient is to be received.]

This, accompanied by the *Statement*, will suffice to remove a person of unsound mind to an asylum, when the further certificates can be obtained.

STATEMENT OF PARTICULARS.

Statement of particulars referred to in the annexed petition.

The following is a statement of particulars relating to the said

(*Name of Patient in full*) _____

N.B.—*If any Particulars are not known the Fact is to be so stated.*

Name of Patient, with Christian Name at length	_____
Sex and Age	_____
Married, Single, or Widowed	_____
Rank, Profession, or previous occupation (if any)	_____
Religious Persuasion	_____
Residence at or immediately previous to date hereof	_____
Whether First Attack	_____
Age on First Attack	_____
Give the dates. WHEN and WHERE previously under care and treatment as a lunatic, idiot, or person of unsound mind	_____
Duration of existing Attack	_____
Supposed cause	_____
Whether subject to Epilepsy	_____
Whether Suicidal	_____
Whether dangerous to others, and in what way	_____
Whether any near Relative has been afflicted with insanity	_____
Names, Christian Names, and full Postal Addresses of one or more Relatives of the Patient	_____
Name of the Person to whom notice of Death to be sent, and full Postal Address if not already given	_____
Name and full Postal Address of the usual Medical Attendant of the Patient	_____

How many previous attacks? _____

Has the Patient been of Sober Habits? _____

Number of Children? _____

Age of youngest? _____

Degree of Education? _____

Signed, _____

When the Petitioner or Person signing an urgency order, is not the Person who signs the statement, add the following particulars concerning the Person who signs the statement.

Name, with Christian Name at length	_____
Rank, Profession, or occupation (if any)	_____
How related to or otherwise connected with the Patient	_____

N.B.—*The Patient must be received into the Hospital before the expiration of Seven clear days from the date of the Judge, Magistrate, or Justice's Order.*

ORDER.

Order for reception of Patient, to be made by the Judge of County Courts, Stipendiary Magistrate, or Justice appointed under the Lunacy Act, 1890.

I, the undersigned (*Name*)_____
being the Judge of the County Court of _____
or the Stipendiary Magistrate for_____
or a Justice for _____specially appointed under the Lunacy Act, 1890, upon the petition of (*Name of Petitioner*)_____
of (*Address and Description*)_____
in the matter of (*Name of Patient*)_____
a person of unsound mind, accompanied by the Medical Certificates of
(A) Names of Medical Practitioners signing Certificates. (A)_____and (A)_____
hereto annexed, and upon the undertaking of the said (*Name of Petitioner*)_____to visit the said (*Name of Patient*)_____personally or by someone specially appointed by the said (*Name of Petitioner*)_____
once at least in every Six Months while under care and treatment under this Order, hereby authorise you to receive the said (*Name of Patient*) _____as a Patient into your Hospital.
And I declare that I have [or have not] personally seen the said (*Name of Patient*)_____before making this Order.

Dated_____
Signed,_____ *Judge of the County Court of*_____
_____ or *Stipendiary Magistrate,*
To the _____ or *a Justice for*____ *appointed*
Resident Physician, *under the said Act.*
_____,

N.B.—*Under all circumstances, if possible, the "PETITION" and "STATEMENT" below to be filled up by the Patient's Relatives. If no Relatives, by the nearest Friend.*

PETITION FOR AN ORDER FOR RECEPTION OF A PRIVATE PATIENT.

In the matter of (A)_____ (A) Name of Patient in full.
a person alleged to be of unsound mind.
To His Honour the Judge of the County Court of _____
or To_____Stipendiary Magistrate for_____
or To_____a Justice of the Peace for_____
The Petition of (B)_____ (B) Name of Petitioner in full.
(1) Full Postal Address, and Rank, Profession, or Occupation. of (1)_____
in the County of_____
1.—I am (2) _____years of age.
(2) At least twenty-one.
2.—I desire to obtain an Order for the reception of (*Name of Patient in full*)_____as a person of unsound mind in_____

3.—I last saw the said (*Name of Patient in full*)_____ at_____ on the (3)_____ day of_____ 189 .

(3) Some day before the date of the presentation of the Petition.

(4) Here state the connection or relationship with the Patient.

4.—I am the (4)_____ of the said (*Name of Patient in full*)_____

If the Petitioner is not connected with or related to the patient, state as follows:—

I am not related to or connected with the said (*Name of Patient in full*)_____

The reasons why this Petition is not presented by a relation or connection are as follows:—

_____

_____

_____

The circumstances under which this Petition is presented by me are as follows:_____

_____

5.—I am not related to or connected with either of the persons signing the Certificates which accompany this Petition as—

(*where the petitioner is a man*) husband, father, father-in-law, son, son-in-law, brother, brother-in-law, partner, or assistant;

(*or where the petitioner is a woman*) wife, mother, mother-in-law, daughter, daughter-in-law, sister, sister-in-law, partner, or assistant.

6.—I undertake to visit the said (*Name of Patient in full*)_____ personally, or by some one specially appointed by me, at least once in every Six Months while under care and treatment under the Order to be made on this Petition.

7.—A Statement of Particulars relating to the said (*Name of Patient in full*)_____ accompanies this Petition.

If it is the fact, add—

8.—The said (*Name of Patient in full*)_____ has been received in the_____ under an Urgency Order, dated the (5)_____

(5) Here insert date of Urgency Order, if any.

The Petitioner therefore prays that an Order may be made in accordance with the foregoing Statement.

*Full Christian and Surname of Petitioner*_____

Date_____

N.B.—If neither of the Practitioners signing the Medical Certificates be the usual Medical Attendant of the Patient, the reason of this must be stated in writing to the Judge, Magistrate, or Justice by the Petitioner.

N.B.—Medical Certificates of Patient's Examination, and the Signatures, are required by the above Statute to be dated *not more than Seven clear Days* previously to the date of the presentation of the Petition.

Medical Practitioners signing the Certificates must not be in Partnership, nor one the Assistant of the other; nor must they be related to one another, as father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law, partner, or assistant; nor must they themselves sign the Petition or Urgency Order, nor must they be related to the Petitioner in any of the ways specified in the Petition.

One of the Certificates shall, *whenever practicable*, be under the hand of the usual Medical Attendant, if any (being a medical practitioner), of the alleged lunatic. They must use the terms specified in the Statute. (*See* marginal notes.)

By Order of the Commissioners in Lunacy.

1.—It is absolutely necessary that the Medical Men should write their Certificates legibly, so as to afford the opportunity of an exact copy being made.

2.—"All alterations in the original Certificates, *unless by the certifying Medical Men*, invalidate them; and *the initials of the latter* must be placed to every change or addition made."

CERTIFICATE OF MEDICAL PRACTITIONER.—Schedule 2, Form 8.

In the matter of (*Name of Patient*)_____

(1) of_____ in the_____ (2) of_____

(3)_____ an alleged lunatic.

I, the undersigned (*Name of Practitioner*)_____

do hereby certify as follows:—

(1) *Insert* RESIDENCE *of Patient.*
(2) *County, City, or Borough, as the case may be.*
(3) *Insert* PROFESSION *or* OCCUPATION *(if any).*

1. I am a person registered under the Medical Act, 1858, and I am in the actual practice of the medical profession.

2. On the_____ day of_____, 189 , at (4)_____ in the (5)_____ of_____, separately from any other practitioner, I personally examined the said (*Name of Patient*)_____ and came to the conclusion that he is a person of unsound mind, and a proper person to be taken charge of and detained under care and treatment.

(4) *Insert* PLACE *of examination, giving the* NAME *of the street, with* NUMBER *or* NAME *of the house, or should there be no number, the* CHRISTIAN *and* SURNAME *of Occupier.*
(5) *County, City, or Borough, as the case may be.*

3. I formed this conclusion on the following grounds, viz.:—

(*a*) Facts indicating Insanity observed by myself at the time of examination (6), viz.:—

_____

_____

_____

_____

_____

(6) *If the same or other* FACTS *were observed previous to the time of examination, the certifier is at liberty to subjoin them in a separate paragraph.*

(*b*) Facts communicated by others, viz.:—(7) (State the NAME IN FULL of the person giving the information, with the address and description.)

_____

_____

_____

(7) *The* NAMES *and* CHRISTIAN NAMES *(if known) of informants to be given, with their* ADDRESSES *and* DESCRIPTIONS.

4. The said (*Name of Patient*)_____ appeared to me to be (*or* not to be) in a fit condition of bodily health to be removed to_____.

5. I give this Certificate, having first read the Section of the Act of Parliament printed below.

Dated_____189 . Signed,_____

Full Postal Address_____

Extract from Section 317 of the Lunacy Act, 1890.

"Any person who makes a wilful misstatement of any material fact in any Medical or other Certificate, or in any Statement or Report of bodily or mental condition under this Act, shall be guilty of a misdemeanour."

N.B.—By Section 28, "Every Medical Certificate made under and for the purposes of this Act, shall be evidence of the facts therein appearing, and of the judgment therein stated to have been formed by the Certifying Medical Practitioners on such facts, as if the matters therein appearing *had been verified on oath.*"

In signing certificates it is well to remember that the most important points are the facts observed by one's self, and these facts bear much greater weight if they can be shown in brief to be associated with a development of nervous disturbance. Thus sleeplessness, restlessness, refusal to take food, threats of suicide, and a tendency to wander from home, would quite suffice without the additional fact which may have been communicated by A—B—, the nurse, that the patient tried to throw herself from the window, and said she was possessed by the devil. Whenever the certificate is strengthened by the addition of facts communicated by others, the name in full of such person must not be omitted. Certificates in England only hold while the patient is in England, and are broken by escape to the Continent, to Scotland, or to Ireland; but if a patient escape from an asylum, and remain in England, the same certificates hold good for fourteen days, during which period he may be recaptured.

Licenses for the reception of more than one lunatic into a private house are granted in the neighbourhood of London by the Commissioners, and in other parts of England by the magistrates in quarter session. Patients under certificates are in direct relationship to the Commissioners, who require a complete copy of the certificates upon which they have been received, as well as a statement within a week of the reception of the patient as to his bodily and mental condition. They also require that a medical visitation book of a prescribed form should be kept and entries made from time to time of the visits paid by some independent medical man to the patient. The frequency of these visits will be decided upon by the Commissioners themselves. All letters written by the patients to the Commissioners in Lunacy must be forwarded unopened; all other letters of patients must either be forwarded to their address, or, being initialled, must be kept for the inspection of the Commissioners on their visit, or of some other constituted authority.

Patients can be transferred from a private asylum into a private house, or from a hospital to a private house, or the reverse, permission having been sought and granted by the Commissioners in Lunacy. When a patient is sent from one establishment to another, the order of the Commissioners, with a copy of the original order, statement, and certificates, must be forwarded with him. Patients cannot be transferred from a private asylum or private care to a county or borough asylum, or the reverse.

Though a patient is of unsound mind and under certificates, his friends and relatives have no right to transact any business in his name unless a power of attorney have been previously granted by the patient; so that it is advisable in all cases where recurrences of insanity occur, or where the patient is sufficiently sensible to understand the threatening illness, to get a power of attorney before he loses self-control. This may save great trouble and expense afterwards.

If it be necessary to transact important business connected with a person of unsound mind, the only legal method is to place the affairs in Chancery, there being a special department of the Court for the purpose. Of Chancery cases there are two groups. The first includes only small properties, that is, under £2000 capital or £100 a year income, and for these the procedure is simple and rapid. It is as follows:—Two affidavits have to be drawn up in a form to be presently referred to, stating in detail the facts of the patient's insanity and his inability to transact business. These affidavits having been laid before the judge in Chancery, with certain other facts as to his affairs and his relations, by the family solicitor, an order may be given by

the Court of Chancery for the administration of this small estate; and two people, respectively called the committee of the person and the committee of the estate, will be appointed.

The following is an epitome of the second and longer process, which is costly and difficult to set aside, and therefore not to be undertaken without grave consideration: for the Court of Chancery will almost certainly realise the property of the patient, and a man's property may be realised at a very great loss while he is of unsound mind. It is therefore best to avoid placing any acute and presumably curable case under the Court of Chancery. When, however, it is necessary to put the affairs of a patient under the care of the Court, the family solicitor generally asks the medical man in charge of the case, or the asylum superintendent, to make a draft affidavit, and he also probably gets some independent medical expert in lunacy to draw up a second affidavit. These affidavits differ entirely from the ordinary medical certificate, there being no restriction as to consultation or separate and independent examination of the patient, and it is best to fill the affidavit up in the following way:—First, state your name, your medical qualifications, your qualifications to judge of the sanity of the patient, more especially your opportunities of having seen him; then give your judgment as to the form of disease from which he is suffering; give in the next two or three paragraphs a statement as to his general behaviour, his intellectual capacity, the delusions from which he suffers, the tendencies to suicide or destructiveness which he may have, and finally, give your judgment as to the improbability of his speedy recovery; or, if you believe him to be fatally or incurably insane, give your judgment that he is so and is unfit to take care, first of himself, second of his affairs. These two affidavits having been presented, in due course notice will be given to the patient that an inquiry or so-called inquisition or commission in lunacy will be held. The patient may then instruct counsel himself or he may demand to be tried by jury and to be defended by counsel and to be present himself. Or he may demand that he may be tried before a Master in Lunacy before a special jury. In one case at least a judge of the Court of Chancery took the position of the Master in an inquiry. In some cases where the patient is extremely suspicious, there may be considerable difficulty in persuading a jury that he is of unsound mind; and in nearly all cases where there is likely to be opposition of this kind, where in fact the patient is still so reasonable as to be able to defend himself, it is better to take any other means that are possible. If the patient be found insane by inquisition, the Court of Chancery in due time appoints one person to look after the affairs of the patient, who is called the committee of the estate, and another to look after the person of the lunatic, who is called the committee of the person.*

A Chancery lunatic remains so though he travel or though he escape to the Continent; he can be transferred from one asylum to another or from an asylum to a private house without any order of transfer from the Commissioners, all that is required being a signed order from the Court of Chancery and the committee of the person.

If a patient recover after he has been made a Chancery patient, it is necessary, before he can have his freedom and control of his property, that the whole of the proceedings should be set aside by a similar process. Two

* In future, after an inquisition, it may be decided that a patient is fit to be at large, but is not fit to manage his affairs.

medical men (preferably those two who have signed the first affidavits for the reception of the patient) should draw up two affidavits, this time stating their belief and the grounds for their belief that the patient is restored to his mind and is now fit to manage himself and his affairs.

The county and borough asylums are intended only for pauper lunatics, but really a large number of patients have to pass from private care to these county asylums because their friends are unable to pay the charges, which range from one guinea per week to almost any sum. Therefore chronic patients have to be provided for by the parish, the friends being called upon to pay for their maintenance in proportion to their ability and to the cost in the asylum. A certain number of county asylums at present receive paying private patients for sums under one pound a week, and it is arranged by recent lunacy legislation that all county and borough asylums may now receive such paying patients. Patients may, under certain conditions, place themselves in registered hospitals or private asylums as voluntary boarders.

IDIOCY AND IMBECILITY.—These two are but grades of the same want of intellectual development, "idiocy" ranging from almost complete absence of intelligence, whereas "imbecility" may approach very nearly to mere eccentricity and oddness.

Idiocy depends in the majority of cases upon structural defect in the brain. A certain number of idiots are the offspring of insane parents; certain others owe their condition to injuries received in early childhood; while certain forms of physical and mental disorder occurring in weakly or nervous children may arrest mental development at a higher or lower point, leaving as the result an idiot or an imbecile. The brain is so delicately constructed that a slight damage done to it during its period of growth interferes with its permanent and normal development.

In looking at the pathology of idiocy, we have to recognise the fact that some children are born with defective brains, so that in some there is very little more than the ganglia at the base: a brain which suffices for the nutrition of the body, but is unequal to the development of the mental functions. In other cases important parts of the brain are absent, its commissural fibres or the radiating fibres being deficient; and in the same category are to be considered the changes produced in the brain by defect of one or more of the senses. The child who comes into the world without sight and without hearing stands a very great chance of being an idiot through the consequent starvation of the brain. In idiots with defective brains we meet with every shade, from the anencephalous idiot, whose brain weighs but a few ounces, to the hydrocephalic idiot, whose brain is deficient though his head is enormous. In certain idiots there is rather a general deficiency than non-development of any one part of brain. All parts are there, but they are not sufficient. The convolutions are found to be broad and few, the grey matter is either not fully developed or is defective in quantity, or there is an enormous overgrowth of the connective tissue, so that in some idiots there are not only enormous heads but weighty brains. Beside all these, there are some children who in aspect are healthy, but who are idiotic or imbecile in consequence of some ill-understood defect in the power of the brain to work. If one may use the term, there are some "functional" or "neurotic idiots" who have fairly sized brains with fairly developed convolutions, and yet with inability to make

use of the brain they have. Some of these belong to the nervous stock generally, and others seem to have had their capacity for intellectual development arrested by some acute disorder of infancy. It is a question whether intermarriage will produce such cases.

The case of the so-called cretinous idiots will be considered in a separate chapter (*infra*, p. 859), and it is therefore unnecessary to describe the theories of the development of idiocy from arrest of growth in the base of the skull.

Most idiots are short-lived, many are defective not only in mind but in body. Many of them have malformations of limbs or a peculiar stunted aspect, with awkward, ungainly walk. Most of them are restless and irritable. Although generally slow of comprehension and dull as far as education is concerned, yet there are a certain number of so-called *idiots-savants* in whom some special faculty of the mind seems to grow at the expense of all the rest. Just as one may meet with a morally imbecile or morally insane person with certain intellectual activities fully developed, so in the learned idiot one may meet musical, mathematical, or mnemonic ability of a very high order.

Idiots have been divided according to their *capacity for speech*, the very lowest being unable to speak, the next group being able to articulate or make use of simple words, a higher grade being able to comprehend short sentences; while the lowest imbecile, somewhat in advance of the highest idiot, would have still greater power, and a higher imbecile would be able to learn and repeat many things, slowly perhaps, but yet surely. Other varieties have been based on the *facial peculiarities*; some idiots having extraordinarily receding foreheads have been compared to fish, and others have the aspect of a Chinaman, while some seem to resemble other races or even some of the lower animals; and when one takes up a book in which the physiognomy of man is compared with the physiognomy of brutes, one is struck with the examples which have evidently been taken in many cases from idiots and imbeciles.*

For our convenience here it will be well to take the groups of idiots as arranged by Dr Ireland. He makes ten groups—(1) Genetous idiocy; (2) microcephalic idiocy; (3) eclampsic idiocy; (4) epileptic idiocy; (5) hydrocephalic idiocy; (6) paralytic idiocy; (7) cretinism; (8) traumatic idiocy; (9) inflammatory idiocy; (10) idiocy of deprivation.

(*a*) *Congenital idiocy.*—The first class contains those cases which start in fœtal life; there is general weakness as a rule, mental and bodily, and the prospect is altogether unfavourable. The same holds good to a great extent with the microcephalic idiot, whose head, according to Dr Ireland, should measure less than seventeen inches; there being a distinct and absolute want of brain the prospect here is bad. In the third class of eclampsic idiots are placed those children who have ceased to develop in consequence of convulsions which have occurred during early childhood. Probably, however, a very large proportion of these come of a nervous stock, and are thus already unstable when some such exciting cause as teething suffices to start convulsions which so modify the brain in its development that it never assumes full vigour. Some of these cases may be trained to simple mechanical work.

* 'De Humana Physiognomia,' Joannis Baptistæ Portæ, Neapolitani, libri iv, ed. altera, Francofurti, 1618. Note particularly the figures of human faces resembling the ox and the pig (pp. 82 and 90). This principle of comparison was derived from Aristotle.

(*b*) The next is a nearly related group containing the *epileptics*. Fully one third of the idiots in the Metropolitan Idiot Asylum at Darenth are children whose idiocy is at all events accompanied by epilepsy. As a rule the epilepsy weakens both mind and body, and in many cases seems to be associated with great moral perversity, the epileptic idiot being hard to manage, and in very few cases suitable for home treatment. A certain number of epileptic idiots outgrow their epilepsy, and though mentally defective may be developed to a moderate intellectual standard. Such patients should be tried with bromide of potassium, but cautiously; food and fresh air with discipline and association with healthier children are the best forms of treatment.

(*c*) With *hydrocephalus*, idiocy is not uncommon, and if the hydrocephalus be great it is almost certain that there will be intellectual defect; but the majority of cases with considerable hydrocephalic and mental weakness die, so that the number seen in a large idiot asylum is not great. A good many of these cases by careful treatment improve, and from idiots may be trained into the class of useful imbeciles, to a certain extent trustworthy, but having organic deficiency of brain which prevents them becoming more than useful drudges. Beside the hydrocephalic idiot, we have to recognise a group of *paralytic* idiots, in whom some local defect of brain occurring in early life has given rise to paralysis of one or more of the extremities, and has left a mental as well as a physical scar upon the brain. In these cases again we can expect but little improvement from treatment; the weakened limbs may be galvanised, friction and passive movements with baths may be used, and some power may be gained in them, just as some slight power may be gained in the faculties.

(*d*) The next group contains the *traumatic* idiot. A number of idiots are found to be males that are firstborn, and a certain number of others are found to have had injuries to their head during parturition, and it is therefore well to remember that the delicate brain, too severely pressed upon by a narrow pelvis, or interfered with unnecessarily or severely by instruments, may be so permanently injured that idiocy results. Injuries before birth, at birth, or soon after birth occurring to children, will frequently prevent development; and there is no doubt that falls and similar injuries in early infant life give rise to a considerable proportion of idiots. Many of these are rather imbecile than idiotic; for the brain, which ought under natural circumstances to have fully developed, has been so interfered with that it only develops partially, and with it the mental faculties also are impaired but not wanting.

(*e*) *Inflammatory* idiocy is very closely allied to the last group, but under this head have been placed the idiots with hypertrophy of the brain. Here there is excess of connective tissue which interferes with the proper development, or at all events the proper function of the grey matter. Beside these, there are also the cases in which some disease of the nose or ears has set up inflammatory changes about the base or sides of the brain and interfered with its general development in a more or less mechanical way. As might be expected, the grades of idiocy from this cause vary very considerably.

(*f*) The only other important group is that associated with *deprivation of the senses*. It is quite certain that among the lower orders a considerable amount of weak-mindedness was formerly, and to a certain extent is still, due to the want of special education for the deaf and dumb. Deaf and

dumb children if not specially educated will from the deprivation of external impressions be but partially developed, and in some cases this deficiency will be more marked than in others. In some, in fact, who would be weak-minded at the best, the additional restriction of their intellect by the want of communication with the outer world leaves them imbecile or idiotic.

The same thing is still more true of those who happen to have loss or impairment of more than one sense: to them the portals of knowledge are closed. Nevertheless if these patients are taken early and trained specially, a large amount of instruction may be given them, and great amelioration of their condition will occur.

Idiots, if in the lower grades, should almost always be sent away from home. If there are other children, the influence of the weak-minded child upon them is distinctly injurious.

CRETINISM,

ENDEMIC BRONCHOCELE, AND THEIR ALLIES

"Le vallée sans soleil donne le crétin."—BALZAC.

"Quis tumidum guttur miratur in Alpibus."—JUVENAL.

Cretinism as an endemic form of idiocy—Its anatomy—Endemic bronchocele—Sporadic cretinism in England—Relation of goitre to cretinism—The cretinoid condition in adults (myxœdema)—The condition which follows removal of a bronchocele (cachexia strumipriva) in man—and the effects of removal of the thyroid in animals—Acromegaly.

THERE is a form of Idiocy, not described in the last chapter, which is constantly associated with remarkable peculiarities of cranial and bodily configuration. Its subjects have long been known as "cretins."* Their mental symptoms do not appear to differ from those of ordinary idiocy. As in that condition, there are all degrees of defect of intelligence, so that a Royal Commission on Cretinism which reported to the Sardinian Government in 1848, divided those affected into three classes, for which they proposed the names of "crétins," "semi-crétins," and "crétineux."

Endemic cretinism.—The most marked form of this curious disease occurs endemically in the Alps, and here cretins are well known to differ from ordinary idiots. They are seldom more than four feet and a half in height, and often below three feet. They have large brachycephalic heads. The features are broad and ugly; the eyes are wide apart; the nose is very flat at the root, and spreads out towards the alæ; the lips are thick; the mouth large, wide open, and allows the saliva to escape. The forehead and cheeks are wrinkled and the skin is coarse and rough. The hair comes low on the forehead and is thick and bristly. The chest is narrow, the belly large, and the limbs crooked. The hands are clumsy and broad, with short fingers. Cretins always look old, and one of twenty years is like one of forty.

The admirable investigations of Virchow have shown that all the peculiarities in the configuration of the cretin's skull and face are the result of premature ossification of the basilar process. In a cretinous fœtus he found complete synostosis of the basi-occipital and basi-sphenoid bones, with a continuous layer of diploë from one to the other, so that no trace of the original separation could be discovered. The basi- and pre-sphenoid bones were also fused together; but this is less important, because their union usually takes place soon after birth, whereas the basilar process and the sphenoid bone should remain separate until at

* Endemic cretinism is an exotic disease, and we therefore have no English word for it. The word is adopted from Switzerland by the French (as *crétin*) and by the Germans (as *Kreidling*). Its origin is uncertain: some would derive it from *chrétien* (in the sense of "innocent"); others from a Romansch word, *cretira*; and others from *creta* in allusion either to the chalky complexions of those who suffer from the disease (Littré) or to the calcareous formation of the districts where it prevails. The German name points decidedly to some connection with *Kreide* (creta, chalk).

least the fifteenth year. The premature completion of this "tribasilar synostosis" (as Virchow calls it) appears to be followed by the cessation of all further growth of the basis cranii in an antero-posterior direction. It also gives rise to a marked deformity in the shape of the internal base, the most constant features of which seem to be that the sella turcica is very narrow, and that the parts in front of the posterior clinoid processes form a greater angle than normal with the clivus or basilar process. Virchow found that the slope of the clivus was unduly steep in one fœtus. But at an early age a steep clivus is normal; and Nièpce has stated that in the cretins whose bodies he examined this part was horizontal; an observation which receives confirmation from a dissection of the author's, to be presently referred to. It would therefore seem that the premature closure of the spheno-occipital suture, although it arrests growth, does not prevent some of the changes in the base of the cranium which should take place during childhood.

Laterally, the cretinous skull doubtless expands abnormally, so as to compensate for the deficiency in its antero-posterior diameter, and this gives to the features of the cretin their peculiar breadth. The sunken appearance of the root of the nose is probably due to deficient forward growth of the vomer.

The configuration of the head and face in cretinism is, then, directly attributable to premature synostosis of the basal sutures. Moreover, some of the peculiarities in the build of the rest of the body may possibly be due to a similar interruption in the growth of other bones. Thus, as Griesinger suggested, the shortening of the fingers and other parts of the limbs may depend on too early union of the epiphyses.

Endemic bronchocele.—The most important fact in the ætiology of cretinism is the very close relation which it holds to *endemic goitre*. In this country as well as in others, goitre prevails in districts in which endemic cretinism is not met with. Indeed, the only place in England where the latter disease has been observed is the village of Chiselborough in Somersetshire; and Dr Petherton, who in 1847 pointed out its presence there, told the author that it has since almost died out. On the other hand, there is probably no district in the world where endemic cretinism occurs without goitre being still more common. Moreover, it has been noticed that when a family migrates into a place where both diseases are met with, goitre is the first to appear; it is only in the second or third generation that cretins present themselves. These facts seem to point to the conclusion that both diseases depend upon a common cause, but that goitre results where it has acted for a shorter time or with less intensity, cretinism when it has acted for a longer time or with greater intensity.

Goitre or bronchocele is not a mere hypertrophy of the thyroid; it is a disease of that organ. If we except cysts and malignant growths and the remarkable disease known by the name of exophthalmic goitre (which will be described under diseases of the circulation), bronchocele is in most cases an endemic disease. Hence its association with cretinism.

Many cretins are also goitrous; but others, instead of possessing an enormous bronchocele, have no thyroid at all, and these are some of the worst. According to the Report of the Sardinian Commission, goitre is absent in one third of the cases of cretinism.

Sporadic cretinism.—It is a remarkable circumstance that in England a form of cretinism sometimes occurs which also bears a close relation to the thyroid, but which is not peculiar to one part of the country. In papers

in the 'Medico-Chirurgical Transactions' (1871) and the 'Pathological Transactions' (1874) the author originally described it as Sporadic Cretinism. In this form of the disease there seems never to be a large goitre; but in the case of a boy who came to Guy's Hospital from Halden, in Kent, a sister had goitre. In most cases the thyroid, so far from being enlarged, is entirely absent, and no trace of it can be discovered on dissection. This fact was first pointed out by Mr Curling in 1850. In most respects sporadic cretinism is identical with the endemic form of the disease. Some of the figures which illustrate the above communications on the subject (figs. 1 and 3, pl. iii, 'Med.-Chir. Trans.,' vol. liv, and fig. 2, pl. xii, 'Path. Trans.,' vol. xxv) show how close is the resemblance between the two diseases in their general characters. Among the patients affected with sporadic cretinism, one, at the age of sixteen years and a half, was only two feet eight inches high; another, when twenty years old, was only two feet four inches high. They have the same broad, square hands and short fingers, the same dry hair, the same rough scurfy skin. Their heads are large and broad; their noses are flat at the root, so that the distance between the eyes is increased; and their mouths are large and gaping, with thick lips.

In the only case in which an autopsy was made (through the kindness of Dr Grabham, of Earlswood) the base of the skull was much altered in shape; the posterior clinoid processes were at a higher level than the anterior, and the sella turcica was very narrow. The clivus was horizontal, and its position seemed to be part of a general elevation of the occipital in relation with the other bones, for the cerebellar fossæ were exceedingly shallow. The patient was twenty-one years old at the time of death.

The intelligence of those who are affected with sporadic cretinism is very imperfect, and many of them present an extreme degree of idiocy, so as to be deprived of the power of speech. Their disposition is generally mild and tranquil. In this respect they seem to differ from the subjects of the endemic disease; for Griesinger says that the latter are unsociable and repugnant to each other. Probably, however, much depends on the conditions under which such patients are placed, and the amount of care and interest bestowed upon them.

Dr Langdon Down has seen twelve and Dr Beach five cases. A typical one is described by Dr F. N. Manning, of Sydney, in the 'Trans. Med. Congr. of Australia,' 1889 (p. 834); and Dr Stirling (*ibid.*, p. 840) records six cases from South Australia, with characteristic photographs.

One of the most curious features of sporadic cretinism is the presence on each side of the neck in the "posterior triangle," outside the sterno-mastoid muscle, of a soft, lobulated, and moveable lipoma. A characteristic case of sporadic cretinism is figured by Dr Fletcher Beach in the 'Trans. Intern. Med. Congr.,' 1881, vol. iii, p. 627, in which there was no thyroid and two large supra-clavicular masses of fat. In one of the author's patients these supra-clavicular swellings were much larger than hens' eggs. Their size seems to be influenced to some extent by the state of the general health. In another case of Dr Beach's ('Path. Trans.,' xxv) they were well marked when the patient first came under observation, but disappeared entirely before death, which was caused by exhaustion after protracted diarrhœa.

These fatty tumours are sometimes found slightly developed in adults who otherwise appear to be in good health. They were very large and well marked in a man weighing fifteen stone, who consulted the writer in August, 1887, for obesity and symptoms of alcoholic excess.

We might expect the sporadic cases to throw light upon the obscure pathology of endemic cretinism. They do not, so far as is known, depend on any external cause. Dr Langdon Down suggested that it is due to alcoholic intoxication on the part of one of the parents at the time of procreation; but this hypothesis has been disproved. Cretinism has more than once been seen in several children of the same parents. Thus, in a family of twelve, three children were cretins of an extreme type, while the rest were healthy, and one was an accomplished oarsman; they were all born in London, and their parents were in comfortable circumstances. Another of the author's cases of sporadic cretinism was that of a girl, who was stated by her relations to have been perfectly healthy until she was eight years old, when she fell ill with what was supposed to be a second attack of measles, and kept her bed for a fortnight. After her recovery her physical development underwent a remarkable change. Her features were previously well formed; they now acquired the cretinous configuration. Her hair, once black and abundant, became light coloured, dry, crisp, and scanty. She ceased to grow; at the age of sixteen years and three quarters she was only four feet one inch in height. Her hands and feet were not larger than those of a child six or seven years old. She had a fatty tumour, the size of a hen's egg, above each clavicle; and no trace of the thyroid could be discovered in front of the trachea. Sporadic cretinism seldom develops itself so late as this, and endemic cretinism is probably always congenital. This case is very difficult of explanation. But taken with the fact that the thyroid is congenitally absent in so many cretins, it certainly suggests that the febrile illness at the age of eight years led in some way to atrophy of that organ, and this to supervention of the cretinous state.

Pathological relation of goitre and cretinism.—Deficiency of the thyroid has probably never been recorded except in cretins, and goitre is a real deficiency though an apparent increase of thyroid tissue, which may be compared to pseudo-hypertrophy of muscles, the bulky lungs of emphysema, and the large white kidney. There is, therefore, a close relation between the two conditions; but what the relation is, and on what common cause they both depend, are questions most difficult to decide.

With respect to goitre, it usually occurs in the valleys of a limestone formation; but drinking water impregnated with lime-salts, magnesia or ferrous sulphide cannot be its true cause. Cases are common, not only in the Alps and Pyrenees, but in Southern Germany, in the Peak district, where it is known as Derbyshire neck, and in mountainous regions in China, in the valleys of the Himalayas and of the Andes, and in other parts of the world. It does not occur endemically in hot, flat, or low-lying districts, or near the sea-coast.

Cretinism, as above stated, is far more restricted in range; but beside the sporadic cases above described, it occurs endemically in the goitrous regions of Switzerland, the Engadine, and the Tyrol; also in the Pyrenees, in Cashmere, and in Peru. It is found among the inhabitants of valleys, because the valleys are the parts inhabited; among those who drink snow-water, because snow-water is drunk among the mountains—for there are no cretins in the Arctic regions—and it is found among a goitrous population, because some unknown cause produces both diseases.

Two views may be taken of the relation of cretinism and goitre. One was put forward by the author of this work in 1871, namely, that they are both antagonistic effects of the same cause, and that goitre is protective

against cretinism; that when the cause begins to act, or acts with but little intensity, the sole effect is goitre; but that if it acts with great intensity, or upon successive generations, it at length produces cretinism as well as goitre. The not infrequent absence of goitre in cretins—when not dependent upon congenital deficiency of the thyroid—might then be attributable to some local morbid change in that organ, by which it is prevented from enlarging as usual under the stimulus of the unknown morbid cause. It is conceivable that when goitre has existed in a family for two or three generations, the structure of the thyroid may undergo deterioration in some of the succeeding generation; and the likelihood of the occurrence of such a local degeneration is perhaps augmented by the consideration that all families in which advanced cretinism is prevalent tend to undergo complete extinction within a very few years. According to the Sardinian Commission it is rare for any family residing in the Valpelline to reach a fifth generation. If it were not for immigration into this valley it would become altogether depopulated. Such a deterioration of structure in the thyroid would of course favour still more the development of cretinism, according to the theory suggested. Large goitres are frequently present in the non-cretinous brothers and sisters of those cretins who themselves have no goitres, or only small ones.

The other explanation of the relation between goitre and cretinism is that they are not antagonistic, but consecutive effects of the same unknown cause; that endemic goitre is not a true hypertrophy, but a degeneration of the thyroid, and that although a local disease, yet when inherited, or when supervening early, or when, most of all, the thyroid is congenitally absent, it produces more widely spread and serious disturbance so as to affect the nutrition of the whole body. We shall presently see that this view is supported by the results of experimental and surgical removal of the thyroid. But before mentioning this artificial cretinism it will be well to describe an allied condition, not endemic but sporadic, not congenital but acquired, and sometimes not developed till long after adult life is reached.

CRETINOID CONDITION IN ADULTS.*—Sir William Gull first described in 1873 ('Trans. Clin. Soc.,' vol. vii) a very remarkable condition which he termed *cretinoid*, and which occurs in adults—most frequently in adult women. It is characterised by a change in the features, which become broad and flattened; the eyes appear too wide apart, the alæ nasi become thick, the lips large, the connective tissue below the eyes loose and baggy, and that under the jaws and in the neck thick, and thrown into folds. The tongue is so large as to embarrass articulation, and interfere with wearing false teeth. The hands are broad and "spade-like." The texture of the skin becomes smooth and the hairs thin and scanty, while individually they are thicker and coarser than before. The general hue is pale, sometimes of a dirty white, recalling that of the cachexia caused by malaria, by syphilis, or by lead, sometimes of a clear lemon-yellow like that of Addison's anæmia; but the cheeks are most commonly rosy, not only from dilated veins, but with a diffused purple which looks like rouge. At the same time the disposition of the patient undergoes an alteration, activity of mind giving place to a gentle placid indifference.

* "A cretinoid state supervening in adult life in women (Gull)."—*Synonyms.*—Myxœdema (Ord), Cachéxie pachydermique (Charcot), Cachexia strumipriva (Kocher), Myxœdème primitive et opératoire (Reverdin)—Acquired or adult cretinism.

A striking case of this malady was under the writer's care in Mary Ward (1885). She had the coarse scanty hair, the sallow complexion, with spots of bright red on the cheeks, looking as if she painted, the subconjunctival œdema, the broad, clumsy hands, thick blubber lips, slowness in answering, and tranquil temper. The contrast with a photograph taken ten years before was most remarkable. Though the "bladders" under the eyes looked ready to burst, acupuncture failed to bring out a drop of serum. Another woman was in another division of the same ward under Dr Taylor, and the resemblance of the two was remarkable. In fact, the physiognomy when fully developed is unmistakable.

The year before (1884) there was a man in Philip Ward in an early stage of the same condition. His deliberate answers, heavy aspect, and placid ox-like demeanour were already characteristic. Afterwards œdema, spade-like hands, and the other features above described developed into a typical picture; so that, when he was subsequently in St. Thomas's Hospital, and was shown by Dr Stone at an examination (1887), every candidate recognised the disease at once, even though he had never seen a case.

The uniformity of aspect of this affection, whether occurring in women or more rarely in men, is not the least remarkable of its characters.

Many cases have been now recorded in England since Sir William Gull first drew attention to the subject; by Dr Ord ('Med.-Chir. Trans.,' 1878; 'Clin. Trans.,' xiii, 1880), Sir Dyce Duckworth ('Clin. Trans.,' xiii, 1880), Dr Cavafy (*ibid.*, xv, 1882), Dr John Harley ('Med.-Chir. Trans.,' 1884), Dr Drewitt ('Clin. Trans.,' xvii, 1884), Dr Nixon ('Dublin Quart. Journ.,' Jan. 1887). Cases have also been recognised in France; and in Germany by Riess, Erb, Senator, and Landau ('Berliner klin. Wochenschrift,' 1886, 1887). An elaborate report on 109 cases collected from various sources was published by the Clinical Society as an appendix to the 21st volume, and forms the most complete account of the disease that exists.

The pathology and causes of this disease are quite unknown. It differs from the sporadic cretinism above described in the absence of deformity of the bones, and in the slight degree in which the mind is affected; both these differences probably depend upon its late development. But it seems probable that it depends on the same essential cause, and differs chiefly from the fact that this operates when the stature, the ossification of the bones, and the mental faculties are already fully developed.

The late Dr Mahomed at one time believed that these patients were only examples of chronic Bright's disease; in many cases there is certainly albuminuria, and in some the other signs of granular degeneration of the kidneys, but in others equally marked the urine is perfectly normal. The apparent œdema is not ordinary anasarca, for serum does not exude on puncturing the skin. In a patient who died at St Thomas's Hospital Dr C. Charles found excess of mucin in the œdematous tissues after death; accordingly the name "myxœdema" has been proposed by Dr Ord for this remarkable cretinoid condition in adults. But the condition which suggested it is not constant, for no excess of mucin has been found when sought for in other cases ('Clin. Soc. Report,' 1888, pp. 47—54).

Artificial cretinism.—Before myxœdema was recognised on the Continent, a remarkable condition which has been called "cachexia strumipriva"* was observed as the result of extirpation of bronchocele by Swiss

* *Struma*, as the equivalent of *scrofula*, meant originally a swollen neck, and was long applied indifferently to goitre and to swollen cervical lymph-glands. In Germany it has been restricted to the former meaning, so that struma means a bronchocele.

surgeons, Dr Reverdin, of Geneva, and Dr Kocher, of Berne. After some months the patients who have survived the operation begin to show mental hebetude, pallor, œdema, and some other of the characters of an adult cretin ('Arch. f. kl. Chir.,' 1883, p. 254).

In the following year Schiff, Wagner, and Sanquirico successfully removed the thyroid in dogs, and found that the operation was followed by cerebral disturbance, tremors, and convulsions, at first clonic but then tetanic, ending in death by coma.

Mr Victor Horsley has repeated these experiments on monkeys ("The Brown Lectures," reported in the 'Brit. Med. Journ.,' Jan., 1885). He finds that (usually within a week) after the operation, fibrillary tremors appear in the limbs, which, like those of paralysis agitans, cease on voluntary movement. The monkey becomes anæmic, with increase of leucocytes as well as diminution of red discs. It sits moping and imbecile. The eyelids and abdomen swell. The temperature falls below normal, all tremors disappear, and the animal dies comatose in five to seven weeks. Two remarkable conditions appear to justify the application of the word myxœdema. One is great swelling of the submaxillary and parotid glands, so that the latter become as it were transformed into muciparous glands; and the other is the great increase of mucin in the connective tissues, especially the tendons and superficial fascia, and its appearance in traces in the blood. These facts rest upon analyses made by Dr Halliburton. Similar experiments have been carried out by Professor Welch, of Baltimore ('New York Medical Record,' 1888, p. 368).

Certainly the agreement of these results with those observed in human beings after thyroidectomy, and of both with the cretinoid condition of Sir William Gull and with sporadic and endemic cretinism itself, are very remarkable, and support the view above taken of the relations between cretinism and goitre.

Acromegalia.—A remarkable case resembling myxœdema occurred in a patient of Dr Wilks, a young lady who was at one time not unpleasing in appearance. Her features underwent a most extraordinary change, and she became so hideous that the boys shouted at her when she showed herself in the streets. Her face became elongated; and her nose, lips, and mouth were enormous. There was not the slightest infiltration or hardening of the skin or subcutaneous tissue. She gradually became blind, and had suffered from neuralgia. Her hands could not be fitted with ordinary gloves. She died after six years, "comatose," but at a distance from town, so that there was no autopsy. The urine did not contain albumen. This (Dr Wilks informs the writer) was certainly an example of what was described by Mons. Pierre Marin in 1886 as *Acromegalie.*

About fifteen cases of this disease have been recorded. The hands, feet, and face become hypertrophied and deformed; the bones and skin are the chief seats of the disorder, which might be confounded with myxœdema or with elephantiasis. A monograph by Erb was published in the 'Deutsche med. Zeitung' for Oct., 1887; and cases by Dr Haddon and Mr Ballance and by Mr Godlee ('Clin. Trans.,' xviii and xxi, 196, 201) in 1888. There is sometimes disease of the thyroid and sometimes hypertrophy of the pituitarium; but the latter condition is not constant. There are as many cases in men as in women.

DISEASES OF THE RESPIRATORY SYSTEM

AFFECTIONS OF THE LARYNX AND TRACHEA

"Sputaque per fauces raucas vix edita tussi."—LUCRETIUS.

The laryngoscope and its use—Arrangement of the subjects of this chapter.

LARYNGEAL PARALYSIS—*Of one or both recurrent nerves—Of the abductor muscles alone—Of the adductors—Aphonia and other disorders of the voice.*

LARYNGISMUS STRIDULUS — *Nomenclature — Symptoms and Diagnosis—Pathology—Ætiology—Prognosis and treatment.*

LARYNGITIS—*Acute plastic inflammation—Croup—Its relation to Diphtheria and to Spurious Croup—Its symptoms and causes, anatomy, diagnosis, and treatment—Acute and chronic catarrh of the larynx—Tubercle—Lupus—Syphilis of the larynx—Œdematous laryngitis—Perichondritis.*

TUMOURS OF THE LARYNX—*Papilloma—Polypus, &c.—Sarcoma and Carcinoma—Laryngeal malformations—Foreign bodies in the larynx.*

OBSTRUCTION OF THE TRACHEA—*External compression from enlarged thyroid, aneurysm, &c.—Intrinsic stenosis from syphilis, &c.—Obstruction by impaction of a foreign body—Symptoms, diagnosis, and treatment.*

THE diseases of the larynx, like those of the retina, have within recent times been made accessible, as they never were before, to actual inspection. This is entirely due to the invention of a special instrument—the *laryngoscope*—which was first introduced into medical practice in Vienna by Türck and by Czermak. Türck, in the summer of 1857, began to examine his hospital patients with a laryngeal mirror, such as had been used in physiological researches a few years before by Manuel Garcia, a singing-master, in London, who read a paper on the voice before the Royal Society. Even this was not the first effort to see the interior of the larynx in the living subject, for it had been attempted, though with but little success, by several other observers, including the younger Dr Babington in 1829 and Mr Avery in 1844. Türck suspended his operations in the winter of 1857–8 for want of sunlight, and he lent his mirrors to Czermak, who, setting to work with artificial illumination, became convinced of the extreme value of laryngoscopy for clinical purposes, and after publishing a paper on the subject in March, 1858, in the 'Wiener medicinische Wochenschrift,' travelled over Germany, France, and England, to make it more widely known. He gave demonstrations on his own larynx by direct sunlight at Guy's Hospital in 1859.

The laryngoscope is now in the hands of every practitioner. In using it, the first thing is to secure a bright light, whether from an Argand gas-burner, an oil lamp, or the sun itself. The patient is placed with his back

to the source of illumination, the rays from which pass over his shoulder. The observer seats himself opposite, on a chair slightly more raised, and throws the light upon the lower part of the patient's face by means of a flat or slightly concave mirror, either held in one hand, or, when both are needed, fixed on a band round the head, by a handle held in the mouth or in a spectacle frame made for the purpose. The patient is next made to open his mouth and to protrude his tongue; and this is gently grasped by the patient himself with his thumb and forefinger in the folds of a napkin, so as to leave both the observer's hands free. In some patients the tongue is an unruly member, and the only plan may be to keep it depressed with a spatula bent at a right angle. The light is thrown into the back of the fauces, and kept steadily fixed there. In the meantime a stalked laryngeal mirror is warmed over a flame, or in hot water, or it may be smeared with glycerine; the object being to prevent its surface becoming dimmed by the moisture of the breath. The patient is now instructed to go on breathing quietly and regularly, and to sound an "a" (as in fate) on rather a high note. This brings the fauces into a position advantageous for the introduction of the laryngeal mirror, which is held like a pen between the finger and thumb, and gently but rapidly passed through the patient's mouth until it reaches the uvula, while the stem lies at the angle of the mouth, so as not to interfere with the entrance of light. The observer should learn to use the laryngeal mirror with the left hand, so as to have the right hand disengaged. In traversing the mouth the instrument must have its face turned downward, and it must take a curved course, being kept close to the palate and as far as possible from the tongue. As it reaches the uvula it must be tilted, so that its face looks forwards as well as downwards. It must also be gently pushed upwards and backwards, lifting the uvula and the velum. While this is being done the back of the tongue, the epiglottis, and the interior of the larynx successively become visible, reflected in the mirror. If this is not the case, slight changes in its position or inclination generally bring them into view. As a rule it should not be carried so far back as to touch the posterior wall of the pharynx, which in many patients is far more sensitive than the velum, but some persons bear the mirror perfectly well, even when it is made to rest on the pharyngeal surface. There is seldom any necessity, so far as purposes of diagnosis are concerned, for continuing a single observation for more than a very short time. Should the interior of the larynx not be completely visible, it is best to withdraw the instrument and reintroduce it a minute or two later.

It may well be supposed that the practical use of the laryngoscope is in some cases attended with difficulties. In this matter, as in all others, habit goes a long way. The student finds himself at first baffled, and fails to see anything, whereas the trained observer succeeds at the first attempt; and the patient who when the mirror is first introduced in his mouth thinks he cannot tolerate its presence, becomes after a few trials indifferent. One trouble is with the tongue, which in some persons arches upwards so as to interfere not only with the passage of the mirror, but also with the admission of light to the back of the throat. They must then be directed to practise before a looking-glass until they can "make a wide throat." Or the spatula must be used. Another obstacle is the presence of enlarged tonsils narrowing the faucial space. This is best overcome by using a small mirror. In some patients, again, the uvula and the velum are so irritable that the slightest contact of the mirror causes choking, or retching, or

cough. Sir Morell Mackenzie recommends that to meet this difficulty small pieces of ice should be sucked for fifteen or twenty minutes before the laryngoscopic examination is begun; this, he says, rarely if ever fails to blunt for a time the sensitiveness of the mucous membrane. The best method, however, of securing anæsthesia is to apply cucaine locally. A 20 per cent. solution brushed over the part, or, still better, applied as a spray, will after a few minutes enable one to examine the most sensitive patient. The alkaline bromides taken in a full dose half an hour previously produce more or less anæsthesia of the fauces.

In some patients, who have affections of the throat or lungs, the introduction of the mirror seems at once to be followed by the entrance of a quantity of muco-purulent fluid into the fauces from below, notwithstanding the repeated use of a gargle. Or the uvula may be so long and pendulous that it curls round the under edge of the mirror, interfering with the view of the larynx, or soiling the reflecting surface. The way to correct this is to use a large mirror, so as to lift up the whole of the uvula. But the most serious difficulty of all is caused by a large epiglottis, which hangs backward over the entrance of the larynx in such a way as to prevent anything else being seen. In many cases this obstacle is easily removed by making the patient sound, or attempt to sound, the vowel *e* (as in *feet*) on a high note. The sound itself cannot actually be produced while the tongue is protruded, but Störk says that the effort to produce it is often sufficient to raise the epiglottis. Sometimes the interior of the larynx can be seen, in spite of a pendent epiglottis, if the mirror is placed rather lower in the fauces than usual and with a more vertical inclination of its surface, the patient's head being at the same time thrown far backwards. But it may happen that all these plans fail. An attempt may then be made to raise the epiglottis by a curved sound brought into contact with its posterior surface. In most persons the back surface of the epiglottis is so sensitive that a choking sensation is produced as soon as it is touched. But the use of the cucaine solution enables one to draw the epiglottis forward with the blunt hook in the left hand, guided by the mirror held in the right.

The parts reflected in a laryngeal mirror retain their proper positions so far as concerns the side of the body on which they seem to lie; the left vocal cord is visible upon the left side of the mirror, the right one upon the right side. But in an antero-posterior direction the image is inverted, as if one were looking at the larynx from behind instead of through the mouth. In other words, the base of the tongue and the epiglottis form the top of the laryngoscopic image, and the arytænoid cartilages are seen below.

The appearance of the epiglottis varies widely in different persons. Sometimes little more than the edge of it is seen, sometimes a large part of its posterior surface, which has normally a bright red colour, apt to be taken for morbid congestion. The rest of the laryngeal mucous membrane is of a paler tint, and the vocal cords are white and glistening.

Arrangement.—Most of the affections of the larynx interfere with the performance of both its functions, the formation of the voice and the passage of air into and out of the trachea; and many of them are also attended with other symptoms, as pain, tenderness, a peculiar cough, and dysphagia, as well as with varied and complicated laryngoscopic appearances. It therefore seems advisable to begin the description of laryngeal diseases with certain affections, of which some give rise only to an impairment or loss of

the voice, others only to interference with the breathing, the results of examination with the mirror being correspondingly simple and definite. These are the paralytic and the spasmodic affections of the muscles of the larynx. Although of secondary importance to the specialist, they possess peculiar interest for the physician from their bearing on diseases of distant structures. Their proper place is among the affections of the nerves or nervous centres, but for reasons of practical convenience it is desirable to take them with other affections of the larynx.

Different writers classify laryngeal paralyses in different ways. Störk first discusses affections of the several muscles one by one, as they may theoretically be imagined to occur; afterwards he passes to more complex forms, in which many muscles are involved together. Mackenzie arranges them according to their supposed seat in the bulb, in the trunks of the vagi, or in their laryngeal branches. For our present purpose it will be sufficient to describe such forms of paralysis as are recognisable in clinical practice, noting the muscle or muscles involved in each.

The spasmodic affections to which the laryngeal muscles are liable will follow, then the several forms of laryngitis, and the new growths of the organ. Lastly, the symptoms and diagnosis of obstruction of the larynx and trachea will be considered.

PARALYSIS OF ALL THE MUSCLES SUPPLIED BY THE RECURRENT LARYNGEAL NERVE OR NERVES.—Among the most frequent paralytic affections of the larynx, as might naturally be anticipated, is one which involves all the muscles supplied by the recurrent nerve either on one side or on both. This, when unilateral, is sometimes spoken of as "hemiplegia of the larynx." If a special name is wanted, it would be better to follow the analogy of the word ophthalmoplegia, invented by Mr Hutchinson for a general paralysis of all the muscles of the eyeball, and to speak of "laryngoplegia" when the muscles of both sides of the larynx are affected, and of "hemilaryngoplegia" when the paralysis is one-sided. The paralysis is not quite universal, since the crico-thyroid muscles must escape. But it does not appear that any appreciable physiological action results from their contraction when the other laryngeal muscles are powerless. Moreover, Türck has observed fatty degeneration and atrophy of the crico-thyroid muscle in a case in which the recurrent laryngeal nerve was alone affected, while the superior laryngeal nerve escaped.

It will be necessary to describe separately the effects of unilateral and of bilateral paralysis of the recurrent nerve.

1. *Unilateral paralysis of the recurrens* is characterised by complete immobility of the corresponding vocal cord, whether the patient only continues to breathe or utters a vocal sound. The position occupied by the cord is usually what is termed the "cadaveric position," the same as that assumed by the vocal cords in the dead body, when no muscular force any longer acts upon them—intermediate between phonation and inspiration. But sometimes the cord is drawn inwards to the middle line by the action of the arytænoideus. The outline of the cord looks concave. The summit of the arytænoid cartilage is placed a little further forwards and inwards than that of the opposite cartilage, so that it looks larger from more of its hinder surface being seen. When a sound is uttered, the opposite arytænoid cartilage moves further than usual, and the unaffected vocal cord is drawn up to and even across the middle line, until it may come close to the paralysed

one, and so the chink of the glottis become oblique. At the same time the summit of the mobile arytænoid cartilage crosses in front of the cartilage on the paralysed side. When paralysis has lasted a long time the affected cord may be seen to be obviously atrophied, and may oscillate backwards and forwards as the stream of air passes over it.

The voice of a patient with paralysis of one recurrent nerve is less altered than might have been expected. It is often weak and hoarse, and sometimes breaks into a falsetto as soon as an attempt is made to speak forcibly. A point to which Gerhardt has drawn attention is that when two fingers are placed, one on each side of the thyroid cartilage, while the patient is speaking, a more distinct vibration can be felt with one finger than with the other. There is not the slightest dyspnœa.

2. *Bilateral paralysis* of all the muscles supplied by the two recurrent nerves is characterised by immobility of both vocal cords in the cadaveric position. It is to be noted, however, that the paralysis is often less complete on one side than on the other.

There is complete aphonia, the voice being reduced to a whisper; and the patient is unable to cough or to expectorate forcibly. There is no dyspnœa, at least in adults.*

Diagnosis.—It must be borne in mind that immobility of the vocal cord, whether on one side or on both, is not in itself proof of paralysis. As Dr Semon pointed out in the 'Medical Times and Gazette' for 1880, precisely the same laryngoscopic appearances may be the result of ankylosis of the crico-arytænoid joints. This fact had, indeed, been recognised to some extent by previous writers, especially in Germany. But it seems to have been supposed that perichondritis, before leading to fixation of the joint, must almost of necessity be attended with suppuration. Dr Semon maintains that non-suppurative arytænoid synovitis may obliterate the synovial cavity, and unite the cartilages to one another. It is of course only when the arytænoid cartilages are so fixed as to bring the cords into the cadaveric position that the case can be taken for one of recurrent paralysis, and then, as Dr Semon admits, diagnosis is sometimes impossible.

Pathology.—The causes of paralysis of the muscles supplied by the recurrent nerve or nerves fall into two groups.

(1) There may be *central* disease of the nuclei of the nerves of the eighth pair, on one side or on both, in the pons; as in bulbar paralysis or multiple sclerosis. Such paralysis is usually bilateral and almost always secondary.

(2) The disease may be *peripheral*, interfering with the roots or trunks of the spinal accessory and pneumogastric nerves near the base of the skull, or with the trunk of the vagus after its separation from the spinal accessory or with the recurrent laryngeal branch. As a rule, the paralysis is in such cases unilateral. Indeed, by far the most frequent cause of paralysis of the muscles of one half of the larynx is aneurysm of the aorta, in which case the affection is most often on the left side. Aneurysm extending to the innominate artery may, however, compress the right recurrent nerve. Again, mediastinal growths of various kinds may interfere with the left nerve,

* This is a point about which there was at one time some divergence of opinion, but it seems now to have been finally settled. Scheck has recorded a case in a boy of seven, whose breathing was in no way interfered with. Ziemssen, however, still says that in deep inspiration the cords may be drawn a little further inwards than before, and that a stridulous sound may be produced.

while either the left one or the right may be pressed upon by an enlarged thyroid, or by cancerous tumours of the œsophagus or the vertebræ. These last-mentioned peripheral causes not infrequently affect each recurrent nerve in succession and produce bilateral paralysis. Mackenzie has published a case in which there were two aneurysms of different parts of the aorta, one of which compressed the right and the other the left recurrent nerve; and in 1866 Dr Bäumler recorded an interesting example of bilateral paralysis of the recurrent nerves, apparently due to the pressure of a large pericardial exudation.

It is a singular fact that peripheral interference with a single vagus sometimes causes precisely the same result. This was pointed out by Dr Bäumler in the 'Pathological Transactions' for 1872, and two years later Dr George Johnson made it the subject of an interesting paper in vol. lviii of the 'Med.-Chir. Trans.' The only possible explanation seems to be that ascending neuritis spreads to the bulb by the centripetal fibres of the affected vagus. If so a lesion involving only the recurrent nerve and not the vagus itself must be incapable of producing the same effect, as was well shown by Dr Semon in the 'Berl. klin. Wochenschrift' for 1883. The further spread from one lateral nucleus to the other may be explained by the existence of a close physiological connection between the nuclei of the two sides. But Lockhart Clarke further showed that some of the fibres of origin of the spinal accessory nerve (which include the root-fibres of the recurrent laryngeal) actually pass across the middle line, being derived from the opposite nucleus; so that it is not inconceivable that disturbance of a single nucleus may directly cause bilateral paralysis.

The paralysed muscles become atrophied, as was clearly shown in Dr Bäumler's case. Indeed, there is no form of paralysis in which the resulting muscular atrophy is more obvious on dissection than paralysis of the muscles supplied by the recurrent nerve. In unilateral cases, in particular, the contrast between the whitish-yellow, shrunken crico-arytænoideus posticus on the affected side and the red fleshy muscle opposite to it is more striking than any similar condition in other parts of the body. The recurrent nerve also, below the point at which it is compressed, is greatly wasted and of a dull grey colour.

The *prognosis* of recurrent paralysis depends upon its cause. In Bäumler's case, in which it was dependent upon exudation into the pericardium, the patient, whose voice had been reduced to a whisper, recovered quickly as the effusion underwent absorption; within four or five weeks from the time when he first became hoarse he could speak nearly as well as ever. As a rule, the primary disease is incurable, and the paralysis persists until death.

A goitre, if this is present, might be actively treated, but it is useless to prescribe strychnia or to apply galvanic or faradic currents.

PARALYSIS OF THE ABDUCTORS OF THE CORDS (THE CRICO-ARYTÆNOIDEI POSTICI).—Since recurrent paralysis is commonly the result of a morbid process gradually destroying the fibres of the nerve or the nucleus from which they arise, it is natural enough that cases should be met with in which some only of the muscles supplied by it suffer, while others escape. But it is a remarkable fact that such incomplete forms of paralysis invariably affect one particular pair of muscles, the *crico-arytænoidei postici* which widen the space between the cords on deep inspiration. Indeed, many cases have been recorded of paralysis of both recurrent laryngeal

nerves, and yet no muscle has been affected except the posterior crico-arytænoid on each side. Gerhardt published the first example of such an occurrence in 1863, and an admirable lecture on the subject by Riegel may be found in the second volume of 'German Clinical Lectures,' edited for the New Sydenham Society in 1877. The reason for this proclivity of the crico-arytænoid is still obscure. When there is a nuclear lesion in the bulb we may suppose that the nucleus for the fibres to the abductors is distinct from that for the other laryngeal muscles, just as we explain how in most cases of bulbar paralysis the lower part of the face is affected while the upper part escapes. But the result is just the same when the disease involves the upper part of the vagus or the recurrent nerve. It would seem that the fibres to the abductors undergo destruction earlier than those to the adductors. Indeed, Riegel actually found in one of his cases, in which both the recurrent nerves were embedded in dense connective tissue, that although the majority of the fibres had undergone fatty degeneration, some still retained their normal structure. During the meeting of the International Congress in London, in 1881, Rosenbach pointed out that in paralytic affections of the limbs there is an analogous fact in the greater liability of the extensors and abductors than of the adductors to suffer. Moreover, when the laryngeal muscles are affected with spasm, the abductors are invariably overpowered by the adductors. Possibly the innervation of the latter group of muscles is better than that of the former.

The laryngoscopic appearances which characterise paralysis of a *single abductor* are that the corresponding vocal cord lies more or less near the median line, and does not move outwards as it normally should when the patient takes a deep breath. On the other hand, during vocalization it moves freely inwards. Hence unless the state of the larynx is carefully inspected while the patient is not, as well as while he is, attempting to utter a sound, this affection will be overlooked.

When both abductor muscles are paralysed, the two cords lie nearer one another than they do in health. The degree to which they are approximated varies with the duration of the paralysis. In cases of long standing they may lie so close together that during inspiration it is scarcely possible to perceive the slightest chink between them; while during expiration they slightly recede from one another. Riegel argues that this extreme narrowing of the glottis is the result of a gradual contraction of the antagonists of the paralysed muscles, exactly analogous to that which occurs in motor paralysis of the eyeball, the face, or the limbs. But in a case recorded by Feith it seems to have come on only a few days after the paralysis.

Another factor in the production of such a very marked stenosis of the glottis during inspiration is in all probability a sucking in of the cords towards one another, in consequence of the diminution of atmospheric pressure upon their lower as compared with their upper surfaces.*

Apart from the results of a laryngoscopic examination, subjective symptoms of paralysis of a single crico-arytænoideus posticus are altogether want-

* In two cases seen by Dr Semon there was a modification of the usual laryngoscopic appearances; the cords were close together only along their anterior two thirds, and diverged posteriorly so as to leave a triangular opening with its base at the interarytænoid fold. It is suggested that this depends upon a limitation of the paralysis to the outer fibres of the crico-arytænoidei postici, their inner fibres escaping. Rühlman has, in fact, endeavoured to show that each of these muscles consists of two portions having different functions: the inner portions, he says, draw the arytænoid cartilages downwards and outwards upon the cricoid; the outer portions rotate the arytænoid cartilages upon their vertical axis.

ing. The patient's voice is of course perfect, and as there is plenty of room for the entrance of air, he experiences not the slightest dyspnœa, even on exertion. It is this fact which gives clinical importance to Dr Semon's observations of the invariable occurrence of paralysis of this muscle as the result of disease of the recurrent laryngeal, or of the vagus trunk or nucleus. He has shown that there are many cases of aneurysm, of mediastinal growth, of carcinoma of the œsophagus, and even of disease at the base of the brain, upon which a routine use of the laryngoscope throws as much light as does the routine use of the ophthalmoscope on cases of cerebral tumour or chronic Bright's disease.

When *both abductors* of the vocal cords are paralysed the symptoms may be of the most urgent and dangerous character. The voice is still unimpaired, unless there is a coincident inflammatory affection of the larynx. But there is often most distressing inspiratory dyspnœa, the air being slowly drawn into the chest with loud stridor, and the patient becoming livid, with cold extremities, and ultimately dying of suffocation. In such cases the laryngoscope is needed, not so much to distinguish the affection from other diseases of the larynx as from stenosis of the trachea.

The mere fact that during inspiration the cords are drawn close together near the middle line is not of itself proof that there is any paralysis of the abductors; there may be a spasmodic affection of the adductors. What marks the difference is the way in which the dyspnœa begins. A primary spasm is rapid in its development; the contraction of antagonists which obstruct the entrance of air in cases of paralysis of the abductors comes on slowly. At first there is difficulty of breathing only when the patient makes some effort or exertion; then it gradually becomes persistent and increases in severity.

With regard to the *causes* of paralysis of one or both of the abductor muscles of the cords, we have already seen it may be either central, or due to some affection involving the upper parts of the vagi, or the recurrent nerves in their course. In the 'Pathological Transactions' for 1882 a case is recorded by Dr Whipham, in which a bilateral paralysis of the abductors was dependent upon implication of the left pneumogastric and recurrent nerves in the walls of a thoracic aneurysm. In some cases this form of paralysis has followed diphtheria, and once it has been a sequela of facial erysipelas. It has been ascribed to exposure to cold; and Mackenzie thinks it may be due to direct pressure on the fibres of the crico-arytænoidei postici through the anterior wall of the pharynx in swallowing. Indeed, a case by Ott is cited, in which it was the result of a piece of meat being impacted for twenty-four hours at the opening of the œsophagus.

Prognosis and treatment.—In some cases recovery takes place after weeks or months, apparently independent of treatment. Much more often the affection remains incurable. Sometimes a large amount of relief to the subjective symptoms, with temporary disappearance of the attacks of dyspnœa, may be attained by the subcutaneous injection of strychnia ($\frac{1}{30}$ grain of the sulphate gradually increased to $\frac{1}{10}$, daily), or by systematic use of faradisation. The proper method of applying the current is by a laryngeal electrode, with a flat, spade-shaped extremity, that can be laid upon the pharyngeal mucous membrane, over the paralysed crico-arytænoid muscles. It is, however, very important that one should not be content with a partial success from this or any other plan of treatment. So long as the objective signs

of stenosis of the glottis continue, there is always the risk of the sudden supervention of a fatal attack of dyspnœa, as in a case recorded by Dr Semon in vols. xi and xii of the 'Clinical Society's Transactions,' in which the patient's life was just saved at the last moment by tracheotomy, and by artificial respiration continued for three and a half hours. The rule, therefore, seems to be that the trachea should be opened, as a measure of precaution, in every case of paralysis of the abductors, if attended with considerable stenosis of the glottis and with marked dyspnœa.

PARALYSIS OF THE ADDUCTORS OF THE CORDS.—There is, in every respect, the most marked contrast between paralytic affections of the abductors and of the adductors of the cords. They differ in their causes, in their symptoms, and in their course. Paralysis limited to the adductors is never due to organic lesions affecting either the fibres of the vagi or of the recurrent nerves, or their nuclei of origin; while it is not at all infrequent as the result of other causes, which seldom or never give rise to paralysis of the abductors.

The laryngoscopic appearances which accompany paralysis of the adductors vary somewhat according to the precise seat of the affection; for it is to be borne in mind that, instead of being (like the abductors) a single pair of muscles, the adductors consist of a group of muscles on each side of the larynx, any one of which may probably be paralysed separately. These muscles are classed together by Henle and Luschka under the name of the *sphincter sive constrictor rimæ glottidis*. Now, the pair of muscles which in this country are known as the thyro-arytænoidei are commonly supposed to have the function of relaxing the vocal cords, thus acting as antagonists of the crico-thyroidei. But the German anatomists divide the muscles in question into two on each side, the "thyro-arytænoidei externi," and the "thyro-arytænoidei interni." The thyro-arytænoidei interni are described as a pair of prism-shaped muscles, each of which has one of its edges projecting into the substance of the corresponding vocal cord. Their function is to straighten and approximate the cords in the act of vocalisation. In other words, they co-operate with, instead of being opponents of, the crico-thyroidei; and they are sometimes spoken of as the "internal tensors," the crico-thyroidei as the "external tensors" of the cords.

If, then, the thyro-arytænoidei interni are paralysed, the effect is that when the patient attempts to speak, the cords, instead of being straight, are both concave, and enclose between them a narrow oval space. If the affection is unilateral, the space appears bounded by a straight and a curved line. The width of the space between the cords depends partly upon the pitch of the sound which the patient is trying to utter, being greater when the pitch is low than when it is high. For we must remember that every degree of loss of power of the vocalising muscles may occur, from the slightest possible paresis up to the most complete paralysis.

Isolated paralysis of the arytænoideus muscle causes the rima *glottidis* to gape posteriorly, between the two arytænoid cartilages, while the cords themselves meet perfectly. The laryngoscopic appearance is then that of a triangle behind, with its apex prolonged into the normal narrow chink.

Another laryngoscopic appearance sometimes observed is that the processus vocalis of each arytænoid cartilage forms an angle inwards; it is believed to indicate a combination of paralysis of the thyro-arytænoidei interni with that of the arytænoideus.

Isolated paralysis of the two crico-arytænoidei laterales is said to produce in the laryngeal mirror a figure with an angle outwards corresponding with the processus vocalis on each side, the rima being quadrilateral and lozenge-shaped. Ziemssen, however, doubts whether such an affection of these muscles ever occurs.

If all the muscles forming the sphincter rimæ glottidis are paralysed at the same time, the rima glottidis, when the patient attempts to speak, forms an oval space; but this is not bounded behind by the point of contact between the two processus vocales, as when the thyro-arytenoidei interni alone are affected; it extends backwards between the arytænoid cartilages to beneath the posterior commissure.

Lastly, according to Mackenzie, it is possible to recognise by the laryngoscope a paralysis limited to the crico-thyroidei muscles, which differ from all the rest in receiving their supply from the superior laryngeal nerves. The appearance which indicates such an affection is said to be that the rima glottidis presents a wavy outline.

Paralytic affections of some of the muscles forming the sphincter rimæ glottidis may be associated with spasm of others; so as to make it impossible to determine the precise character of such cases.

The main symptom in all cases of paralysis of the adductors of the cord is impairment of voice, extending from hoarseness up to the most complete aphonia, so that the patient may be utterly unable to speak except in a whisper.* Paretic states of the various muscles give rise not only to hoarseness, but to an undue sense of fatigue in speaking or singing, and to inability to maintain the voice for long or to shout.

FUNCTIONAL DISORDERS OF THE VOICE.—*Aphonia* is not only a symptom of paralysis of the adductor muscles from organic and permanent cause. It is also a functional and transitory condition. Probably in some of these cases there is no true paralysis, for many patients who have complete aphonia nevertheless continue to be able to cough, and also to sneeze; and these reflex acts are accompanied with a laryngeal sound, which clearly proves that for this performance the cords can be perfectly well brought into contact. This is especially apt to be the case with hysterical women, who, in fact, are the most frequent subjects of paralysis of the adductor muscles. Mackenzie says that he has met with such an affection in girls only eight or ten years old; but hysteria is by no means unknown at such an age. Phthisis is another disease in which paralytic aphasia is not infrequent. Mackenzie in 1865 examined at the Brompton Hospital thirty-seven consumptive patients in whom the voice was affected, and found that in eleven of them "the affection was purely functional." Sometimes paresis of the thyro-arytænoidei and transverse muscles follows an attack of laryngeal catarrh; it may then continue long after the mucous membrane ceases to show congestion. Mackenzie says that this frequently occurs in public speakers, especially in clergymen (*aphonia clericorum*). In other cases paralysis of the adductors of the vocal cords appears to be caused by the direct action of cold upon the affected nerve-twigs or muscles. Sometimes

* Whether such a total loss of voice is ever the result of the isolated affections of individual muscles seems to be doubtful. Ziemssen says that this effect can hardly be produced even by paralysis of the thyro-arytænoidei interni, so long as the crico-arytænoidei laterales and the arytænoideus remain in action, and bring together the processus vocales of the two arytænoid cartilages.

it is the result of over-exertion of the voice, as in singers; such cases are usually slight and transitory. But Mackenzie speaks of paralysis of the thyro-arytænoidei interni as being occasionally the result of an actual "sprain" of the muscular tissues in some undue effort at vocalisation, and as then proving exceedingly intractable. Paralysis of the transversus (or of some of the other muscles) seems to be sometimes dependent upon gummatous or other lesions directly destroying the substance of the muscle. Mackenzie also mentions poisoning by lead or by arsenic as a possible cause of paralysis limited to one or more laryngeal muscles; a case which he cites occurred in a painter, and is described as complete loss of power of the adductor of the right vocal cord.

It is a peculiarity of hysterical aphonia, which (as we have seen) is dependent upon paralysis of some or all of the adductors of the cords, that the patient is apt to regain the voice suddenly under the influence of violent emotion. The recovery in such cases may be either transitory or permanent. Since the introduction of the laryngoscope it has become the usual practice to treat such cases by the application of a powerful induced current to the interior of the larynx, and this leads to brilliant success. The method is as follows:—One electrode is connected with a metal plate fastened upon a necklet which is put round the patient's neck so that the metal plate rests on the front of the larynx. The other electrode consists of a small metal ball or sponge fixed to the end of a long curved stem, which can be passed down into the space between the vocal cords. The stem of this "laryngeal electrode" transmits no current until the end of it has entered the larynx; at that moment the operator with his finger presses down a key by which the circuit is completed. All observers seem to be agreed that the only way by which it is possible to count upon a successful result from this procedure is that of using on the very first occasion a current of great power, which is of course exquisitely painful, and makes the patient involuntarily utter a loud articulate cry; whereupon the electrode is instantly withdrawn. If less than this be attempted at first, Dr Semon finds that electricity often fails altogether. Mackenzie speaks of having cured cases of six, seven, eight, and even ten years' standing. Sometimes it is necessary to repeat the application of the induced current several times and during many weeks before a permanent result is attained. In some cases, however, the introduction of an electrode within the larynx is not required; it is sufficient to apply a current across the neck from one side to the other, the electrodes being placed one over each thyroid cartilage. Mackenzie has also occasionally succeeded with stimulating steam-inhalations of oil of Calamus aromaticus, or of creosote; or with a strong solution of nitrate of silver (ʒj ad ℥j) applied with a brush or as a spray.

Alteration of the pitch of the voice.—To complete the description of functional disorders of the voice certain cases must be mentioned in which there is an alteration in its pitch. Störk relates instances of children with an unduly low voice, and others of young men with an excessively high pitch, after their voice has broken at puberty. The remedy, in the former case, is to practise speaking with a falsetto voice; in the latter, with a bass voice. This is often perfectly successful, if sufficient perseverance be shown. Sometimes the desired change in the pitch of the voice is brought about very rapidly; for instance, a young man, aged eighteen, who for about a year had spoken in a falsetto voice which contrasted ridiculously with his

broad and well-built figure, was told to utter the vowel *u* for an hour daily in as deep a voice as possible, at the same time holding the head fixed, this last direction being merely for the purpose of keeping up his attention; on the fourth day his voice became normal, and from that time it remained so.

Spasms of the tensors of the vocal cords is another curious affection. This is characterised by a state of the voice so peculiar as to be at once recognised by those who are familiar with it. The following is Mackenzie's description:—"The patient is often able to produce some notes, either in his own natural voice, or in a slightly muffled tone; but, while he is speaking in this way, the current of the voice seems to be partially interrupted, and the sound conveys the idea of an arrested action of the respiratory muscles. In fact, it is very much like the straining and rather suppressed voice of a person engaged in some act requiring the prolonged and steady action of the expiratory muscles (parturition or defæcation). The patients often complain that they cannot get their voice out. After speaking a word or two, or even several sentences, in this peculiar tone, the patient may again utter a few words in a comparatively healthy voice, and then may immediately relapse into the diagnostic intonation." Or there may be a complete absence of sound, the lips moving in the usual way for the utterance of words and phrases, which nevertheless are lost in silence. A clergyman is described as having been greatly distressed by the fact that while he kept on reading the service some of the words dropped soundless from him.

Mackenzie, in 1880, had seen only thirteen cases of this kind; eleven of them were in men, ten being clergymen, and the eleventh a barrister; two were in women, both of whom had had constantly to speak to deaf relatives. Doubtless, therefore, it is the result of over-use of the voice. The onset of the affection was sometimes gradual, sometimes sudden; in the latter case it was attributed by the patients themselves to "catching cold." No treatment was permanently successful in any instance.

LARYNGISMUS STRIDULUS.*—Obstruction of the glottis by spasm is present in many diseases and not in one only, and in this disease there is, or may be, spasm of many other muscles besides those of the larynx. Hence it is both less and more than spasm of the glottis. Laryngismus stridulus in its extreme forms exhibits an organised series of phenomena, comparable only with those of an epileptic fit. As we shall presently see, there have been doubts as to whether it is dependent on a primary disturbance of the central nervous system, or whether it is reflex and due to peripheral irritation. But however this may be, its proper place in the nosology is certainly among the neuroses, where it is placed in the last edition of the College of Physicians' 'Nomenclature of Diseases,' and the only reason for discussing it in this place is that clinically its symptoms have to be studied in relation with those of laryngeal diseases. The name of laryngismus stridulus was invented by Dr Mason Good, and has since been very generally adopted; that of "child-crowing" was proposed by Dr Gooch. At one time it was called "thymic asthma," under the idea that it was caused by enlargement of the thymus. This hypothesis was maintained by Kopp in 1830, but was disproved by Bednar in 1852, and by Friedleben in 1858.

* *Synonyms.*—Spasmus glottidis—Spasmodic croup—Child-crowing—Thymic asthma—Millar'sches Asthma.

Symptoms.—In its simplest form laryngismus stridulus consists of a disturbance of the natural rhythm of the respiration, such that the child (for the disease is one of childhood) first holds its breath and then makes a more or less noisy inspiration. This occurs again and again at varying intervals —perhaps especially on the first waking from sleep—but neither parents nor nurses may attach much importance to it, thinking it is merely a "catching in the breath." Gradually the paroxysms assume a more serious character, or they may have begun in a severe form. The child shows signs of great distress and alarm. Its neck and back become arched, its chest and abdomen rigid, its eyes turned upwards, and its limbs tonically contracted, the thumbs being bent inwards, the fingers extended, and the wrists flexed, while the legs are thrust out, the soles turned inwards, and the toes stretched wide apart. Its face, at first pale, may turn purple, or of a ghastly leaden colour. Sometimes the fæces and the urine are discharged involuntarily, sometimes there is a noisy expulsion of flatus. After a few seconds, or a minute or two at the longest, the spasm yields. In all probability, while it lasts, the glottis is completely closed. As it passes off a chink is formed, through which the air can slowly enter, making a loud crowing sound. This usually ends the seizure, but sometimes two or more paroxysms occur in rapid succession. According to Steffen in 'Ziemssen's Handbuch,' a few unrhythmical and noisy expirations, and one or more whistling or crowing inspirations, precede the stoppage of the breath which constitutes the central feature of the attack. After the paroxysm is over the child frets or cries for a little while, or falls asleep; or it may at once seem to be as well as ever, returning with as much zest as before to its toys, or to any game in which it may have been engaged.

Fatal event.—But it does not always happen that a seizure of laryngismus stridulus ends favourably. Sometimes, instead of relaxing, the spasm persists until life is extinct. There is then, of course, no crowing sound, and there may be nothing whatever to indicate the cause of a death for which the parents are utterly unprepared. Some years ago the author made an autopsy on the body of an infant aged sixteen months, which had died suddenly and had been brought to the hospital by its mother. Nothing was found to account for such an occurrence, but on inquiry next day it was found that the child had previously had attacks of "child-crowing." Steffen relates the case of an infant six months old, who was one night taking the breast when it was attacked with slight spasm of the glottis, after which it went on sucking. However, the attack returned more severely, and the child fell backwards. Within a quarter of an hour Steffen was at the spot. The child had been laid in its cot under the idea that it was sleeping. He found it livid and dead, without any signs of spasm of the limbs or of any other part than the larynx. Sometimes, however, death is preceded by tremulous twitchings of muscles, or by a more or less complete epileptiform convulsion, exactly as is the case in other forms of asphyxia.

As may well be supposed, after death from laryngismus stridulus the brain and its membranes are found gorged with blood, but there is no reason whatever for imagining that the congestion is antecedent to the paroxysms or concerned in producing them. When the disease has been of some standing the lungs may, according to Steffen, be found emphysematous; if this is really the case, in uncomplicated instances of an affection in which there is only inspiratory dyspnœa, it has an important bearing on the theory of emphysema in general.

Pathology.—It is clear that many other muscles besides those of the larynx are concerned in the production of attacks of laryngismus stridulus. The preliminary stoppage of breathing may be perhaps attributed to a mere inhibition of the respiratory centre, rather than to spasm. Steffen says that with few exceptions the diaphragm and the chest walls are "in the inspiratory positions" when the pause occurs, but he can hardly mean that they are in the position which they normally occupy when inspiration is completed; for if so, how is the prolonged inspiratory movement that immediately follows to be satisfactorily accounted for? Even as to the part played by the intrinsic muscles of the larynx at the time when the crowing inspiration takes place there has been some difference of opinion. Some writers have doubted whether there is only (as is generally supposed) a spasm of the sphincter of the glottis, and have thought that there must be also paralysis of the opposing crico-arytænoidei postici. Now, in experiments on rabbits it is found that if the superior laryngeal nerve on one side is divided, and its central end is then excited by a faradic current, the result is a strong bilateral adduction of the vocal cords. But if stimulation of the centres in the bulb thus normally tends to evoke adduction, the only question that remains is whether, when it occurs, the nerve-fibres to the abductors are left out of the reflex circle, or whether these muscles are overpowered by their antagonists. The preponderant action of the sphincter seems best explained by the latter hypothesis, supported as it is by the facts already mentioned with regard to the relative liability of the two groups of muscles to paralysis.

Ætiology.—The most obvious fact in the ætiology of laryngismus stridulus is its relation to *rickets*. According to Steffen, at least nine tenths of all cases occur in rachitic children. By Elsässer it was thought that craniotabes was in a special way associated with the development of laryngismus, but this appears not to be the case. It is doubtless in consequence of their having all in turn been sufferers from rickets that laryngismus has been sometimes noticed in several children of the same parents. Dr Reid mentions a family of thirteen, of whom only one escaped laryngismus and four died of it. Children affected with laryngismus are not infrequently fat, so that their parents fondly imagine them to be hearty and strong; but this is quite compatible with their being highly rachitic. The relation of laryngismus to other rachitic spasmodic neuroses, tetany and carpo-pedal contractions, has been already noticed (p. 711).

A curious point, noticed by Henoch, is that laryngismus stridulus is of far more common occurrence in the early part of the year than later on. Dr Gee, among sixty-three cases, observed no fewer than fifty-five between the months of February and June inclusive. His explanation is that the extent to which children are kept indoors during the winter increases the irritability of their nervous centres.

The *age* at which laryngismus stridulus begins is generally from four months to two years. But Dr Reid relates cases in infants only a few hours after birth; and others occur in children of various ages up to nine years.

There is, however, one remarkable fact which shows that rickets can only be looked upon as a predisposing cause of laryngismus, namely, that the latter affection is much more frequent in boys than in girls. Steffen cites figures from different observers, showing that the proportion of males to females is higher than that of two to one. In relation to it he alludes to the circumstance that membranous croup also is far more commonly seen

in male children than in female; and this is still more markedly the case with false croup. Such an indication of a relationship between laryngismus and other diseases of the main air-passages no doubt tends to make one hesitate in assigning a principal place in its pathogenesis to an over-excitability of nerve-centres in the bulb.

What, however, are the facts as regards the occurrence of tetany and carpo-pedal contractions in children of the two sexes? On reference to some of the recently recorded cases of tetany it appears that the patients have been chiefly boys. And although chorea is well known to be more common in girls than in boys, one must remember that its seat is probably not in the bulb, but in the large basal ganglia; and, moreover, that it occurs at a later age, when the course of development may probably have altered the relations between the two sexes as regards the susceptibility of the nervous centres. In this connection it is perhaps worth noting that many more males than females, whether children or adults, succumb to tubercular meningitis.

Whatever may be the real bearing of these facts upon the ætiology of laryngismus, we may without hesitation reject another hypothesis which a few years ago had many supporters, namely, that it is always of reflex origin and dependent upon some irritation conveyed upwards to the nervous centres from the periphery. This was declared by Dr Marshall Hall to be the "only true mode of viewing" the disease, and he maintained that the cause of it was almost always to be found in a morbid state of the teeth, in disorder of the stomach, or of the bowels. A few years earlier, Dr Hugh Ley had endeavoured to prove that laryngismus was the result of mechanical irritation of the vagi nerves by enlarged bronchial or cervical glands. He did in fact show that glandular enlargement was often present. But it certainly may be absent; and no morbid anatomist who has learnt how frequently in autopsies upon children the vagi nerves are found surrounded by caseating glands, will assign to them any important share in its ætiology. When the nervous centres are in a morbid state, with gradually increasing irritability of their cells, explosions may be directly brought about by stimuli conveyed from internal organs, just as in severe cases they can be excited by merely touching the surface of a child's body. The analogy of epilepsy and of many other neuroses is quite in favour of this hypothesis. Still, the fact that what must be a constant stimulation produces intermittent effects shows how preponderant is the paroxysmal state of the nervous centres.

Diagnosis.—There is no other affection with which laryngismus stridulus can be easily confounded by those who know its symptoms. At the moment of an attack, if there had been none previously, or if the child's history were unknown, one might suppose the obstruction of the larynx to be due to a foreign body. It would be quite right to pass one's finger to the back of the throat to settle the question; but the subsidence of the spasm would show the real nature of the disease.

Prognosis.—The natural course of laryngismus stridulus, when undisturbed by treatment, varies greatly in different cases. Sometimes the attacks continue to be slight, and occur at wide intervals; sometimes they increase in severity and in number, until there may be thirty or forty in the twenty-four hours. In either case they may after a few weeks gradually become less frequent, until at length they cease entirely. Some writers have said that one case in every three proves fatal; but if mild and severe cases be reckoned together the prognosis is much more favourable. It is worse when

the child is very young; and according to Steffen it is better in girls than in boys.

Treatment.—The treatment of laryngismus stridulus is, in the first place, that of rickets—sunlight, fresh air, good food, cod-liver oil. A change to the country or the sea-side is often quickly followed by the subsidence of the attacks. Dr Ringer strongly advocates sponging with cold water twice or thrice daily. Of drugs, the most trustworthy is bromide of sodium, of which from three to five grains may be given as a dose. Many find syrup of chloral hydrate (ɱx—xx) the most efficient medicine; it is readily taken by young children, and is perfectly safe.

It is right to look out for any condition of distant parts that may possibly be concerned in irritating the nervous centres. If the gums are hot and tense they should be lanced. If the bowels are loaded a few aperient or vermifuge doses should be given; but it will not often be found that great results are thus attained, either in diminishing the severity or in reducing the frequency of the seizures.

When a paroxysm is so prolonged as to require treatment cold water may be dashed over the face and head, the body being perhaps immersed in a warm bath; or a bottle containing ammonia may be held to the nostrils. The inhalation of chloroform is recommended by some writers. In severe cases the nurse should be taught beforehand how to act should the emergency arise. Even if life should have apparently become extinct it may sometimes be restored by artificial respiration, as was pointed out many years ago by Mr Johnson in the fifth volume of the 'Dublin Hosp. Reports.'

Inflammatory affections of the larynx.—Laryngitis differs widely in its seat, its symptoms, and its course. Some forms mainly affect the mucous membrane, whereas others start in the deeper structures of the larynx. Clinically superficial laryngitis presents two varieties: first, those attended with dyspnœa so severe that it threatens life—these are commonly known as cases of *croup;* secondly, cases in which the chief symptom is impairment of the voice—*acute* or *chronic* catarrh of the larynx.

CROUP.*—In 1765 Dr Francis Home, of Edinburgh, published a tract of sixty pages, an 'Inquiry into the Nature, Causes, and Cure of the Croup,' a disease which he declared to be entirely unrecognised by medical writers, although it was known to the common people of Scotland by several distinct names, of which "croup" is one. In each of the *post-mortem* examinations which he made he found the trachea lined by a more or less complete membranous layer. Thirty-six years later, in 1801, another Scotch physician, Dr John Cheyne, wrote on the same subject a work which has become classic; his views upon the pathology of croup were the same as those of Home. In the meantime the existence of the new disease had become matter of common knowledge in England as well as on the Continent. The relation of certain other affections of the throat to Home's disease were discussed by his successor, so that in Cheyne's work a controversy was begun, which even now is not finally settled.

The word Croup is of English origin, and applies to the peculiar sound of the breathing in this disorder. It is properly a clinical term, but has

* *Synonyms.*—Cynanche trachealis—Cynanche stridula—Angina suffocatoria—Morbus strangulatorius.—*Fr.* Angine couenneuse trachéale ou membraneuse; Le croup.—*Germ.* Häutige Bräune; Der Croup.

unfortunately been associated with the pathological condition of membranous laryngitis. We have to inquire: (i) whether the membranous "croup" of the Scottish writers is distinct from the disease which has since been called *diphtheria;* (ii) whether it is distinct from a milder affection of the air-passages, named *false or spurious croup, stridulous angina, stridulous laryngitis, spasmodic laryngitis,** or *inflammatory croup.*

(i) *Croup and diphtheria.*—The controversy in regard to this point was definitely begun by Bretonneau, of Tours. In his earlier 'Memoirs on Diphtheria,' 1821—1826, he made it his principal object to prove that that disease and croup are identical. This view was adopted by his pupil, Trousseau, by Guersent, Barthez, and almost all the other leading French physicians. In England it was for a long time repudiated, but latterly it has met with a much more favourable reception. The late Dr Hillier advocated it in 1862, and since then Dr Johnson, Dr Semple, Sir John Cormack, and many others.

In the first place, we must remember that both Home and Cheyne were perfectly acquainted with the fact that the disease which they described was liable to be confounded with one which affected the larynx secondarily, having its original seat in the fauces. Home, in quoting Dr Hare's graphic account of the "morbus strangulatorius" in Cornwall (which was epidemic diphtheria in its most typical form), says that that complaint "appears more nearly allied to the malignant sore-throat, although it sometimes attacked the trachea." And Cheyne begins his section on diagnosis by remarking that he had seen several children, whom he would have supposed to be suffering under the second stage of croup had he not discovered sloughs upon the tonsils and uvula.

One argument in favour of the identity of membranous croup with diphtheria adduced by Sir William Jenner is that mucous membranes do not pour out lymph upon their surface when inflamed by ordinary irritants as serous membranes do; so that an affection of the larynx attended with the formation of false membranes must be a specific inflammation. But, both for the fauces and the air-passages, it is certain that this rule is far from being absolute. In the 'Guy's Hospital Reports' for 1877 (p. 384) seven cases were recorded by the author, in which those parts presented appearances indistinguishable from diphtheria as the result of scalds of the throat by boiling water, which the children had sucked from the spout of a teapot or kettle; one case of a boy, who got a bean into his right bronchus, and whose larynx and trachea were coated with lymph; two cases (already referred to at p. 263) of children whose fauces were irritated, one by a piece of hot potato lodging in the throat, the other by a burning stick, and in whom the morbid action took the same form; one case of a man aged twenty-four who was admitted for a cut throat, and who died of a plastic inflammation of the larynx, trachea, and bronchial tubes; three cases in adults in which membranous laryngitis was secondary to cancer of the pharynx, tubercular ulceration of the vocal cords, and syphilitic disease, for which tracheotomy had been performed; and, lastly, two cases, both in adults, in which a similar affection was associated with acute or a chronic pneumonia—sixteen in all. Many of the patients, indeed, had had tracheotomy performed some days before death, and it might be plausibly argued that a badly cleaned tube, if it had before been used for a case of diphtheria, might

* By an unfortunate confusion some writers have designated as "spasmodic croup" the entirely different complaint "*laryngismus stridulus,*" described above.

have inoculated the parts with the specific disease. This, however, would not apply to some of the above cases, nor to one recorded by Mr Parker ('Clin. Trans.,' 1875) of a child who had scalded its throat with hot water, and from whose trachea false membranes were drawn up by means of a feather almost immediately after the operation. Moreover, both Rietz and Oertel found it easy to set up a plastic inflammation of the trachea in dogs and rabbits by dropping a few minims of Liquor Ammoniæ into it through an external wound. Oertel performed this experiment on seventeen animals, and succeeded in every instance in producing artificial "croup"—*i. e.* a traumatic membranous laryngitis.

It was long believed that the false membranes in croup and in diphtheria presented constant differences, microscopical and chemical; but we now know that in diphtheria itself they vary in their appearance, in the relation which they bear to subjacent parts, and even in their histological characters, according to the part of the mucous tract upon which they are developed. This fact overthrows all the histological distinctions between the two diseases.

Oertel believes the presence of micrococci to be essential to diphtheria, but in the false membranes which he set up artificially in animals by dropping Liquor Ammoniæ into the trachea, he found micrococci, though in small numbers, and only in the more superficial layers. It is not certain that the micrococci which are generally present in diphtheritic membranes possess the importance which Oertel attributes to them (*supra*, p. 261); but their non-occurrence in croup might still be distinctive of that disease. As a matter of fact, however, other observers have failed in cases of diphtheria to detect the micrococci in false membranes below the glottis.

Another distinction between diphtheria and croup, on which some observers have laid great stress, seems to be capable of ready explanation. It is the clinical fact that marked symptoms of depression—a dry, brown tongue, sordes on the lips, petechiæ on the skin, hæmorrhages upon the internal serous surfaces—are present in the former, but absent in the latter disease. In many cases of diphtheria, however, no such symptoms show themselves until several days have elapsed; and since croup destroys life rapidly by the mere effects of the presence of false membranes in the air-passages, one could not expect it to be attended with signs of depression and of septicæmia, even if it is a modification of diphtheria.

Again, the fact that a definite exposure to cold has immediately preceded and apparently excited an attack of membranous laryngitis, seems to be no proof that the case is not one of diphtheria. At least Sir William Jenner says that he has seen cases which arose in this way, and which he believed to have been diphtheria, because albumen showed itself in the urine; and in the chapter on diphtheria (p. 262) Dr Yeat's observations are recorded (which seem to refer to faucial diphtheria occurring in adults) as to the frequency with which, during an epidemic, those persons were attacked who had immediately before been exposed to the night air. Was it diphtheria which (as Watson relates) seized Dr Gregory's twin children on the same night, after walking together in the evening during a cold wind?

On the whole, it appears manifest to the present writer that no criteria based either upon morbid anatomy or upon clinical symptoms avail to distinguish laryngeal diphtheria from croup; that is to say, that the case

in which diphtheria is limited to the air-passages are really undistinguishable from membranous laryngitis with symptoms of croup.*

Notwithstanding Dr Fagge's arguments in the 'Medico-Chir. Trans.' for 1879, and in the first edition of the present work, there is increasing agreement that the disease as we see it in London is one—faucial diphtheria or laryngeal diphtheria or both. Traumatic cases of membranous laryngitis may occur, and occasionally idiopathic cases of the same kind; but "croup" expresses a certain combination of symptoms, and though these may depend on membranous laryngitis, they may equally depend on acute laryngeal catarrh or spasm. In any case it is most undesirable to use "croup" as a pathological term.

(ii) *Croup and catarrhal laryngitis.*—The next question is, what relation to the croup of Home and Cheyne is borne by those cases of "croup"—not infrequent—in which the air-passages are certainly without false membranes? Bretonneau is supposed to have been the first who indicated clearly the points of difference, and his name for the cases in question was "stridulous angina." But the English writers of the end of the last century were well acquainted with the clinical history of non-membranous laryngitis, which was known to them as "spasmodic croup" or "spurious croup." Its peculiar characters are fully set forth in a paper which Mr Field, before the Medical Society of London, read in 1796.

The most distinctive features of this non-membranous croup are the suddenness of its onset and the alarming nature of the symptoms which it presents from the very commencement. A child who is apparently in perfect health, or who may have had a slight cold for a day or two, goes to bed without any sign of laryngeal affection, and falls asleep as quietly as usual. About eleven o'clock, or at midnight or a little later, he suddenly starts up in a state of extreme excitement and terror. He coughs incessantly, making a hard, hoarse, barking noise. He pants for breath, and each inspiration is attended with a loud crowing sound. His voice is hoarse and it may be very feeble, but it is not whispering as in "true (membranous) croup." His face, at first flushed, afterwards becomes pale and covered with a cold sweat. The parents are alarmed and the nearest

* Cases in which there is an affection of the fauces, such as would be commonly called diphtheria, are much less often traceable to contagion when the air-passages are affected than when they escape. There seems to be a regular descending scale of contagiousness, according as the morbid process falls with less intensity upon the tonsils and palate and with more intensity upon the larynx and trachea. Moreover, whereas diphtheria attacking the fauces is common in adults, nearly all the cases at Guy's Hospital in which these parts are but slightly affected, and the brunt of the disease has fallen upon the air-passages, have been in children below five years of age. Now, it is very improbable that such differences should exist, either as regards the contagiousness of the disease, or as regards its occurrence at a special period of life; but one sees at once that the recognition of these differences is exceedingly favourable towards the inclusion within the domain of diphtheria of cases in which the larynx and trachea are alone attacked, the tonsils and palate remaining free. For the latter cases are likewise peculiar to children, and their non-contagiousness is the very point on which the whole discussion turns.

Is it not preferable to adopt another solution of the difficulty which involves no such improbabilities? It is to assume that the greater number of the seven cases referred to at p. 882 were really not examples of diphtheria at all, although the fauces were affected. After all, it seems an absurdity to draw a fixed line at the edge of the epiglottis, and to say that so long as an inflammatory process is limited to the parts below it the case may be one of simple membranous croup, but that if it spreads above this line it must be due to the specific poison of diphtheria. No doubt the great difference in histological structure between the mucous membranes of the larynx and pharynx appears favourable to such a view. But we know that, whether in diphtheria or after a scald of the throat, no obstacle to the *descent* of an inflammatory process is offered by this difference in structure. Why then should it be a bar to the *ascent*, when the air-passages are first attacked?—C. H. F.

medical man is sent for in haste. But, instead of the child getting worse, each paroxysm of coughing is rather less severe than the preceding one; and after half an hour, or in two or three hours, he becomes calm and sleeps. In the morning, when he wakes up, his cough is still hoarse and barking, but it is not so hard; his respiration is attended with little or none of the whistling sound; his voice has nearly regained its natural tone. During the day the child is as cheerful as before and has but little cough; his pulse is not accelerated; he is scarcely, if at all, feverish. On the following evening the symptoms may return, though not often as severely as at first; and occasionally they may repeat themselves for several nights in succession with gradually diminished intensity.

It is doubtful whether attacks of this kind ever prove fatal. Trousseau speaks of having seen three cases in which death occurred. But the only one of which he gives details is that of a schoolboy, thirteen years old, who was suddenly seized with dyspnœa on walking in the morning, and who seems to have died at the end of about four hours. On *post-mortem* examination the laryngeal mucous membrane was found to be reddened and the arytæno-epiglottidean folds were a little swollen; the vocal cords were swollen, and on one of them "there was a slight membranous concretion, possessing, however, none of the characters of diphtheritic false membrane." Probably severe laryngitis would have developed if the patient had lived a little longer. It certainly was not a typical case of spurious croup as regards the hour at which the attack began, and the boy was much beyond the usual age.

At present, therefore, the pathology of spurious croup is a matter of inference only; but there can be little doubt that it depends upon catarrhal laryngitis, complicated with spasm of the glottis.

Another feature of this complaint is its tendency to recur again and again in the same individual. A child who has once had it is always likely to be attacked a second time if he is exposed to cold or wet; and up to the age of fourteen or fifteen every slight catarrhal affection is apt to be accompanied with the peculiar hard barking cough. When one hears that a person suffered from "croup" repeatedly during childhood, one may pretty safely conclude that the disease was "spurious," *i. e.* is not membranous laryngitis.

Cheyne, in the second edition of his work, discusses at considerable length the relation between the "Croup" of Home and this "spurious" or "spasmodic" affection, with the description of which he was familiar through the writings of Field and of Ferriar, a physician of Manchester, who had published an essay on the subject in 1810.* And he comes to the conclusion that there are no just grounds for admitting two kinds of croup. The affection in question "occurs," he says, "in those families which are subject to genuine croup; it arises from the same exciting causes (exposure to cold); it prevails during the same weather." He goes on to say that in many cases in which the breathing afterwards becomes permanently affected, the symptoms are for the first few days most marked during the early part of the night, the patient in the daytime seeming to have nothing the matter with him but a cough.

There are, however, two criteria which point very strongly to the

* After reading Ferriar's essay it seems to the writer probable that his cases of "true croup" would now be recognised as diphtheria, and that those of "spurious croup" would be called acute spasmodic laryngitis.

existence of "spurious" (catarrhal) as separate from "true (membranous) croup." One is the sudden onset of the complaint with all its symptoms in full force. The other is its liability to return again and again in the same patient. If spurious croup were merely an undeveloped or milder variety of membranous laryngitis, one would expect that those who are subject to it would be very apt, when the exciting cause happens to be powerfully in operation, to have it in its severe form. Yet it is a question whether a single instance has been recorded.

There can be no doubt that Cheyne included, in his description of croup, cases of "stridulous angina." Indeed, it would seem that every case which he details, as an example of spurious croup, recovered.

Trousseau says that although "stridulous laryngitis" (as he terms the affection) is very common, he had had only one case in his wards at the Hôtel Dieu, a principal reason for this being the sudden way in which it declares itself, and the rapidity with which it yields, so that children attacked by it are very seldom brought to hospitals. But in Guy's Hospital, between the years 1867 and 1876, there were admitted ten cases at least of croup (and probably several more) in which recovery took place, and in which there was no proof of the formation of false membranes. In many of these cases the symptoms were continuous for some days in succession; and the clinical reports very seldom say anything about previous attacks of a similar kind. Perhaps it may be that in London a form of non-membranous croup is of frequent occurrence which in Paris is not met with.*

* It is a striking fact that Dr George Johnson—who is anxious to draw a sharp line of distinction between the cases of membranous laryngitis (which he regards as examples of diphtheria) and those in which no such membranes are found (which he terms "infantile laryngitis" or "inflammatory croup")—is obliged to extend the definition of the latter affection beyond the narrow limits which had been set by the earlier English and by the French writers. In fact, in the last edition of Sir Thomas Watson's 'Lectures' (in which Dr Johnson's views are adopted) the whole description of "croup" is transferred bodily to the new "infantile laryngitis." This is no longer the harmless affection of Bretonneau and Trousseau, but a disease which "proves fatal sometimes within twenty-four hours and often within forty-eight hours," and which "may continue for five or six days before it terminates." Now, in the course of many years there appears to have been at Guy's Hospital only one case fatal, in which false membranes were not found after death. But, as already remarked, it often happens that there is no evidence of their presence during life.

It is an instructive fact that in two out of eleven cases of "idiopathic croup" which came under Dr West's care between 1839 and 1849, when diphtheria (at least in an epidemic form) was not prevailing in this country, there was "a scanty formation of false membrane upon the velum and tonsils." No doubt, in an individual case, the possibility that the disease may be diphtheria can never be absolutely negatived. The most important points against it would be the absence of a history of contagion, the circumstance that no other person in the house or in the neighbourhood had had anything that could possibly be set down as diphtheria, and (with due allowance for the facts stated at p. 263) there having been a direct exposure to weather or to some considerable change of temperature immediately before the commencement of the attack.

It must be admitted that belief of the frequent occurrence of true membranous croup in children, distinct from laryngeal diphtheria, is antagonistic not only to that of French and most modern English writers, but also to those of German pathologists on the subject. Even those who theoretically admit the existence of membranous laryngitis and tracheitis independent of diphtheria, in practice seem to assign to diphtheria almost all the cases that they see.

It is, however, an important question whether this may not depend upon the fact that on the Continent non-specific membranous croup is really a much more rare disease than in England. Cheyne makes the assertion that it is far less known in the temperate than in the northern regions of Europe; but one can hardly tell on what facts such a statement is based. Even in Great Britain the disease appears to be irregular in its distribution. Home states that in his time it was far more frequent in Leith and Middleburgh, which are near to the seashore, than in Edinburgh, and in that city Dr Alison found it most prevalent in those parts

Symptoms.—"Croup" as distinct from diphtheria and dependent on acute laryngeal catarrh with spasm, commonly begins with but slight severity. The child is noticed to be feverish, refusing its food, but asking frequently for water. Its voice is hoarse; it sneezes frequently; it may have rather a shrill cough. It complains of no pain in swallowing. After some hours, or not until the lapse of four or five days, symptoms characteristic of the disease first begin. Their onset is generally gradual, but it may be sudden and may come on in the night.

Of all these symptoms the most important is dyspnœa. The breathing is not only hurried but noisy. Each inspiration is attended with a peculiar whistling sound, which may be audible at a considerable distance; and a similar sound, or one of a more snoring character, may accompany the expiration. On uncovering the child's chest, one sees that the structures above the sternum and the clavicles, and the lower intercostal spaces, are all drawn inwards each time the child breathes. And as the disease advances, the epigastrium, the false ribs, and even the lower portion of the breast-bone itself, form part of a deep hollow, produced by the action of the diaphragm; this muscle being arched upwards into the chest by the atmospheric pressure, can only drag its attachments backwards towards the spinal column, where it contracts.

The cough, at first harsh and clanging, gradually grows husky and at length is inaudible. The voice, from being hoarse, becomes whispering, and is finally extinguished. When the child attempts to speak, its lips can be seen to move, but not the slightest sound is heard. The nostrils dilate with each effort to breathe. The head is thrown backwards as far as possible, and the spine is curved in the same direction. It is not very obvious why this peculiar attitude should be adopted, but the supposition is that the trachea is stretched, so that air can better pass by the side of the false membrane. Ferriar once saw the corpse of a child who had died of croup resting on its head and its heels, as if it had died of tetanus.

Besides its persistent difficulty of breathing, a child labouring under croup is also liable to frequent exacerbations. If lying or sitting in bed, the little patient starts up and throws itself into the arms of its mother or nurse; but the next instant begs to be put back into its crib. It clutches at anything within reach, or at its own throat, as if to tear away the obstacle to the free entrance of air. After a time it sinks back exhausted and may fall asleep. There is still a doubt whether such paroxysms depend upon muscular spasm, or upon the impaction of portions of false membrane or inspissated secretion in the chink of the glottis. Some observers think that the more constant dyspnœa is the mechanical result of a swollen

which are lowest in situation. Wet and marshy spots are said to be favourable to its occurrence. Some of the medical men practising in Norwich say that it is unknown in that city, which has a very dry and bracing air, although it is much exposed to cold easterly winds.—C. H. F.

An assailant of the position so ably defended by the author in the first edition of this work would need acquaintance with hospital and private practice, and with children's diseases in town and country, as well as in different climates, such as very few physicians possess. For a most valuable summary of facts and opinions on this important subject the reader is referred to the 'Report of a Committee appointed by the Royal Medical and Chirurgical Society,' ten years ago, "to investigate the relations of Membranous Croup and Diphtheria." There are numerous cases tabulated (64 by Dr Dickinson, 63 by Dr Gee, and 88 by Dr Fagge), and replies to a series of question put by the committee from a large number of physicians to London hospitals, as well as from others in the country and abroad (vol. lxii, 1879).

laryngeal mucous membrane, or of muco-purulent matter upon its surface, and refer the paroxysms to spasm of the laryngeal muscles.*

It seems likely that such extreme dyspnœa would be attended with albuminuria. But in the series of cases recorded by Mr Lamb in the 'Guy's Hospital Reports' for 1877, there was only one in which the symptom was detected, and here there were other grounds for believing the disease to be diphtheria.

Event.—When croup is to terminate favourably, the little patient's breathing becomes easier, its cough softer and more loose.

Remissions in the symptoms are not infrequent, especially in the morning hours. The child breathes better; his cough is less distressing; he partially regains his voice; he may ask for food, or get a little quiet sleep. But such a change sometimes ends in rapid exhaustion.

The approach of a fatal termination is usually indicated by symptoms of asphyxia. The cheeks and lips become pale and bluish; the forehead is covered with a clammy sweat; the child ceases to take notice, and lies with half-closed, drowsy eyes; the breathing becomes shallow; the pulse rapid and intermittent; and the extremities cold. Death is often preceded by convulsions.

Ætiology.—The chief exciting cause of croup is by all writers said to be exposure to cold. Dr Alison noticed that it was often produced by a child sleeping in a room newly washed, and consequently that in Edinburgh cases frequently occurred on Saturday night. Cheyne said that in all but three of the cases of croup which he saw there had been exposure to the weather.

It is stated that croup is more frequent in the winter than in the summer months, but this is doubtful. Perhaps, like acute pneumonia, it may attack those who are exposed to chill after a hot day, or during the cold weather which in our climate may occur at any season.

The *age* at which a child is most apt to be attacked with croup is between two and seven years. The complaint is rarely seen in infants at the breast; indeed, both Home and Cheyne thought that children weaned early were especially liable to it. In adults croup is unknown.

All writers agree in stating that boys are more often affected than girls, and this has been thought to distinguish cases of croup from diphtheria. Thus Sanné says that out of 1575 cases of "diphtheria" admitted into the wards of M. Barthez (where the number of beds for males and females is equal), 813 occurred in boys, 762 in girls. On the other hand, in 101 cases of "croup" occurring at Prague, Steiner found that seventy-seven occurred in males, twenty-four only in females. He defines croup as an inflammation of the air-passages attended with the formation of false membranes, and no doubt most of his cases were due to the contagion of diphtheria. The experience of a single institution like Guy's Hospital is too limited to afford a secure basis for a comparison of the numbers of the two sexes, but they show no considerable preponderance of males among the cases of diphtheria, while they do show it among cases regarded as non-membranous croup.†

* Niemeyer attributed them to paralysis of the same muscles, and says that after death their substance is found to be watery, pale, and soft. Of course, it is only during the act of inspiration that a whistling sound could be produced by paralysis; and, accordingly, he drew the distinction that when such a sound accompanies the expiration it is invariably caused by the presence of false membranes obstructing the glottis. This is very theoretical.

† This fact, taken with the difference in the frequency of the two forms at certain periods of the year, is undoubtedly an argument against the view which I have maintained as regards the connection between membranous and non-membranous croup.—C. H. F.

Diagnosis.—The principal points involved have already been mentioned in the discussion of the relations of croup to the "spurious" affection and to diphtheria. One must never forget that in reality none of the special symptoms do more than indicate the existence of laryngeal obstruction. The distinction between croup and other diseases of the larynx is based upon the acute character of the attack and upon the early age of the patient rather than upon anything in the symptoms themselves: but even children are liable to other affections attended with stridulous breathing.

A post-pharyngeal abscess, for example, sometimes gives rise to "croupy" dyspnœa and cough, although it is not obvious why this should be the case. One day when the author was visiting his patients at the Infirmary for Children the house surgeon reported that he had just been called to see a case of supposed croup, in which he had felt an abscess at the back of the fauces with his finger, and that relief was afforded as soon as the matter was let out. In a little child, eighteen months old, a patient of Dr Hudson, at Waltham Abbey, the respiration was rather of a snoring character than croupy, but there was a brassy cough. The abscess seemed to have commenced in a suppurative affection of the cervical glands, which had made its way inwards, instead of pointing externally. According to Steiner post-pharyngeal abscess occurs chiefly in infants at the breast, except when it depends on disease of the spine; it is more insidious in onset than croup.

Sir William Jenner speaks of abscess at the side of the larynx as another affection which may cause great distress in breathing by compressing the tube, and as not being always easy of recognition. The possible presence of a foreign body in the air-passages must never be forgotten. Laryngeal papillomata are too slow in their effects to be mistaken for croup.

The *prognosis* of croup depends on our definition of the word. That of membranous laryngitis or diphtheria is very grave indeed; that of acute catarrhal laryngitis with croupous symptoms is good, much better than for laryngismus stridulus in infants.

The *treatment* of "croup" also depends on our pathological diagnosis. That of "membranous croup" has been fully discussed in the chapter on diphtheria (p. 274).

Non-membranous croup or spasmodic laryngitis requires no active treatment; but it is usual to give an emetic of two to five grains of powdered ipecacuanha, with or without one sixth of a grain of tartarised antimony, repeated at intervals of ten minutes until free vomiting takes place. Jenner says that besides removing from the stomach any source of reflex irritation, and relaxing spasm by the nausea and faintness to which they give rise, these medicines also promote secretion from the laryngeal and bronchial mucous membrane, and so relieve the catarrh. He has observed that cases left to themselves last much longer, going on for two or three days, whereas, as soon as an emetic has acted, the child generally falls asleep at once. However, he follows it up with a dose of calomel and jalap. Another plan of treating croup is that recommended by Graves, of squeezing a sponge out of hot water—as hot as the hand can bear—and applying it beneath the chin, changing it as often as it gets cool, for ten or twenty minutes, until the skin becomes vividly reddened. One must always warn the relatives of a child who has had one attack of croup that it is likely to have others if it is exposed to cold or wet weather, and allowed to get chilled. Such children must therefore have special care taken of them, particularly as regards the warmth of their clothing; but they should be accustomed to

have the neck and chest sponged over every day with cold water, and Steiner suggests that they should gargle the throat with it several times a day. A child who has repeatedly suffered from this affection is sometimes left with a permanent hoarseness of voice.

Membranous laryngitis in the adult.—There is no question that diphtheria with membranous laryngitis is common to all ages, and that the disease to be next described, acute catarrhal laryngitis, is even more common in adults than in children. But whether or no children are liable to membranous inflammation of the larynx as an idiopathic sporadic disease apart from diphtheria, it is generally supposed that in adults the mucous lining of the larynx and trachea is incapable of producing a false membrane, except under the specific action of diphtheritic contagion. It is certain that fibrinous membranes are not produced in adults as they are in children, by traumatic inflammation—scalds of the throat, for instance; but, nevertheless, there are undoubted cases in proof that the adult larynx and trachea may become the seat of acute sporadic idiopathic inflammation, which leads, not to pus and catarrhal products, but to a fibrinous exudation and a false membrane.

The most conclusive one known to the writer is the following:—A pregnant woman was admitted under his care into Guy's Hospital, in December, 1879, with severe dyspnœa from laryngitis. She was taken ill with shivering, after exposure to cold, spat up membrane on the following day; and two days later a complete cast of the trachea with its bronchial ramifications was got rid of. She died after a week's illness, having previously miscarried; and there was found laryngitis, tracheitis, and bronchitis, with lobular bronchopneumonia and pleurisy. The pyrexia had been moderate; there was no hæmoptysis, no albuminuria, and, *post mortem*, all the other organs were sound. The fauces had been free throughout. Histologically the false membrane consisted of leucocytes and scanty fibres without blood-discs or epithelium ('Path. Trans.,' vol. xxxi, p. 30).

Laryngeal Catarrh with Aphonia.—From what has been said in the previous section it will be evident that this pathological term needs further clinical definition; for in cases of "spasmodic laryngitis" or "spurious croup" (or non-diphtheritic croup), catarrh of the larynx is probably present. But it is in clinical practice impossible to draw a broad line of distinction between cases of inflammation of the laryngeal mucous membrane attended with dangerous dyspnœa (whether from swelling or from spasm of the larynx, or both) and those of which the chief symptom is impairment of the voice. The latter are commonly known by the name of catarrhal laryngitis; and this, again, may be either acute or chronic.

1. *Acute aphonia* is a very common affection, but comparatively seldom comes under medical advice. Some persons are very subject to it; whenever they catch a cold they become hoarse or lose their voice, and this condition lasts for several days or even for weeks. It is particularly apt to occur in those who are intemperate and in those who habitually use the voice much, particularly in the open air; the reason being that in such persons the laryngeal mucous membrane is constantly more or less congested. It is common in those who are exposed to smoke and irritating gases, as firemen and workers in chemical factories. It may also arise as a complication of some acute disease, as enteric fever, but especially measles. The

chief symptom, beside the impairment of the voice, is expectoration of a little tough mucus, which comes away with a short hawking effort, hardly amounting to a cough. With the laryngoscope, according to Ziemssen, one may find that there is reddening and slight swelling of certain parts of the interior of the larynx, especially the hinder ends of the vocal cords, the interarytænoid space, or the false cords. During attempted phonation the cords may leave an oval space between them, exactly as though the internal tensors were paralysed. In somewhat more severe cases Störk speaks of the cords as looking red, dry, and lustreless, or even as being covered with yellowish-green crusts formed of dried-up exudation. They may also become ecchymosed, and sometimes their surface shows superficial excoriations.

The *prognosis* in acute catarrhal laryngitis is generally favourable. It must not be forgotten, however, that cases in which the early symptoms were those of mere catarrh sometimes run on into œdematous laryngitis; and, on the other hand, the affection, if neglected, may become chronic, and may then be exceedingly intractable.

In the *treatment* one of the most important points is that the patient should entirely abstain from using the voice. He should remain in a room of which the temperature is kept uniform. Steam inhalations should be frequently employed. Mackenzie advises the addition of tinct. benzoin. comp., or of succus conii (℥ij with gr. xx of sodæ carb. exsiccat.), or of lupulin (℥ss) to the hot water used for inhalation, the temperature of which should be from 140° to 160°. The patient should be encouraged to drink freely of demulcent liquids, such as barley water, linseed tea, and the decoction of cetraria or of althæa. Warm milk or *lac cum sevo* is a useful beverage. If there is great irritability of the larynx, evidenced by cough and tickling or pricking sensations in the throat, it should be kept in check by opium or morphia, or (according to Störk) by chloral or by lactucarium. A mustard-leaf applied over the larynx and trachea often gives immediate relief. Störk and others recommend, at the commencement of the disease, that a strong solution of nitrate of silver should be brushed over the cords, but Mackenzie thinks that this is better left undone.

The best way to overcome the liability to acute laryngeal catarrh, in those who are subject to frequent attacks of it, is to make the patient gradually accustom himself to daily sponging with cold water, and to exposing the throat in the open air without wraps, even in the winter. No one can avoid allowing a draught to play upon the neck from time to time indoors, or in a corridor, or in passing from house to carriage; and the more a person endeavours to keep the part protected the more surely will he suffer when the occasion arises. It is also important to live, especially during the night, in airy, cool, well-ventilated rooms. Warm, light clothing should be worn. A long stay by the sea-side in the autumn, or in the bracing air of Scotland or Switzerland, often does a great deal towards diminishing the susceptibility to catarrh in the following winter.

2. *Chronic laryngeal catarrh* often arises out of the acute affection, especially in persons who, in spite of hoarseness of voice, persist in attempting to continue duties requiring loud speech. Such cases are common in clergymen, schoolmasters, costermongers. Another frequent cause is extension downwards from a "granular pharyngitis." Störk believes that a liability to this form of chronic faucial irritation often passes by inheritance

from parent to child. He also thinks that it may be set up by a course of mercury or of iodine, or by inoculation of the nasal cavity with gonorrhœal or leucorrhœal discharge. As a rule, chronic catarrhal laryngitis occurs in middle life; it is more common in men than in women.

The principal *symptom* of this affection is hoarseness of voice, which may pass on to complete aphonia. The degree to which the voice is impaired may vary very much at different periods of the day. Mackenzie remarks that it is often greater when the patient first begins to talk after an interval of silence than it is a few minutes later, after he has gone on speaking for a time. The attempt to use the vocal cords often gives rise to a painful sense of fatigue, and there frequently are also complaints of a feeling of dryness or soreness in the throat, and of a tickling sensation leading to a constant desire to hawk or to cough. The expectoration is scanty, generally a viscid grey mucus, but sometimes yellow and puriform.

The laryngoscope shows all gradations of change, from a slight local injection and swelling of some part of the mucous membrane of the larynx, up to the most extensive and diffused redness of the whole interior of the organ. Mackenzie remarks that one vocal cord may be of a bright red colour while the other is white; he also says that the congestion may be limited to a small portion of one cord, this being always on the outer or attached side of it. Small pieces of mucus are often seen adhering to the mucous membrane at different points; in the form of whitish threads they may pass across from one cord to the other; in cases of long standing the whole surface of the larynx may be covered with secretion. During vocalisation the cords in many cases fail to meet one another. This may be due to swelling of the interarytænoid mucous membrane, which is sometimes so extensive as to form a convex projection even when the arytænoid cartilages are as far apart from one another as possible. But in addition to this a paretic state of the muscles is not seldom present. Ziemssen says that this is most frequently unilateral, in which case the opposite cord may pass across the middle line to meet the affected one.

Sometimes certain parts of the larynx are greatly thickened. Ziemssen says that this is especially apt to be the case with the epiglottis, the arytæno-epiglottidean folds, and the false cords. Lewin maintained that thickening of these folds is a particularly marked feature of "clergyman's sore-throat," but this is disputed by Mackenzie. Störk relates in detail a case in which the whole of the interior of the larynx was affected with an extreme degree of hypertrophy of its mucous membrane, so that there was great dyspnœa, rendering tracheotomy inevitable. The disease had been of fifteen years' duration. From the right false cord there grew a hard, solid, fibrous tumour, of the size of a hazel-nut; every part of the interior of the larynx was thrown into enormous folds and ridges. Polypoid excrescences are, indeed, not uncommon results of a chronic laryngeal catarrh. Another affection which has sometimes been observed under similar circumstances is a thickening of the mucous membrane below the glottis, reducing the channel for the passage of air to a narrow chink or ring; in almost every instance it has been necessary to open the trachea. Lastly, the vocal cords themselves sometimes become granular on the surface, a condition which has by Türck been designated "chorditis tuberosa" or "trachoma." It has been supposed to depend upon a partial dermoid change in the epithelium; but in one case Wedl found microscopically only connective tissue and nuclear overgrowth.

Enlargement of the mucous glands is not infrequent in chronic laryngeal catarrh. Mackenzie speaks of seeing their enlarged orifices upon the epiglottis and upon the posterior parts of the cords in some cases, either as pale specks on a congested surface, or as small red circles on a pale surface. Another morbid appearance, which Ziemssen regards as an accidental complication of catarrh, is a dilatation of the veins of the mucous membrane, especially upon the epiglottis or upon the cords.

There has been some difference of opinion among writers as to whether chronic catarrhal laryngitis is or is not apt to cause erosion or ulceration of the laryngeal mucous membrane. Störk says that such a result is not infrequent, and that when an ulcer forms over the *processus vocalis* it may give rise to expectoration of blood in sufficient quantity to suggest the presence of tubercular disease of the lungs.

This observer lays stress upon the occurrence of a vertical fissure in the interarytænoid mucous membrane. It is not peculiar to cases of chronic catarrh. Störk speaks of it as being extremely frequent, occurring in as many as 50 per cent. of those who attend his out-patient practice. The fissure, as the result of the traction upon its sides, assumes a rhombic form; so that the upper part of it, which is alone visible in the laryngeal mirror, appears triangular. Its detection is often very difficult; the patient must be placed in the position required for inspection of the trachea with the laryngeal mirror. The symptoms are not always very marked. Störk had seen a singer of reputation, whose voice remained perfect after such a fissure had existed for many years. Generally, however, there are symptoms which appear to be identical with those of chronic laryngeal catarrh. And sometimes the subjacent arytænoideus muscle becomes paralysed, in which case the patient's life may be made almost unendurable by the running down of fluid into the larynx whenever he attempts to swallow or even to lie down.

The course of chronic laryngeal catarrh is generally very tedious and protracted, one reason for this being the fact that patients will seldom carry out the necessary treatment with sufficient perseverance, imagining that they ought to be well in two or three weeks, and neglecting all precautions as soon as they begin to improve a little. There are, however, among schoolmasters and clergymen many who go to the opposite extreme, being so nervous and fidgety about their throats that they may almost be classed with hypochondriacs.

A good many cases recover perfectly under careful management. A very important point is that rest should be given to the voice, the patient either using a slate to express his wants, or at least speaking only in a whisper. He must also abstain from smoking, be very moderate in taking alcohol, and avoid all highly seasoned foods. Locally, if the larynx is irritable, the patient should make use of a spray containing bromide of potassium or carbonate of soda with a little morphia. But the chief therapeutic results are to be expected from the use of astringent sprays containing tannic acid (gr. j—v ad ℥j) or alum (gr. j—x ad ℥j), or from the systematic application of astringent solutions to the interior of the larynx by means of a brush. Mackenzie says that he generally employs a solution of chloride of zinc (gr. xv ad ℥j); he applies this daily during the first week, on alternate days during the second and third weeks, and afterwards less frequently. Ziemssen lays great stress on the value of the topical use of solid nitrate of silver fused upon the end of a

laryngeal probe; this he repeats at intervals of a week or a fortnight; it causes violent spasm, which, however, is at once relieved by filling the throat with cold water. When there is an interarytænoid mucous fissure, the application of solid caustic is the best treatment; this must, however, be done with great exactitude, for if the surrounding healthy mucous membrane is touched instead of the sore, the patient's sufferings will be aggravated instead of being relieved.

In many cases of chronic catarrh, electricity applied locally aids in restoring the voice; and some patients find benefit from a course of the waters at Aix-les-Bains or at Ems.

When great thickening of the squamous epithelium from long-standing laryngitis is present, the case becomes what Virchow described as *pachydermia laryngis*, in a paper read before the Medical Society of Berlin, July 27th, 1887. There is a flat diffused swelling of the membrane towards the back of the vocal cords.

TUBERCULOUS DISEASE OF THE LARYNX AND TRACHEA.—In the medical literature of the end of the last century is first mentioned a disease called *laryngeal phthisis*. This name is not a good one, because it suggests the idea that an affection of the larynx may give rise to wasting and to other symptoms like those of pulmonary phthisis, without there being any lesion of the lungs. That such is sometimes the case has, indeed, been asserted by Trousseau and by some other writers, and may appear to be clinically supported; but the experience of all pathologists is that when a tubercular affection of the larynx is found after death the lungs invariably contain tubercles also, and present other lesions of evidently long standing, even when there has been no evidence of disease during life. If laryngeal tubercle is seen in cases of acute general tuberculosis, old as well as recent disease of the lungs is always present; consequently, although it cannot be denied that tubercles might form in the larynx earlier than in any other part of the body, the pathological evidence points at present to the conclusion that primary tuberculous disease of the larynx does not exist.

Pathology.—The view that the laryngeal affection which accompanies pulmonary phthisis is dependent upon the formation of tubercles was originally advanced by Laennec. It was soon afterwards disputed by Louis, and since that time the fact has been repeatedly affirmed and as positively denied. There can be no doubt that one very seldom sees conspicuous grey or yellow tubercles in the larynx like those in the ileum. But the general belief of English pathologists has always been that the laryngeal affection is really tubercular, not merely because it occurs in a large proportion of cases of pulmonary phthisis, and often with tubercular ulceration of the intestine, but also because many laryngeal ulcers have thick and caseating edges. The question was practically settled by the careful investigations of Heinze, of Leipzig, who published a monograph on the disease in 1879. The basis of his work was a microscopical examination of the tissues in fifty cases of phthisis, in each of which there was disease of the larynx or trachea; for in many instances the morbid process involves not merely the upper but also the lower air-passages, sometimes as far as the bronchial tubes.

Heinze found that in forty among his fifty cases tubercles were plainly recognisable in the larynx; in thirty-nine of these forty there was ulceration, in one there was tubercular infiltration of the mucous mem-

brane without ulceration; in eleven of the forty there was likewise tubercular ulceration in the trachea: in eight the trachea contained ulcers which could not be shown to be tubercular. With regard to the remaining ten cases, in eight there were laryngeal ulcers not certainly tubercular, but in five of these the trachea showed tubercular ulcers, and in two there were no ulcers in the larynx, but tubercular ulcers in the trachea. In other words, there were only three out of the whole fifty cases in which tubercles were not detected either in the larynx or in the trachea, or in both. The tubercles were plainly visible to the naked eye in hardened sections. They lay partly in the mucous membrane, partly in the submucous tissue, but always on a plane superficial to the laryngeal mucous glands. They had often undergone more or less complete caseation. With regard to the non-tubercular ulcers of the larynx and trachea, Heinze states that there was nothing in their external appearance to distinguish them from those that were tubercular. In every instance, too, they were very superficial—in fact, little more than erosions, and they were generally confined to the vocal cords. It is, therefore, quite open to question whether they were not originally preceded by a formation of tubercles which had softened and been cast off by ulceration, as is believed by Virchow to be very frequently the case when a case of laryngeal phthisis fails to show a definitely tuberculous character. In the trachea, on the other hand, there seems to be no doubt that ulcers occur which are really non-tubercular; they appear as minute depressions of yellow colour surrounding the mouths of the tracheal glands.

In one point only Heinze goes too far; this is when he asserts that, from the exceedingly small size of laryngeal tubercles, those writers must be in error who in some exceptional instances have recognised tubercles in the larynx in making autopsies, or even by the laryngoscope during life. Türck, for example, figures a larynx from a dead subject, in which, besides tubercular ulcers, there was what he describes as miliary tuberculosis plainly visible to the naked eye. And tubercles, both grey and caseating, have several times been seen at Guy's Hospital in the laryngeal tissues. Considering how tubercles vary in size in other organs, those of the larynx may well be much larger in some instances than in others.

Koch's tubercle bacillus is sometimes found in the granulations of a laryngeal ulcer, but not abundantly; and great care must be taken that, if the secretion covering it contains them, they are not pulmonary in origin.

The fact that tuberculosis of the larynx never occurs independently of pulmonary phthisis, and that probably it never precedes the latter, naturally suggests that the upper air-passages become infected by the passage through them of tuberculous sputum. This notion was started by Louis, but he thought that the sputum acted merely as an irritant upon the parts with which it came into contact. At present one can form a far more definite conception of the way in which tubercle bacilli, settling upon the laryngeal or upon the tracheal mucous membrane, may germinate and invade the tissues.

Ziemssen maintains that a continuous tract of ulceration can sometimes be followed from a vomica in the upper lobe of one lung along the corresponding bronchial tube (which alone of all the bronchial tubes may be affected) through the trachea to the larynx.

Sex and age.—That tubercular disease of the larynx and trachea should be more frequent in men than in women might have been anticipated from

the fact that this is the case with pulmonary phthisis, but the preponderance of males over females is far greater; according to Heinze 33·6 per cent. of male phthisical patients have ulceration of the larynx, but only 21·6 per cent. of female phthisical patients.

The *age* at which tubercular laryngeal affections are absolutely most frequent is between twenty-one and thirty; but among fatal cases of phthisis the proportion in which the larynx is found diseased is relatively larger at a more advanced age, namely, between forty-one and fifty for males, between thirty-one and forty for females. During childhood tubercular disease of the larynx is not common; among nearly 400 cases Heinze found only nine in patients under the age of fourteen; in none of these was there ulceration of the trachea: one was an infant of eleven months.

Symptoms.—When the upper orifice of the larynx is affected, there is often extreme dysphagia, every attempt to swallow causing great pain and distress. The voice becomes hoarse and weak, and ultimately it is reduced to a whisper. In some cases, however, in which the true cords are entirely destroyed by ulceration, it is believed that the false cords may vibrate so as to produce harsh deep tones. The effort to speak is often painful. The cough becomes weak and hoarse and toneless.

Pain, cough, and aphonia are, however, common to other forms of common laryngitis, and it must not be supposed that marked symptoms of laryngitis occur in every case of phthisis in which the larynx is found after death to be affected with tubercular ulceration. A few isolated ulcers are often found when no laryngeal affection had been suspected during life; and experience in the deadhouse teaches that extensive superficial catarrh of the mucous membrane possesses far more constant clinical significance than does the presence of localised ulcers, however deep. On the other hand, there are many cases of phthisis attended with hoarseness or aphonia, in which no tubercle in the larynx can be detected, whether during life or in the dead body.*

Diagnosis.—In the laryngeal mirror what chiefly characterises cases of phthisis attended with impairment of voice, when there is no local tubercular disease, is the extremely *anæmic* state of the mucous membrane of the larynx.

Heinze lays great stress upon the recognition of *tubercular infiltration* of the mucous membranes, which (he says) is quite peculiar to this disease, and which was present in twenty-one of his forty cases. Its most frequent seats were the false cords and the arytæno-epiglottidean folds. He speaks of it as forming in the dead body a smooth tense swelling, of a greyish-white or greyish-yellow colour, often presenting on its surface the appearance of fine pale yellow granules, or spotted with points of hæmorrhage. Over the summits of the arytænoid cartilages tubercular infiltration gives rise to swellings which Heinze compares to two rounded sugar-loaves, and Mackenzie describes as "pyriform." The epiglottis, when it is affected,

* In 'Virchow's Archiv' for 1877, Fränkel, of Hamburg, endeavoured to find an explanation of such cases in the occurrence of lesions in the laryngeal muscles, the fibres of which he showed to have undergone a granular change ending in complete absorption of their substance and emptying of the sarcolemma, while at the same time the nuclei of the internal perimysium underwent increase. It does not appear that the patients who furnished the material for his observations had had any definite paralysis, and indeed the lesions which he detected were distributed equally over all the muscles. But paretic states of the thyro-arytænoideus internus are said to be not uncommonly present in phthisis, and Gerhardt has described paralysis of the right recurrent nerve as the result of its having become involved in thickened pleura covering the apex of the right lung.

appears rounded and thickened, or "turban-shaped." The false cords become greatly swollen, so that they lie in the same vertical plane with the true cords or even overhang them, while the entrances into the ventricles of Morgagni are greatly narrowed. Thickening of the interarytænoid mucous membrane gives rise to a local bulging or excrescence at the back of the glottis, to which Störk draws attention as characteristic of tubercular disease, it being all the more so because neither polypi nor other new growths are ever seen in that position. The cords themselves very rarely exhibit a true tubercular infiltration, but they may become swollen and rounded, a change which Heinze found to be dependent upon the presence of numbers of small round cells between their fibres.

In the trachea it is only in the posterior membranous part that tubercular infiltration occurs.

Tubercular *ulcers* have no distinguishing characters except their localisation, which is usually at the attachment of the vocal cords to the arytænoid cartilages. They are rarely either deep or hæmorrhagic.

Among Heinze's fifty cases, ulceration of the cords was present in no fewer than forty; on both sides in twenty-seven, on one or the other side separately in ten, at the anterior commissure in three; in eleven cases one or both of the cords were completely destroyed. In the laryngeal mirror an ulcer upon one of the cords may appear either as a narrow linear fissure, or as an excavation of its edge, situated upon a more or less reddened surface. When the process of ulceration is further advanced it often happens that the cord looks as if it were split up into two or three separate longitudinal bands, with very irregular edges, arranged one above the other like a short flight of steps.

The false cords are not so often ulcerated; of Heinze's cases only in fourteen, but in eight of these on both sides.

Over the arytænoid cartilages ulcers were present in twenty-three of Heinze's cases, sometimes towards the bases of the cartilages, sometimes upon their summits or towards the pharyngeal surface. Those which lie towards the bases of the cartilages are almost always bilateral. They are seldom, if ever, visible in the laryngeal mirror. They have a peculiar tendency to penetrate deeply into the tissues, so that they often reach the perichondrium and lead to necrosis of the cartilages. In the dead body their outline is seen to be irregular, their edges smooth or fringed with papillary outgrowths, their surface uneven and dirty grey.

The epiglottis was ulcerated in twenty-six of Heinze's cases, generally upon its laryngeal surface, sometimes upon its border, never on its lingual surface. Often there are great numbers of small round shallow ulcers; sometimes they run together into a large irregular excavation. The surface upon which they lie is often but little reddened. In some cases the substance of the epiglottis becomes eaten away from its margins inwards, so that it may present one or more deep fissures, with pointed processes between; or it may be reduced to a mere stump. In these cases the ulceration of course affects the lingual surface as well as the laryngeal. Indeed, the whole larynx may be found covered with ulcers varying in shape, in size, and in depth.

It must not be imagined that the detection of ulcers by means of the laryngeal mirror is always an easy matter, even when they come fully within the field of observation. Störk remarks that they are often recognised, not so much by the presence of an obvious depression in the mucous membrane,

as by a change in its colour and a deficiency in its natural lustre. Unskilled observers often take patches of puriform mucus for ulcers.

When the presence of ulceration of the larynx is established, it still remains to be considered whether it is tubercular. We have seen that in catarrhal inflammation ulcers are seldom found; so that the diagnosis generally lies between "laryngeal phthisis" and syphilis; the points of distinction will be discussed under the latter head (p. 900).

It must not, however, be supposed that, in the *post-mortem* room at any rate, ulceration of the larynx is of rare occurrence, apart from either tubercle or syphilis. In acute *pneumonia* ulcers over the arytænoid cartilages are not infrequent; and ulceration has been seen there, or upon the vocal cords, in two cases at Guy's Hospital in which there was double pleurisy with pericarditis, in two cases of erysipelas, in one case (probably pyæmic) in which there were abscesses both in the liver and in the brain, and in two cases of Bright's disease. One of these last-mentioned cases occurred in a man, aged twenty-four, who had been hoarse for three months before his death; all that was observed by the laryngoscope was that his cords were at one time reddened, but afterwards pale; at the autopsy a narrow linear ulcer extended over each cord for a quarter of its length. Heinze gives details as to eight cases in which ulcers were found in the larynx after death from various diseases.

Prognosis and treatment.—The author has met with three cases of phthisis in which the larynx after death presented what appeared to be the cicatrices of ulcers that had healed; and both Ziemssen and Heinze have recorded similar instances, some of which occurred in patients who had been repeatedly examined with the laryngoscope. One of Heinze's cases is that of a man whose right cord was much ulcerated; two or three years later this was found to have healed, and there were also cicatricial bands on the left cord, which must have become affected in the interval. Both this patient and another appeared to owe their recovery to residence in a southern climate.

At the meeting of the International Congress in London in 1881 Rossbach and some other observers spoke very positively with regard to the occasional "curability of laryngeal phthisis," while Krishaber virtually denied it. The former speaker attributed considerable value, at an early stage of the disease, to the inhalation of antiseptic agents; the latter declared that no such treatment was of the slightest use. Ziemssen lays stress upon the importance of giving absolute rest to the voice by maintaining perfect silence, or speaking only in a whisper, for months together. Mackenzie speaks of the local application of perchloride of iron, in strong solution, as sometimes greatly diminishing the irritability of the mucous membrane and so relieving the troublesome cough. Nothing, however, is so serviceable in this disease as the local *insufflation of morphia* as a powder, in a dose of $\frac{1}{8}$—$\frac{1}{2}$ a grain mixed with half a grain of starch. A special instrument called an "insufflator" is made for the purpose: it consists of a hollow vulcanite tube, one end of which is bent downwards so that it can be directed towards the orifice of the larynx; in the middle of the tube is a hole, through which the powder is introduced, and which is then closed by a moveable covering; the other end of the tube is connected with a piece of elastic tubing. In using this instrument the operator places the elastic tubing in his own mouth, and introduces the vulcanite tube into the mouth of the patient. He then blows the powder down into the

larynx, just at the moment when the patient is drawing his breath. Mackenzie says that as the greatest diminution of the sensitiveness of the affected parts occurs in rather less than an hour, it is well, when there is much pain in swallowing, to introduce the morphia at about that interval before the time of taking food. It may be repeated twice daily. If distress is caused by liquids entering the air-passages, all fluid nourishment should be thickened with arrowroot or corn-flour; and the patient is more likely to swallow well in taking off a good draught than in sipping. Sometimes it is necessary to administer food through an œsophageal tube for weeks together. Dr Wolfenden recommends the patient, in extreme cases, to drink only when lying in the prone position, or supporting himself on his hands and knees. *Cocaine* lozenges, taken before food, are of the utmost value in these cases, and proved so particularly in a severe one lately in Guy's Hospital, when all other means failed.* Tracheotomy is now and then required when there is extreme dyspnœa; but this operation seems in no way to retard the further progress of the laryngeal affection. In fact, all ordinary treatment of laryngeal phthisis must be regarded as only palliative. To this statement, however, exception ought perhaps to be made if severe and active cauterising agents are applied at an early stage. The actual cautery is sometimes used, but the application found most useful appears to be a 50 per cent. solution of lactic acid. This treatment was introduced by Dr Heryng, of Warsaw, and has been adopted by Krause and other specialists in Germany and England. Mr Symonds reports well of its results, especially if the lactic acid is applied after the tuberculous ulcer has been scraped with a curette.†

The duration of life in cases of tubercular disease of the larynx is seldom long. Mackenzie gives a list of 100 cases, of which seventy-nine ended fatally in from six months to two and a half years after "throat symptoms had become troublesome;" in nine only did death occur within six months; twelve were prolonged over a period of from thirty to forty-nine months. Ziemssen, however, maintains that the quickly fatal course of "laryngeal phthisis" depends rather upon the disease of the lungs developing itself rapidly than upon the affection of the larynx directly shortening the patient's life. According to this writer, when the pulmonary disease assumes a chronic form, laryngeal ulcers may exist for years.

LUPUS OF THE LARYNX.—Within the last few years a small number of cases have been recorded, in which lupus of the skin has been associated with a like disease of the epiglottis and the larynx. The affection is characterised by the presence of nodules, which may be as large as peas, and of ulcers, some of which have undergone partial cicatrisation. It is stated that the ulcerated epiglottis often looks as though a heart-shaped piece were taken out of the middle of its free edge. Otherwise there is nothing in the laryngoscopic appearances which could distinguish lupus of the larynx from the effects of syphilis; nor are there any peculiarities in the symptoms, which consist of hoarseness, sore-throat, dysphagia, and perhaps dyspnœa. Consequently the diagnosis of a case which Ziemssen gives as one of laryn-

* Mr Symonds remarks that a 10 per cent. solution of menthol in olive oil is a very soothing application, and more permanent in its effect than cocaine.

† Dr Clifford Beale has also written with judicious reservations, but decidedly in its favour, in an excellent paper on "Laryngeal Affections in Phthisical Persons" ('Birmingham Medical Review,' April, 1890).

geal lupus, in a girl of twelve, whose skin was free, depends chiefly upon the fact that treatment with iodide of potassium proved a failure. What is recommended for this disease is the systematic administration of cod-liver oil and energetic cauterisation with nitrate of silver. Scraping with a curette and the electric cautery have also been employed.

A remarkable case of fatal chronic laryngitis which was regarded by the writer as lupus occurred in a young man who was long under his care in Philip Ward in 1887. There was no lupus of the skin, and no signs or history of syphilis, but the epiglottis was seen to be ulcerated, and there was no appearance of tubercular ulceration of the cords, nor of phthisis. He was therefore put under iodide of potassium and mercurial inunction, but with no good result. Emaciation gradually came on, and he died after about a year's illness. Deglutition had been only possible for some time by the local use of cucaine, and the immediate cause of death was gangrenous pneumonia from particles of food gaining entrance to the air-passages. There was no proof of syphilis discovered after death, and only a trace of obsolete phthisis at the apex of one lung. The epiglottis was entirely destroyed; the cords were much less affected. The appearance of the larynx was quite unlike that of tubercular disease (museum, No. 1697[1]).

Mr Symonds reports only two cases from the Throat Department at Guy's Hospital. In both there was great thickening of the epiglottis, with but little destructive ulceration.

LARYNGEAL SYPHILIS.—Affections of the larynx resulting from syphilis are by no means rare, whether compared with other effects of the venereal poison or with other diseases of the larynx: their occurrence is to some extent determined by local irritation. They are frequent in persons whose throats are exposed to cold, and who abuse the voice, as, for example, in costermongers. And Mackenzie found that, contrary to what is believed to be the case with tubercular laryngitis, the larynx was far more apt to suffer from syphilis during the winter than during the summer. The commonest age is, as might be expected, between twenty and forty; but among those who suffer from laryngeal affections, as remote sequelæ of syphilis, it is not uncommon to find persons up to the age of fifty or sixty, or even seventy.

Some of the earlier laryngeal manifestations of acquired syphilis may occur within two or three months after infection; its more remote effects may appear five, ten, twenty, or even thirty years afterwards, when all other indications of the disease have long disappeared, and when the patient has almost forgotten it. However, perusal of some twenty cases that have at different times come under observation in the *post-mortem* room at Guy's Hospital leaves a decided impression that severe lesions of the larynx are much more often associated with cutaneous eruptions and other obvious signs of lues than are syphilitic affections of the viscera, as the brain or the liver.

In some cases, especially those of recent infection, the larynx shows nothing more than a superficial catarrh. In this there is nothing characteristic. Ziemssen warns his readers against supposing that a livid-red or dirty-brown injection of the mucous membrane justifies a diagnosis of syphilis. Nor does the protracted duration or obstinate recurrence of a laryngeal catarrh afford grounds for concluding that it is not simply inflam-

matory, although in the case of the pharynx such facts have sometimes a marked significance. Störk speaks of syphilitic catarrh of the larynx as so transitory that patients are seldom treated for it.

Next in order of development come *mucous patches*, or "flat condylomata" (p. 287). As to their frequency, widely different statements have been made. Some almost deny that they occur in the larynx; others say that they are often to be seen by the laryngoscope. Ziemssen says that their chief seats are the cords, the posterior laryngeal wall, and the false cords. They may also appear on either surface of the epiglottis. According to Mackenzie they differ from pharyngeal condylomata in being yellow rather than white, and in being generally accompanied by less marked congestion of the surrounding mucous membrane; he also says that they are less apt to undergo superficial ulceration, and that they generally disappear quickly, even without treatment.

At a later period of the disease *gummata* are of not infrequent occurrence in the laryngeal mucous membrane and submucous tissue. They are described as generally forming small rounded elevations, from the size of a pin's head to that of a small pea, of the same colour with the rest of the laryngeal surface, isolated or collected together into masses of considerable size. They may be seated upon the epiglottis, the posterior wall of the larynx, the false cords, or the surface below the glottis. In 1874 Mr Norton showed to the Pathological Society a gumma larger than a pigeon's egg, which occupied the right arytæno-epiglottidean fold, and reduced the air-passage to a mere chink, so that it caused suffocation. Laryngeal gummata often ulcerate, but sometimes they long remain stationary.

Cicatrices frequently form within the larynx, and produce an extraordinary amount of deformity. Sometimes a web is formed between the cords, as in several cases recorded by Elsberg, of New York. In other cases the epiglottis is dragged down and fixed to the side of the pharynx, or the parts forming the entrance of the larynx may be puckered together, so as to reduce it to a small round hole. Papillary outgrowths of considerable size are occasionally developed in the neighbourhood of syphilitic cicatrices, and increase the obstruction to the passage of air.

Syphilitic *ulcers* in the larynx are often observed. During the earlier stages of the disease they are generally superficial; afterwards they are apt to eat deeply into the tissues, and cause great destruction.

It has been much discussed whether syphilitic ulcers present any characters by which they can be distinguished. Türck maintained that some of these cases can be recognised at the first glance by their more or less circular form, by their excavated surface coated with a whitish-yellow material, by their edges, which are sharp, sometimes much raised, and surrounded by an inflammatory areola. A point of great importance is the comparatively rapid development of syphilitic ulcers. Störk remarks that a patient with extensive destruction of the epiglottis as the result of syphilis may still retain a fresh, healthy appearance, which is never the case where such disease is of tubercular origin. Tubercular ulcers are smaller than syphilitic, except when several have coalesced together; they are often numerous, affecting both sides of the larynx at once, whereas syphilitic ulcers are usually solitary; they are generally seated upon a surface which is pale and anæmic instead of being reddened. But ulcers of the larynx sometimes occur in persons who, having had syphilis, are also affected with phthisis; it may then be quite impossible to make a positive decision.

In some difficult cases great help is afforded by the presence of ulceration of the pharynx, or of the base of the tongue, which in tubercular disease of the larynx is very infrequent. Destructive ulceration of the larynx by syphilis is often preceded by a like affection of the fauces, which passes continuously from the pharynx to the epiglottis. At the seat of the earlier lesions cicatrices may often be observed to have already developed, and this is conclusive as to the syphilitic character of the affection, for in tubercular disease such partial cicatrisation is never seen.

A practical point in diagnosis is that while tubercular laryngitis makes the parts very sensitive, so that examination with the mirror is sometimes impossible without the local application of morphia in mucilage or of cocaine, the syphilitic larynx is, like the tertiary ulcer of the skin, singularly insensitive, so that such cases are the best for the beginner to practise on with the laryngoscope.

The *symptoms* of syphilitic disease of the larynx generally include hoarseness of voice, which may pass on to complete aphonia. Pain is often entirely absent; but if the epiglottis or some other structure at the entrance of the larynx is affected, swallowing may be exquisitely painful, although even in that case there may be an entire freedom from pain at all other times. It is wonderful how well some patients manage to swallow, even when they have lost a large part of the epiglottis; the base of the tongue is carried backwards, and keeps even fluid from passing into the interior of the larynx. Cough is often troublesome, and if there is extensive ulceration, muco-purulent exudation and blood may be expectorated in considerable quantity. In one case at Guy's Hospital hæmoptysis occurred to such an extent that the patient was thought to have phthisis; and Türck has recorded an instance of fatal hæmorrhage from an extensive ulcer of the left side of the interior of the larynx, exposing a necrosed piece of the cricoid cartilage. This is very rare in cases of tubercular ulcers of the larynx. Otherwise it is chiefly by the supervention of œdematous laryngitis, or perichondritis of the larger cartilages, that syphilitic disease of the larynx tends directly to destroy life. But Türck and Ziemssen believe that, in the case of the arytænoid cartilages, incurable necrosis is far less apt to follow deep ulceration when syphilitic than when it is tubercular. In some cases at Guy's Hospital a fatal termination has resulted from pneumonia, which was probably set up by the entrance of purulent matter from the affected parts into the bronchial tubes.

The *treatment* of syphilitic disease of the larynx should generally consist mainly in the administration of mercury, and inunction is perhaps to be preferred to other methods, as it is often important to produce an effect quickly. Spray inhalations with a weak solution of bichloride of mercury are said to be sometimes very serviceable. Sometimes it is advisable to brush over the affected parts with dilute tincture of iodine, or with a mixture of two parts of iodine, two of iodide of potassium, and ten of glycerine. When mercury has already been freely used, full doses of iodide of potassium should be prescribed internally. Tracheotomy is sometimes necessary, and cicatrices may have to be cut through with endolaryngeal instruments. Mechanical dilatation by means of tubes, without cutting, has been lately much employed, and with excellent results, as in a recent case of Mr Symonds', in a boy at Guy's Hospital.

Congenital syphilitic laryngitis is not uncommon, but is usually of only symptomatic importance, by causing aphonia. Cases, however, have occurred

of deep and severe ulceration leading to death. Two in brothers, aged between six and three, are recorded by Dr Semon in the 'Pathological Transactions' for 1880, and a third in a girl eleven months old by Dr Thomas Barlow, in the same volume.

Fränkel has recorded an instance in which a syphilitic infant, less than three months old, died from laryngeal stenosis, as the result of perichondritis with exfoliation of the cricoid and of the left arytænoid cartilages.

INFLAMMATION OF THE DEEPER LARYNGEAL STRUCTURES.—The inflammatory affections of the deeper laryngeal structures fall into two groups, those of the submucous tissue and those of the cartilages. The former may be described under the name of "Œdematous" or "Phlegmonous Laryngitis," the latter under that of "Laryngeal Perichondritis."

Œdematous or phlegmonous laryngitis.—This is the affection which is commonly but inaccurately termed *œdema glottidis*, the name having been originally given to it early in the present century by Bayle. The inaccuracy is twofold: on the one hand, the part diseased is not the *glottis*, or space bounded by the vocal cords, for (except in very rare instances) they remain free from swelling, which really affects the entrance of the larynx above; on the other hand, the morbid process is not a passive *œdema*, but inflammatory exudation of serum loaded with leucocytes into the submucous tissue, or diffuse purulent infiltration, or the formation of an abscess. Consequently there are some cases to which the designation œdematous laryngitis, others to which that of phlegmonous laryngitis, is more applicable. Perhaps a better name than either would be "submucous laryngitis," for the exudation is beneath the mucosa.

Œdema of the larynx in the sense of non-inflammatory dropsy is frequently seen in the bodies of those who have died of Bright's disease, or of heart disease, when the epiglottidean folds forming watery swellings are often of considerable size; such cases do not appear to present special symptoms during life, and probably have no clinical significance.

On the other hand, true œdematous laryngitis is one of the most rapidly fatal of diseases. The appearances, whether at the bedside or in the *post-mortem* room, vary somewhat with the exact locality of the inflammation. When the epiglottis is involved, it forms a turgid round mass, perhaps as large as the end of one's thumb, and often consists of two lateral rounded halves pressed closely together, so as to leave only a narrow gap between them. This may be either felt by the finger passed into the fauces or seen in the laryngeal mirror; occasionally it may be directly visible when the tongue is depressed with a spatula. Sometimes the tissues in front of the epiglottis are included in the swelling. In other cases the parts most affected are the epiglottidean folds, which may be converted into two large globular masses, tense and resisting, so that they feel like swollen tonsils. The mucous membrane covering the cornicula laryngis and that between the arytænoid cartilages may also share in the swelling, so that the movements of the cartilages are greatly impeded. Within the larynx the inflammation usually affects the false cords, which bulge downwards as well as inwards, so as to overhang and conceal the true cords. The latter are very seldom involved in the swelling, but Risch has recorded ('Berl. klin. Wochenschrift,' 1866) a case in which, having actually removed the larynx

within ten minutes of the patient's death, he found the true cords swollen to the breadth of half a centimetre and pressed against one another, so as completely to close the glottis. A similar instance occurred at Guy's Hospital in 1873; the patient was a woman who was found moaning on the ground in the street, and who died before she could be brought into the ward. In some cases the effusion is limited to the structures below the cords, constituting what Gibb termed a "subglottic œdema." Mackenzie speaks of such cases as generally chronic rather than acute; but Ziemssen cites five instances, one of them observed by himself, and all verified by the laryngoscope, in which the symptoms were urgent and rapidly developed.

The colour of the affected parts, as seen during life, is generally a bright red. After death they look much paler, the arytæno-epiglottidean folds in particular appearing gelatinous, and having often a yellowish-green colour from infiltration of pus into their tissue. When they are incised in the *post-mortem* room, however, it is often found that no fluid escapes from them, even under pressure. Sometimes the inflammation extends to the laryngeal muscles, which may be full of suppurating points.

Among the *symptoms* of œdematous laryngitis the most important is dyspnœa, which may increase with extraordinary rapidity until it destroys life by suffocation. Inspiration is commonly attended with a loud whistling sound. There is some pain in the throat, increased by speaking, and the larynx is tender when handled in the neck. The voice is not always much altered, but as a rule it becomes first hoarse and then extinct. The cough is hollow or sometimes toneless. If the voice, as is sometimes the case, becomes affected before the breathing, the explanation seems to be that the interarytænoid mucous membrane has been the first to become swollen. Another sign that this part is attacked is, according to Störk, an incessant dry jerking cough. If the epiglottis is inflamed at the beginning, there is intense pain in swallowing, and in all cases much distress is occasioned by the accumulation of buccal and pharyngeal secretions.

Störk lays great stress upon the significance of dysphagia as the earliest indication of commencing laryngitis in some cases, and upon the importance of using the laryngoscope whenever there is difficulty of swallowing, not obviously accounted for by an affection of the fauces. A thorough examination with the finger often at once clears up all doubt as to the nature of the disease. But this must be done with caution, lest it set up an attack of suffocative dyspnœa.

Ætiology.—Submucous laryngitis occurs chiefly in young adults between the ages of eighteen and thirty-five, and in men more often than in women. It is rarely seen in children.

As a primary affection, it appears to be generally due to some septic influence, such as is commonly but vaguely described as "blood-poisoning." Mackenzie says he has met with it in hospital physicians, in medical students, in nurses, as well as in persons exposed to emanations from bad drains. Sometimes it appears to be directly dependent upon exposure to cold, as in Trousseau's case of a man who, having drunk too freely at a wine-shop, was turned out into the street on a cold night, and fell asleep there, to wake with a violent sore-throat, which in an hour or two produced the most extreme dyspnœa. Sometimes it arises by direct extension from facial erysipelas, and sometimes it occurs in the course of smallpox, or of enteric fever. It is frequently the immediate cause of death in *angina Ludovici*, a diffuse inflammation of the connective tissue of the neck, with brawny

infiltration, which may or may not pass on to suppuration. In a case that occurred at Guy's Hospital in 1863 it was secondary to chronic suppuration in the fibrous tissues about the hyoid bone. It is often developed by extension from perichondritis of the laryngeal cartilages, or follows chronic tubercular laryngitis. Other frequent causes of submucous laryngitis are scalds of the throat, the entrance of a foreign body into the larynx, and the swallowing of mineral acids or other corrosive poisons.

Some writers describe it as being occasionally the immediate cause of death in cases of Bright's disease. Fauvel, indeed, maintained that it may be the earliest symptom of that disease. A recent case at Guy's Hospital in which acute inflammation of the submucous tissue of the larynx occurred as a secondary complication, was that of a man, aged twenty-seven, who died in 1878 of epileptiform convulsions. He had complained of sore-throat and of shortness of breath, but no symptoms had been observed indicative of laryngeal mischief. Both epiglottidean folds were found infiltrated with pus, but especially the left one. There was also much exudation of puriform lymph round the pharynx and at the base of the tongue. In the following year a man, aged thirty-four, who was lying in the hospital with cirrhosis of the liver, was attacked one day, at 11 a.m., with sore-throat, followed, at 4 p.m., by severe laryngeal symptoms, and by rapidly fatal dyspnœa at 10 p.m. After death the left epiglottidean fold was found moderately swollen with an effusion of serum, the right one smooth and shining, and greatly enlarged by infiltration with a semi-solid purulent substance. In a case of Bright's disease, which terminated fatally by dyspnœa in 1866, the autopsy showed that the cause was not ordinary œdematous laryngitis, but the exudation of a shreddy lymph-like material upon the mucous membrane of the larynx below the cords, extending down to about the eighth ring of the trachea.

Ziemssen relates the case of a young man who was attacked with extreme distress of breathing after eating some bread, and who ran off to the surgeon, thinking there must have been a needle in it, which was sticking in his throat. The laryngoscope showed that the right sinus pyriformis (outside the larynx) contained a pointed splinter of wood, which was at once removed with a pair of forceps. Only a quarter of an hour altogether had passed, yet there was considerable œdema of the right epiglottidean fold.

In cases of poisoning by corrosive liquids, laryngeal symptoms sometimes, after setting in suddenly and quickly reaching an alarming height, so that tracheotomy appears imminent, subside with no less rapidity.

In *treating* a case of œdematous laryngitis it is often well, at the commencement, to apply leeches to the neck over the sides of the larynx. Mackenzie recommends that bromide of potassium should be given freely, and that the patient should constantly suck ice. Trousseau speaks highly of spray inhalations impregnated with tannin or alum. If, however, the entrance of the larynx is found to be very greatly swollen, the best thing to do is to scarify the tissues thoroughly, so as to give the exudation an opportunity of escaping. A number of shallow parallel incisions should be made, the best instrument for the purpose being a "laryngeal lancet," or small double-edged knife mounted on a curved stem; but in an emergency an ordinary bistoury, covered up with adhesive plaster to within a quarter of an inch of its extremity, answers very well. If relief does not speedily follow tracheotomy must not be delayed. Above all, the patient must not be left,

even for a few minutes, until an opening into the trachea has been made, for a paroxysm of dyspnœa may set in at any moment, and may end fatally before there is time to fetch a surgeon.

Laryngeal perichondritis.—Several writers in the latter part of the eighteenth century recorded cases of suppurative laryngitis, with destruction of one or more of the laryngeal cartilages.

The usual opinion was that the disease began in the cartilages themselves, which (it was supposed) first became ossified and then necrosed. But it is generally admitted that the starting-point of such cases is in the perichondrium. Indeed, as far back as 1850, Dittrich, a very keen observer, pointed out, in an interesting article in the 'Prager Vierteljahrschrift,' that in a young subject it is not uncommon to find, when a small portion of the cricoid, happening to lie bare in an abscess cavity, is converted into a dirty yellow, calcified mass, that the rest of it and all the other laryngeal cartilages are in a perfectly normal state. The inference suggested by such observations is confirmed by a case at Guy's Hospital in 1859, in which the right ala of the thyroid and the right half of the cricoid were alike necrosed, whereas the left halves of their cartilages had escaped. Moreover, ossification of the affected cartilages is not always present, even in adult life. In a remarkable case of a child, eighteen months old, who died with "croupy" symptoms, we once found that part of the left half of the cricoid, which was bathed in pus, had undergone absorption, so that there was a gap in it, with thin smooth edges of perfectly natural appearance. And a year previously, in 1874, in the body of a man, aged thirty-three, the back part of the cricoid was necrosed, lying loose in an abscess cavity, while its anterior part was represented by a narrow edge of healthy cartilage, thinning off into fibrous tissue. Lastly, there is every reason to believe that disease of laryngeal cartilages is often not merely dependent upon an affection of the perichondrium, but secondary to ulceration which began in the mucous membrane. Probably this is the correct explanation of many of the cases in which such disease arises in the course of phthisis, enteric fever, smallpox, or syphilis. Dittrich suggested, in the paper already quoted, that in certain cases, occurring in persons confined to bed, necrosis of the cricoid is an indirect result of the pressure of this body, especially when ossified, against the vertebral column. His idea was that the pressure first caused ulceration and sloughing in the two opposed surfaces of the pharynx, and that then the affection of the anterior pharyngeal wall spread to the perichondrium. The morbid process would thus be strictly comparable with that which is concerned in the formation of ordinary bedsores; and Dittrich gave one case in which, the patient being a phthisical man, aged thirty-one, numerous bedsores were present at the time of death. He also recorded in detail two out of several cases in which, in bedridden patients, he had found that both surfaces of the pharynx showed local patches of ulceration, without the cricoid cartilage having as yet become involved in the disease. Störk satisfied himself that in severe enteric fever necrosis of the thyroid cartilage may arise in a similar way from pressure against the spinal column; and Ziemssen says that in old people, in whom the cricoid is ossified, that cartilage may be affected with perichondritis as a consequence of the repeated introduction of œsophageal bougies. Sometimes such disease is produced by direct injury, as in a case, recorded by Störk, of a man who was struck in the right side of the neck by a piece of

wood, which flew up while he was attending to a circular saw. In some cases, perhaps, it is the result of exposure to cold. In others no cause can be discovered.

Laryngeal perichondritis is much more common in males than in females. An analysis of twenty cases, collected from the pathological records at Berlin, showed that the period of life at which it most often occurred was between twenty and thirty years of age. But probably this was dependent upon the circumstance that eighteen of the twenty patients either were tuberculous or died of enteric fever. For at Guy's Hospital the disease, as a primary affection, has been more frequent in persons from thirty to fifty than in those who were younger. One patient was a girl under two years old, one a boy of nine, and one a man of sixty-three.

Hitherto we have spoken of perichondritis as though its necessary result were to produce suppuration and destructive changes in the subjacent cartilage. But there is every reason to believe that this is not always the case. It was remarked above (p. 871) that ankylosis of the crico-arytænoid joints may probably be caused by development of fibrous tissue as the result of perichondritis. Ziemssen records the case of a young man, in whom, during the course of enteric fever, a dark red flat projection appeared over one processus vocalis, causing hoarseness and severe pain. During convalescence this gradually diminished, and under favourable circumstances it might possibly have entirely subsided; but he insisted on going out, and after three days' exposure to weather and indulgence in alcohol, returned in a state of such severe distress that tracheotomy had to be performed.

When necrosis does occur, the cartilage is sometimes extruded from the abscess-cavity. An arytænoid is often expectorated entire; the larger cartilages commonly break up into fragments, which come away one by one. Störk speaks of having seen cases in which suppuration went on for years. When the necrosed material is completely got rid of, the cavity may be closed up by fibrous tissue.

The *symptoms* of laryngeal perichondritis vary with the exact seat of the affection. At first there is little to distinguish them from those of other subacute or chronic diseases of the larynx. The patient usually complains of hoarseness of voice or of aphonia; there may be dysphagia, cough, more or less definitely localised pain and tenderness; presently dyspnœa sets in, which may rapidly increase until it threatens suffocation. Sometimes the spontaneous evacuation of the contents of an abscess-cavity affords great relief to this symptom. In some cases of perichondritis enlargement of the cervical glands is a marked feature. In one case at Guy's Hospital they were found at the autopsy to be as large as plums. The putrid discharge which is formed in some cases is probably a direct cause of danger to the patient's life, by dropping into the air-passages and setting up pneumonia that may rapidly pass on into gangrene: two patients in Guy's Hospital died from this cause.

Perichondritis of the *thyroid* cartilage sometimes shows itself on the outer, sometimes on the inner surface of the cartilage. In the former case there is swelling, œdema, and at length fluctuation over one of the alæ or over the pomum Adami; the affected part is very tender when pressed upon. In the latter case a swelling usually appears in the position of one sinus pyriformis within the arytæno-epiglottidean fold on one side, or even below

the vocal cord, as in an instance recorded by Störk, in which it was mistaken for a polypus. Not infrequently both surfaces of the thyroid are affected in succession, so that when the abscesses have discharged themselves, milk or any coloured fluid can be injected through a sinus in the neck and run into the larynx, or a probe can be passed from without inwards until it is visible in the laryngeal mirror.

Perichondritis of the *cricoid* cartilage usually affects its posterior rather than its anterior wall. It causes marked dysphagia. Another effect to which it sometimes gives rise is paralysis of the crico-arytænoidei postici muscles, so that the cords appear fixed near the median line. In a case that occurred at Guy's Hospital in 1861 it is noted that the voice remained clear, although there was extreme dyspnœa. Sometimes the symptoms develop themselves with extreme rapidity. Ziemssen cites a case of Pitha's which ended fatally in a week from its commencement. Where suppuration occurs, the abscess may discharge itself into the pharynx, into the larynx, or into both canals at once. In some cases a swelling can be seen in the laryngeal mirror, bulging below one of the vocal cords; such a swelling has been mistaken for a solid new growth.

Perichondritis of an *arytænoid* cartilage leads to swelling and œdema of the surrounding soft parts, which may of course be visible in the laryngeal mirror. The mobility of the corresponding vocal cord is more or less interfered with, and the voice may be much impaired. Experience in the deadhouse impressed the author with the conviction that neither aphonia nor any marked alteration of the voice is nearly so constant a symptom of disease of an arytænoid cartilage as seems to be generally supposed. Cases of phthisis, in which complete exfoliation had occurred, had sometimes been free from laryngeal symptoms during life. In such cases there is a good deal of indurated fibrous tissue in the place of the cartilage, which seems to fix the cord and enable the muscles to act upon it sufficiently to maintain its functions. When an arytænoid has been exfoliated, an obvious falling in of the soft structures is often to be seen with the laryngoscope.

More than one of the laryngeal cartilages not infrequently suffer; one or both of the arytænoids, together with a part or the whole of the cricoid.

The *treatment* of perichondritis, if the disease is detected early enough, may sometimes be begun with leeches, the application of an ice-bag to the throat, and other antiphlogistic measures. When an abscess is recognised, whether outside or inside the larynx, it ought at once to be incised. Störk relates a case in which, having punctured a swelling below one of the cords and let out a quantity of pus, he subsequently brought the cavity to close by the systematic application of nitrate of silver to its interior.

In almost all cases, however, *tracheotomy* is required sooner or later; and when dyspnœa has once set in there is great risk in delaying it. The immediate result is almost always successful, but it rarely happens that the swelling of the laryngeal structures afterwards subsides enough to allow of the removal of the cannula. Schröter has had much success in the treatment of such cases by mechanical dilatation, at first with vulcanite tubes, and afterwards with pewter plugs, about an inch and a quarter in length, which can be left in the larynx for several hours at a time. Having been introduced through the mouth, the plug is held *in situ* by being bolted into the convex surface of the cannula which the patient is wearing.

LARYNGEAL TUMOURS.—New growths in the larynx are by no means very rare. From a clinical point of view it will be convenient to describe first benign, and afterwards malignant tumours.

With regard to the *causes* of benign growths in the larynx, almost the only fact hitherto ascertained is that they seem often to arise out of the irritation connected with chronic catarrh of the laryngeal mucous membrane. They are most frequently seen in persons who use the voice a great deal. They are far more common in males than in females, and, according to Causit, among children affected with laryngeal growths, boys are more numerous than girls.

Papilloma.—This, which is sometimes more accurately designated as *fibroma papillare*, is the commonest of all laryngeal tumours. It consists of a series of pointed or bulbous papillary excrescences, sometimes of small size, sometimes forming a large mass like a cauliflower, which may almost fill the cavity of the larynx. Their most frequent starting-point is from one or both of the cords, especially near their anterior extremities, or from the angle between the cords. But sometimes they arise from the false cords, or even from the epiglottis, seldom or never from the mucous membrane covering the arytænoid cartilages or the parts adjacent to them. Their colour may be either whitish, or pink, or red. They cause more or less alteration of voice, or even complete aphonia; cough, which may torment the patient greatly, and which may be of a "croupy" character; dyspnœa, which sometimes ends in actual suffocation. It now and then happens that the fragments of papillary growths become detached in the act of coughing, and are expectorated. Otherwise it is only with the aid of the laryngeal mirror that their presence can be accurately diagnosed. They not infrequently occur in young children. When removed by operation they are very apt to return, sometimes within a few months. Störk relates a case which came again and again under his observation during a period of thirteen years.

In cases of chronic laryngitis with papillary growths, Virchow finds that the lesion is usually situated towards the anterior ends of the cords. This *pachydermia verrucosa* is apt to be recurrent, but is not malignant. He looks on all the cases as simple, local, and only superficially hyperplastic, in which he finds a sharp line of distinction at the base of the epithelial growth separating it from the fibrous tissue beneath. Where, however, there is any trace of epithelium in the fibrous tissue, he considers the case suspicious. Neither the sessile nor the papillary swellings ought to show anything of an epithelial character below the border-line between the laryngeal epithelium and connective tissue. If there is no epithelium beneath this border-line, then, notwithstanding papillary outgrowths, he considers the disease to be local and benign.

Dr Percy Kidd has described a case of papillary tubercular tumours growing from the interarytænoid fold of mucous membrane in a man aged fifty, who died of phthisis with subsequent tubercular ulceration of the larynx and also of the colon. No giant-cells were discovered, but the other histological characters were those of tubercular growths, and characteristic bacilli were found abundantly ('Clin. Trans.,' 1884, vol. xvii, p. 156). Dr Kidd quotes only one other case of a young man from whose larynx several tubercular tumours were successfully removed by Schnitzler ('Wiener med. Presse,' April 8, 1883).

Fibroma, or fibrous polypus of the larynx.—This forms a round or pear-

shaped swelling, generally pedunculated but sometimes sessile, smooth or more or less lobulated, hard or more rarely soft in consistence, whitish or bright red in colour, varying in size up to that of a hazel-nut or even larger still. It is a solitary growth, its development is exceedingly slow, and it never recurs when it has once been removed by operation. Its most frequent starting-point is from one of the vocal cords, but sometimes it is attached to some other part of the larynx. Ziemssen figures one as large as a walnut, which arose from the mucous membrane covering the posterior surface of the cricoid cartilage. Growths of this kind most frequently occur in adult or middle-aged patients. Störk speaks of them as sometimes becoming ulcerated on the surface, so that they bled. In some few cases a fibrous polypus has become detached spontaneously and has been expectorated. With the laryngoscope the existence and the seat of this sort of tumour are generally easily recognised. Almost the only other possibility is eversion of the sacculus laryngis. Such a specimen, taken from the body of a man who had had no laryngeal symptoms, was shown to the Pathological Society in 1868 by Dr Moxon, and is now in the Museum of Guy's Hospital; it appeared like a semi-elliptical tumour hanging down in front of one of the cords, and could easily be replaced. Dr Lefferts, of New York, has diagnosed this affection in the living subject. The writer had a case in Mary Ward which presented this difficulty, but Mr Symonds found that it was not possible to push the projection into the tube of the larynx, as can be done when the mucous membrane is everted.

The symptoms produced by a fibroma of the larynx vary with its seat. Unless it is at a distance from the glottis the voice is almost always more or less affected, one reason for this being that even if the growth does not actually interfere with the apposition of the cords the surrounding mucous membrane is sure to be affected to a greater or less extent with catarrh. When a polypus has a pedicle of some length it may rise between the cords during phonation, and rest upon their upper surface, whereas during inspiration it falls down between them. The occurrence of dyspnœa is very uncertain. Dr Mackenzie had a patient who invariably slept with her hand resting under the neck, and who would immediately wake up with distress of breathing whenever by chance her hand slipped away. In a case recorded by Lieutaud about a century ago, the patient died of sudden suffocation as the result of stooping out of bed to pick up a book which had fallen on to the floor. He had been conscious some time of the presence in the larynx of something which he could not get rid of by coughing.

Mucous cyst.—This is sometimes found upon the epiglottis, as in a case which occurred in 1863 to Mr Durham, who has recorded it in vol. xlvii of the 'Med.-Chir. Trans.' The patient was a boy, aged eleven, who had suffered for some months from dysphagia, from hoarseness and feebleness of voice, and from attacks of dyspnœa which came on especially during sleep. The cyst, which was situated upon the laryngeal surface of the epiglottis, was incised, and gave exit to a glairy, thick, muco-purulent matter; it is therefore evident that the cyst was inflamed, and, indeed, the epiglottidean folds themselves were swollen and œdematous. In other cases a similar cyst has been found in the ventricle of Morgagni.

Dr Edis has recorded an instance in which there was a cyst of the size of a hazel-nut in the larynx of an infant, who died of suffocation thirty-seven hours after birth. Dr Abercrombie showed to the Pathological Society in 1881 a remarkable case of congenital cyst of the crico-thyroid

membrane, which blocked the glottis so much that the child (a female infant who died on the fourteenth day after birth) had never been able to cry or to breathe properly ('Path. Trans.,' xxxii, p. 33).

In some rare cases a laryngeal tumour has been a *myxoma*, a *lipoma*, an *angioma* (Mackenzie), or an outgrowth of the thyroid body penetrating the crico-thyroid membrane.

Treatment of benign tumours.—Mackenzie advises that small growths on the epiglottis or on the false cords should be left alone if they give rise to no inconvenience; he has observed several cases in which small "warts," after reaching a certain size, have ceased to grow.

Various instruments have been devised for the purpose of removing laryngeal tumours through the natural passages, or (as it is termed) by the "endo-laryngeal method"—knives (guarded or unguarded), cutting forceps, crushing forceps, guillotines, écraseurs, the galvano-cautery, have all found their advocates. It is needless to enter into details with regard to them, because it is not likely that any medical man would attempt to use them without having had special training, nor without consulting the works of those who have devoted themselves to the study of laryngeal affections. In choosing an instrument for a particular case, the degree of hardness of the growth and the character of its pedicle form important considerations; they must be determined as far as possible by the use of a laryngeal probe. It is not advisable to use an anæsthetic unless tracheotomy has previously been performed. The local use of cocaine in solution or as spray has, however, now superseded all other attempts to produce anæsthesia.

A point which must be remembered is that, *cæteris paribus*, more skill is required in the removal of a very small laryngeal growth than of one which is larger. Both in this country and abroad an extraordinary degree of skill has now been attained in the performance of endo-laryngeal operations. The immediate result of the introduction of laryngeal forceps, or of any other instrument, is the production of a violent spasm, with a feeling as of impending suffocation, but this quickly passes off. When there are a large number of papillomata in the larynx, repeated endo-laryngeal operations are of course necessary, which may run over a period of several weeks.

In cases in which it is difficult or impossible to operate through the natural passages, the question arises whether recourse should be had to "thyrotomy," or the division of the thyroid cartilage in the median line, with separation of its halves, enabling the surgeon to seize the growth or growths and to clear out the whole cavity of the larynx on a single occasion. This procedure, which had been adopted for the removal of foreign bodies nearly a century ago, was vigorously advocated by Mr Durham in a paper read before the Royal Medical and Chirurgical Society in 1871. But subsequent experience seems to have greatly limited the range of cases within which alone its performance can be justified. At the International Congress in 1881 opinions were almost unanimous with regard to this question. It was urged that the operation is attended with considerable danger to life from hæmorrhage, or from other consecutive evils, among which pneumonia and necrosis of cartilages with suppuration perhaps take the principal places. Further, it was shown that a permanent impairment or loss of voice is a not infrequent result of thyrotomy, though, on the other hand, there are many recorded cases in which the voice has been perfectly restored. Lastly, it was pointed out that in some patients there is great difficulty in getting the alæ of the thyroid cartilage wide enough apart to enable the operation to be

successfully completed, and that experience does not at all confirm the expectation that the risk of recurrence of multiple papillomata is diminished by the adoption of this procedure as contrasted with endo-laryngeal methods. Most of those who attended the Congress thought that even in young children (in whom multiple papillomata are so common) thyrotomy is seldom necessary. Krishaber related the case of a child, aged six, in whom he succeeded in rapidly removing a number of tumours without a laryngoscope by sliding a pair of forceps along his index finger into the larynx. One criticism it is fair to make upon the speeches delivered at the Congress; it is that laryngologists, not being so much accustomed to ordinary cutting operations, have probably in the performance of thyrotomy met with greater difficulties and obtained less satisfactory results than might occur to hospital surgeons in the like cases.

A mucous cyst in the larynx requires only to be incised and to have its interior rubbed with caustic. Contrary to what might have been expected, it seems seldom or never to fill again.

Malignant growths in the larynx are sometimes *Sarcomata*, generally of the spindle-cell kind. Ziemssen speaks of there being more than twenty recorded instances of such an affection; its seat is usually on or near one of the vocal cords. Mackenzie figures a sarcoma which he describes as growing from the posterior surface of the cricoid cartilage; it had a papillomatous character.

Carcinomata of the larynx usually belong to the keratoid variety, such as are commonly called epitheliomata. They must be rare in comparison with cancers of other parts, for in the *post-mortem* room of Guy's Hospital only some four examples have been met with between 1854 and 1883. All of them occurred in patients between the ages of fifty-eight and sixty-five. According to Ziemssen, however, they are not uncommon relatively to other laryngeal growths; he speaks of having collected 147 cases, of which thirteen had come under his own observation. Among the patients there were many more men than women. In one curious case the development of the disease was preceded, at an interval of some months, by a fracture of the thyroid cartilage, the result of an attempt at strangulation.

The larynx sometimes becomes affected with cancer by extension from the pharynx or from the base of the tongue. But in the cases now under consideration the starting-point of the affection is in the laryngeal mucous membrane, its original seat being generally one of the cords, one of the ventricles of Morgagni, or one of the false cords. In a case that occurred at Guy's Hospital in 1875, the amount of the growth at the time of the patient's death was remarkably small; the left arytæno-epiglottic fold showed a whitish thickening, with puckering, as of a healed ulcer, two or three lines in diameter; the thickening extended down to the false cord on that side: until the microscope revealed the structure of a carcinoma, it was doubtful whether a new growth was present. In another case, observed in 1862, there was a raised patch, somewhat papillary in character, growing from the left cord and the parts around. But in many instances, as the disease spreads, extensive ulceration occurs. The structures outside the larynx become infiltrated with the growth, which may protrude into the pharynx, or form an obvious tumour in the neck. The ulcerated surface within the larynx may pour out an abundant ichorous discharge mixed with blood, or may even be the seat of copious hæmorrhage. In such cases the breath

becomes horribly fœtid. Perichondritis, leading to suppuration and to necrosis of cartilages, often occurs as a complication. Death may be due to œdematous laryngitis, or (as in two out of four cases at Guy's Hospital) to pleuro-pneumonia and empyema.

The laryngoscopic diagnosis of carcinoma of the larynx is by no means always easy. At an early stage, when there is little beyond a diffuse infiltration of the mucous membrane, the case may be taken for one of perichondritis; and at a later period, when an ulcer has formed, it is often difficult to distinguish between syphilis and cancer, as in a remarkable case recorded by Ziemssen, in which he fortunately gave iodide of potassium with rapid and complete success, the patient being an old man of sixty-eight. Of the other symptoms *hoarseness*, seldom amounting to complete aphonia, is the most constant and generally the earliest. According to Ziemssen, indeed, it is almost the rule that there is a "prodromal hoarseness," lasting a year or two; and in several of the cases which he collected this was prolonged during three, four, or five years, and once even during twenty-six years. It is certainly difficult to suppose that the affection had a definitely malignant character throughout such long periods; and, indeed, Ziemssen's statements with regard to the duration of laryngeal cancer appear scarcely consistent with what one knows of the rate of progress of a similar affection of other parts: he speaks of several cases which lasted three or four years, and of some which lasted even six, ten, or fifteen years. Next to hoarseness, *pain*, which may either be seated in some one spot within the larynx, or referred deeply to the pharynx, is the most conspicuous symptom. And Ziemssen lays stress on the frequent radiation of pain into one or other ear; this pain in the ear he associates with the auricular branch of the vagus: he found it present in five out of thirteen cases in which inquiries were made about it, and sometimes when there was no pain in the larynx itself. As a rule, dyspnœa occurs sooner or later; it may be especially marked when the patient is lying down. There may also be dysphagia. In all cases of suspected carcinoma of the larynx careful search must of course be made for enlarged cervical glands; but Ziemssen says that they can seldom be detected within the first six months, and often not for a year, or even longer. However, in a case of Mr Durham's, in 1879, which was yet in an early stage, there were already two flat subcutaneous nodules, one near the right clavicle, the other on the edge of the left sterno-mastoid muscle. Secondary cancer of the viscera is very rare.

The *treatment* of carcinoma of the larynx can often be only palliative; but Ziemssen's case, already referred to, shows that whenever there can be a doubt as to the nature of the disease the patient should have the benefit of the chance afforded by a course of iodide of potassium. Indeed, at the London International Congress, in 1881, Dr Semon spoke of having seen this salt produce improvement in cases of cancer. Tracheotomy is generally required sooner or later; the average duration of life after this operation is said to be not more than a year.

It is chiefly in cases of malignant tumour that the question of the "total extirpation" of the larynx has to be considered. This operation, originally performed for syphilitic stenosis by Dr Watson, of Edinburgh, in 1866, was carried out in 1873 by Billroth with temporary success in a case of cancer. In a paper read in 1881 by Dr Foulis, of Glasgow, before the International Congress, reports of thirty-two cases are collected, in twenty-five of which the disease was carcinoma. In fourteen out of the twenty-five death occurred

within sixteen days after the operation; and in not one of the remainder was life known to have been prolonged more than nine months, the only patients who were stated to be alive when the paper was read being two who had been operated on three months previously. However, Bottini, of Turin, had one very successful case, in which the larynx was extirpated for sarcoma; in 1881, six years after the operation, the patient was well, and had been able to work in the fields and to act as a postman. This evidently suggests the possibility of a like success in cases of cancer, if they could be operated upon at a sufficiently early stage. Moreover, although the condition of those patients who have survived used to be very miserable, there being great difficulty in deglutition, in consequence of the large opening in the neck, which could not be closed, yet recent improvements in technique have greatly increased their comfort. The voice, moreover, can be more or less successfully restored by the use of an artificial vocal apparatus, such as was originally invented by Störk.

The results of about twenty partial removals of the larynx are on record, and of these nearly a fourth are reported as living twelve months after the operation. In more than 60 cases the larynx has been entirely removed; and of these a third died from the effects of the operation, 16 within a year from return of cancer, and 9 remained apparently cured sixteen months or more after the operation.*

In the case of the late Emperor Frederick, carcinoma of the larynx began as usual in a papilloma, and after its nature was recognised at a still early stage extirpation was advised. The fatal termination occurred about ten months later. In a more recent case of the same terrible disease, the affected half of the larynx was removed by Dr E. Hahn, of Berlin, and not only has life been prolonged till the present time, but the voice has been sufficiently preserved to enable the patient to continue his duties as the magistrate in a London police court.

Malformations of the Larynx.—This will be the most convenient place to mention certain congenital abnormalities of the larynx that occasionally give rise to clinical symptoms. One such appears to be almost confined to female infants, and causes the act of inspiration to be attended with a loud crowing noise, which is nearly constant, continuing even during sleep and after the administration of chloroform, though it is louder during the day. It is sometimes increased by exposure of the body to cold, or in other ways. The noisy state of the breathing is present from the time of birth, but disappears entirely at the end of about a year. Dr Lees has had an opportunity of making an autopsy in a case of this kind, in which death was due to diphtheria; and he found ('Path. Trans.,' 1883) that the epiglottis was folded on itself, like a leaf on its midrib, the arytæno-epiglottic folds being almost in contact. This condition had been seen in the laryngeal mirror during life, and it is probably not uncommon, for Dr Lees had seen three other cases, and it has also been recorded by Dr Gee and by Dr Barlow. It is important as simulating laryngismus stridulus.

Another and a much more serious malformation, described by Mackenzie, consists in a longitudinal bifurcation of the epiglottis, forming two flaps which (in a case that he saw) fell into the larynx, and caused constant symptoms of laryngismus from the first week and death at the end of four months.

* These numbers are taken from an abstract in the 'Brit. Med. Journ.' of June 16th, 1888, from Dr Max Scheier's paper in the 'Deutsche med. Wochenschr.' of June 7th.

Lastly, a congenital band of mucous membrane sometimes connects together the anterior parts of the cords. Mackenzie has recorded such an instance in vol. xxv of the 'Pathological Transactions.' The patient was a young lady, aged twenty-three, who had had complete aphonia from birth, never having cried, even as an infant. There seems to have been no dyspnœa. Laryngoscopically the web was seen as a flat membrane during inspiration, but on attempted vocalisation it became folded up, and protruded so as to resemble a tumour, of red colour, and of about the size of a haricot bean. It was excised, and the patient immediately afterwards spoke, and soon acquired a perfectly natural voice. In a case of Dr Poore's, exhibited at the International Congress in 1881, the patient, a girl of thirteen, could speak, but with a peculiar falsetto tone of voice; she had been liable to attacks of dyspnœa from infancy.

Foreign Bodies in the Larynx.—Not an uncommon cause of severe laryngeal symptoms is the entrance into the upper air-passages of foreign bodies of various kinds. As a rule, such bodies are sucked down into the larynx during a deep inspiration, as the result of coughing, laughing, sneezing, or talking while there is something in the mouth. In children the accident sometimes occurs during the night, in consequence of the habit of sucking a toy before going to sleep; and it may even happen to an adult who wears false teeth, unless he is always careful to take them out at bedtime. It is surprising what large things will sometimes enter the larynx. Mackenzie relates a case of a boy, aged six, who went to sleep with a toy engine in his mouth; during the night it was drawn into the air-passages, and tracheotomy had to be performed. The cause of the sudden attack of dyspnœa which had occurred was not discovered at the time, but some months later the toy was found impacted in the subglottic region, and was removed by thyrotomy.

The symptoms produced by the entrance of a foreign body into the larynx are generally at first very violent; there is a most distressing sense of suffocation, the face becomes cyanotic, the inspiration is prolonged and whistling, a cold sweat breaks out, the patient tears at his throat with the hands, under an irresistible impulse to relieve himself of the cause of his sufferings. Such a case may end fatally within a minute or two by asphyxia. But it is important to remember that if the air-passages are completely closed there is sometimes no obvious trouble with the breathing; the patient falls dead at once, and it may be only at the autopsy that the real cause is discovered of what had seemed to be an attack of syncope. In the act of vomiting, for example, it may happen that there is inhaled into the larynx a soft, pulpy mass, which entirely fills it.

In many cases a foreign body, having passed into the larynx, at once falls through into the trachea; the indications of laryngeal irritation soon subside, and are followed by a fresh set of symptoms, which will be discussed in the next section. In other cases, again, the foreign body is quickly coughed out into the mouth, after which it perhaps is swallowed, and ultimately passed through by the bowels. It may thus happen that laryngeal symptoms—brassy cough, dyspnœa, and alteration of voice—which were present for some little time, entirely disappear; and it is then difficult to decide whether there is still something in the air-passages or not. Another class of cases in which a diagnosis is not always easy occurs in hysterical women; such persons seek advice for tickling or pricking sensations

in the throat, which they declare to be due to the presence of a needle or a pin, or a bristle, but which are really "paræsthesiæ" of neurotic origin.

Finally, when a foreign body is of large size, or when it has pointed ends, it generally becomes fixed in the larynx, and may remain there, as in a case already alluded to, for a great length of time. At any period œdematous laryngitis may set in, attended with severe dyspnœa. But if the foreign body is impacted in the ventricle of Morgagni, there may be merely pain and cough, with perhaps some degree of hoarseness, but no dyspnœa. In such cases the diagnosis rests entirely upon repeated laryngoscopic examination.

When there is a foreign body in the larynx it has, of course, to be removed in one way or another, and generally by surgical interference. In most cases tracheotomy is required as a preliminary measure.

OBSTRUCTION OF THE TRACHEA.—The trachea and the main bronchi are liable to but few affections except such as are common either to the larynx above or to the tubes within the lungs below, and the clinical importance of such affections attaches itself always to the narrower rather than to the wider parts of the air-passages. Hence there is no need to give a separate account of tracheitis: the plastic form has been described under diphtheria; the tubercular with tubercular laryngitis, and the catarrhal form will be dealt with under bronchitis.

But there are a variety of diseases which at some point may narrow the calibre of the trachea or of the bronchi, with the result of producing a definite and characteristic group of symptoms. Of these diseases some have their seat outside the walls of the air-passages; others originally affect the walls of the air-passages themselves; and others, as in the case of foreign bodies, obstruct their channel from within.

Since it is often an accident whether in a given case the part narrowed is the lower end of the trachea or one or both of the bronchi, it is useless to attempt to separate the affections of these several parts from one another, particularly since both are often involved at the same time. All that is possible is to describe the special symptoms which in certain cases indicate that the obstruction is altogether limited to one of the bronchi, leaving the trachea free. The general designation, *obstruction of the trachea and bronchi*, includes the whole group.

A. Of the diseases which, starting *from outside*, may obstruct the air-passages and so cause what may be termed *stenosis* from *compression*, the following are the most important:

1. *Tumour of the thyroid.*—It is well known that a bronchocele may compress the trachea in the neck, flattening it from side to side, so as to make it "scabbard-shaped," and often pushing it out of the straight line or bending it. It is by no means the largest goitres which are most apt to have this effect; much depends upon the exact situation of the growth, and upon the condition of the overlying muscles, which oppose resistance to its extension outwards, but which were in one case found by Virchow to be in a state of fatty degeneration. Another point of great importance, for a knowledge of which we are also mainly indebted to this writer, is that the middle lobe of the thyroid, when it becomes enlarged, sometimes passes down behind the sternum so as to compress the trachea backwards against the spine. He even maintains that such a "substernal goitre" may be present without there being any obvious

swelling of the thyroid in the neck. This, however, seems to be doubtful. Ross, of Zürich, in a very able paper in vol. xxii of the 'Arch. f. klin. Chirurgie,' has drawn attention to a peculiar change in the tracheal cartilages which occurs as the result of the presence of a goitre, and renders them soft and flaccid. The way in which he recognises this after death is by dissecting off all other structures from the larynx and trachea and then placing them upright; the tube collapses at some one point, bending sharply so that its channel becomes completely closed. A like collapse is believed by him to be the cause of the supervention of sudden fatal dyspnœa as the result of goitre; he supposes that patients instinctively have to maintain the head in such a position as to avoid this occurrence, but that the muscles become relaxed during fainting, or sleep, or chloroform narcosis, or as the result of weakness. Dr Bristowe, for example, relates, in vol. iii of the 'St Thomas's Hospital Reports,' the case of a woman who was admitted for feverish symptoms, but who was one day suddenly attacked with intense difficulty of breathing, followed in a minute or two by blackness of face and insensibility. Fortunately he was close at hand, and finding that she had a tumour in the front of the neck, part of which was evidently cystic, he had this punctured, with the result that two or three ounces of a reddish-brown fluid were removed, and that she was quickly restored to health. In other cases enlargement of the thyroid is due not to a mere overgrowth of its tissues, but to the presence of a hydatid or of a malignant new growth, which may perforate the trachea and protrude into its channel; a well-marked instance of the latter event occurred at Guy's Hospital in 1873.

2. *Thoracic aneurysm.*—Among twenty-seven cases of aneurysm taken without selection from the *post-mortem* records of Guy's Hospital, there was interference with the trachea or with one of the main bronchi in every one. In fourteen the sac arose from the arch and pressed straight backwards upon the lower end of the trachea itself, flattening it, and often adhering closely to its walls. Probably in several of these cases the pressure extended also to one or both of the bronchi. But what is surprising is that in no fewer than seven cases the aneurysm seems (from the description given in the case-books) to have pressed solely upon the *left* bronchus: in three of these the sac arose from the summit of the arch on its left side, and pressed mainly upon the upper or upon the anterior surface of the tube; in the other four it came from the descending part of the arch and pressed forwards upon the posterior surface of the air-tube. On the other hand, there were only two cases in which the sac, having its origin in the right side of the arch, compressed only the *right* bronchus. The remaining four cases were examples of what is commonly termed aneurysm of the innominate artery; in them the sac pressed upon that part of the trachea which lies behind the upper part of the sternum or in the root of the neck.

It is difficult to say how many of the twenty-seven cases were characterised by other symptoms which actually did indicate, or might have indicated, the real nature of the disease during life. But in three instances the sac was of very moderate size. One, which flattened the trachea, was a round pouch "of the size of a walnut" (as seen at the autopsy), arising by a definite orifice from the posterior walls of an aorta severely affected with arteritis deformans. Another, which compressed and opened into the left bronchus, was "no bigger than a marble." The third,

which likewise interfered with the left bronchus, was "of the size of a small plum."

3. *Mediastinal tumour.*—In the period during which the twenty-seven cases of aneurysm were observed in the deadhouse at Guy's Hospital, there occurred nearly an equal number of cases in which the great air-passages were narrowed by mediastinal new growths; and among twenty-four of them in which details are given as to the exact seat of the lesion there appear to have been eight in which the obstruction affected the lower end of the trachea or both bronchi (sometimes much more on one side than on the other), six in which it was limited to the right bronchus, and ten in which it was limited to the left bronchus. In every instance the new growth invaded the walls of the air-passages, thickening them, and not merely pressing upon them from without. Indeed, there are two other cases besides those already mentioned, in each of which it is expressly reported that although the bronchus on one side was penetrated by the tumour, there was no narrowing of its calibre. Among the whole number of cases there seems to have been hardly one in which, if marked symptoms of stenosis were present, there were not also observed other symptoms and physical signs sufficient to show that the obstruction was due to disease beginning outside the air-passages. The pathological reports seem to justify the inference that mediastinal growths seldom invade the trachea or the bronchi at an early period in their development. It must, however, be remembered that they are not likely to be seen in the deadhouse at this stage, since, unlike aneurysms, they do not commonly destroy life suddenly and unexpectedly by hæmorrhage. In an interesting case of lymphosarcoma of the mediastinal glands, recorded by Weil in the 'Deutsches Archiv' for 1874, all the symptoms and signs of tracheal obstruction disappeared suddenly eight days before death; at the autopsy it was found that this was due to the giving way of the softened mass, which must have poured its substance into the air-passages, although the sputa had shown no fresh appearance even under the microscope.

4. *Mediastinal abscess.*—Abscesses of various origin may compress the trachea or a bronchus. A striking case is recorded by Schnitzler, in the 'Wiener Klinik' for 1877. The patient was four years old; an abscess as large as a child's fist pushed the trachea forwards and to the right; its starting-point was caries of the second and third dorsal vertebræ.

5. *Caseous disease of the bronchial glands.*—This is commonly given as one of the causes of obstruction of the trachea or of a bronchus, especially in children. Vogel, however, says that although there may be slight flattening or indentation it does not go on to actual stenosis. On the other hand, Widerhofer, in Gerhardt's 'Handbuch,' describes this occurrence, and also cites instances in which after prolonged dyspnœa abscesses dependent on disease of the bronchial glands discharged into the air-passages with relief to the urgent symptoms.

6. *Carcinoma of the œsophagus* is mentioned by Riegel and other writers as an occasional cause of stenosis of the trachea. But although it frequently invades the air-passages, it is rare for it to produce symptoms indicating interference with the entrance of air. As already remarked (p. 871), it may cause a bilateral paralysis of the abductors of the glottis, and so render the performance of tracheotomy necessary. In all probability the emaciation which is so marked a symptom of œsophageal cancer is attended with great diminution in respiration.

7. *A dilated left auricle*, secondary to mitral stenosis, may compress the left bronchus, as was first pointed out by Mr Wilkinson King in 1838, and as may be still seen by his preparations in the museum of Guy's Hospital. Friedreich has recorded an instance in which pressure on the bronchus from this cause was actually diagnosed by physical signs four years before the patient's death. At the autopsy, made by Virchow, it was found that only a very narrow channel was left.

B. Of the diseases which, starting *in the walls of the trachea* or of the main bronchi, may narrow the calibre of the air-passages, some are exceedingly rare. Demarquay, for example, is cited by Riegel as having observed a case in which such an affection arose from ulceration in a case of glanders. Langhans, in the fifty-third volume of 'Virchow's Archiv' (pl. xiii), recorded in 1871 an instance of primary carcinoma, having its origin in the mucous glands of the lower end of the trachea and right bronchus, which destroyed the life of the patient, a man of forty; it appeared as a warty affection of the lining membrane, extending also by infiltration into the muscular and fibrous external coats. Whether a simple local inflammatory process is capable of thickening the walls of the lower air-passages, so as to obstruct their calibre, is doubtful. Andral and Wilks are quoted by Riegel as having reported such cases; but the observations of Wilks refer to syphilitic stenosis only.

Syphilis is, indeed, by far the most important cause of obstruction of the lower air-passages, if the diseases producing compression from without be excluded. Gerhardt, in vol. ii of the 'Deutsches Archiv,' alluded to twenty-two examples of it of which he had made an analysis; and seven instances presented themselves in the *post-mortem* room of Guy's Hospital between 1861 and 1874. Sometimes the disease is limited to a single spot in the trachea, as in a specimen, taken from a patient of Dr Bright's, which is contained in the museum of Guy's Hospital, and in which opposite the second ring there is a contraction, as if produced by a ligature. Much more often it extends along the trachea; it may occupy its whole length, and may be prolonged into one or both of the bronchi.

The bronchi are seldom affected when the trachea escapes. But Wilks, in the 'Guy's Hospital Reports' for 1863, relates a case in which the right bronchus alone was stenosed; and in another case, observed at the hospital in 1875, the lesion was found to have attacked only the left bronchus and the upper branch of the right. Both bronchi were narrowed, with the trachea free, in a third case ('Path. Trans.,' vol. xxviii, p. 336). The mucous membrane is commonly raised into a series of irregular bands and ridges, which Wilks has taught us to regard as the cicatrices of former ulcers. Gerhardt, indeed, has reported a case in which at the time when death occurred from a form of chronic pneumonia there was simply an unhealthy ulcer with raised edges, occupying the right bronchus and one of its branches for about an inch, and exposing the bronchial cartilages. But the view taken by German pathologists generally is that the fundamental lesion is a diffused thickening of the whole tracheal wall, raising its lining membrane into folds and prominences. They describe ulceration, more or less extensive, as of not infrequent occurrence, but they regard this as secondary. It may spread deeply, setting up perichondritis, and leading to ossification and necrosis of the tracheal or bronchial cartilages, which may be exfoliated and appear in the sputa; or it may penetrate to the tissues outside the air-passages, and

form an external abscess. In a case that occurred at Guy's Hospital in 1865 there was perforation of the aorta, so that the patient died of sudden hæmorrhage. In other instances the tracheal rings, instead of being exposed and detached, become atrophied and bent on themselves or dragged one over the other. Whether syphilitic stenosis affecting a bronchus ever leads to its complete obliteration is doubtful. When such a condition has been found it has been regarded as congenital. Thus Ratjen, in vol. xxxviii of 'Virchow's Archiv,' described a case occurring in a man aged forty-nine, whose left bronchus was converted into a fibrous cord for an inch and a half of its length, the corresponding lung being quite airless, while the right lung was enormously enlarged, and apparently in a state of true hypertrophy, its air-cells being of normal size. But, as Cohnheim observes of this case, the presence of pigment in good quantity in the collapsed left lung is clear proof that it had at one time been functionally active.

With regard to the age at which syphilitic stenosis of the trachea proves fatal, it is worth noting that the large majority of cases at Guy's Hospital have been in persons between forty and fifty years old, and it has occurred in men far more often than in women. Among the cases collected by Gerhardt there was a far wider range of ages; one patient was under ten, and another under twenty: probably these are the two cases which he refers to inherited syphilis. Two instances of syphilitic stenosis in children twelve years old are given by Widerhofer.

c. Obstruction of the lower air-passages may be due to a *foreign body*. As already remarked (p. 915), a foreign body which enters the larynx through its upper orifice rarely remains fixed there, unless it is either very large or pointed in shape. Beans, peas, nut-shells, pebbles, small coins, fragments of bone, often fall into the trachea. Sometimes they remain free for a time, moving up and down as the patient coughs. One may then be able to feel the impact of the foreign body against the side of the trachea with the fingers placed outside the patient's neck, as was observed by Mr Lucas in the case of a child with a pebble in its air-passages ('Clin. Soc. Trans.,' xv). Even in that case there were physical signs which rendered it probable that the pebble lay in the right bronchus in the intervals between the fits of coughing.

As a rule, such bodies soon become fixed in the right bronchus or in one of its main divisions; the reason why they enter it rather than the left bronchus being that the fork between the two is slightly to the left of the middle line, so that the opening into the right bronchus is rather the more direct. Sometimes, however, the left bronchus is the one into which a foreign body passes; and sometimes each bronchus in turn, the body becoming dislodged by cough, and falling now into one, now into the other. In certain cases the cause of obstruction is not, strictly speaking, a foreign body at all; it may be a tooth or a fragment of uvula, or a pharyngeal polypus separated by the hand of the surgeon; it may even have found its way into the air-passages by ulceration from the living tissues, as when it is a necrosed laryngeal cartilage, a concretion from a bronchial gland, or a hydatid from the liver. An accident which has several times happened after tracheotomy is that a portion of the tube has become detached from the rest and has dropped into the trachea. Altogether the literature of foreign bodies in the air-passages is very extensive, no fewer than 374 recorded cases having been collected and analysed by Kühn.

As may be supposed, foreign bodies are most frequently found in the air-passages of children and of lunatics. But other patients also, when attacked by sudden and violent symptoms as the result of this accident, may be altogether ignorant of the cause. Hamburger is cited by Riegel, in 'Ziemssen's Handbuch,' as having recorded a case of an old man aged seventy, who fainted after a journey, and was found in a state of dyspnœa, with evident obstruction of the right bronchus. An emetic was given, which led to the expectoration of a green pea swollen to the size of a bean; and afterwards it was learnt that when he was eating peas one day, something had gone the wrong way.

Symptoms of stenosis of the lower air-passages.—Of these the most important is *dyspnœa;* as contrasted with laryngeal stenosis that of the trachea is characterised by difficulty of breathing without loss of voice. One must, however, remember that the power of speaking well and even loudly is not in itself proof that the seat of an affection attended with severe dyspnœa is not in the larynx; for in bilateral paralysis of the abductors of the vocal cords precisely this combination of symptoms is met with, as has already been shown (p. 875). Moreover, it frequently happens that the voice in cases of tracheal stenosis is weak, thin, and devoid of sonorous quality, from deficiency in the stream of air reaching the larynx from below. A further point to be borne in mind is that syphilitic disease of the larynx is often combined with a like disease of the trachea; a patient may have lost his voice as the result of a syphilitic affection of the larynx, but the dyspnœa from which he suffers may nevertheless be dependent on mischief lower down, so that, if tracheotomy should be performed, the operation may turn out a failure. Again, in many cases of aneurysm or of mediastinal growth, tracheal stenosis is accompanied by paralysis of laryngeal muscles, as the result of pressure upon one or both of the recurrent laryngeal nerves.

Whether or not the voice is affected, it is therefore essential to make a thorough laryngoscopic examination in all cases of suspected stenosis of the trachea or of the main bronchi. Moreover, it is sometimes possible, especially if the larynx is healthy, to make a direct diagnosis of the nature of a tracheal lesion by examination with the mirror. The lower part of the windpipe, with its bifurcation and the orifices of the two bronchi, is said to have been first seen in the person of Czermak himself by Elfinger. Türck has described the conditions most favourable to a successful exploration of these parts. The patient should be seated with the body and the neck upright and the head bent slightly forward, the object being to bring the axis of the larynx and that of the trachea into a straight line. The mirror must be placed against the soft palate, rather further forwards than usual, and with its surface nearly horizontal. The observer should sit at a lower level than the patient. The illumination must be very bright, and the light should be thrown into the mouth horizontally, or rather from below. An aneurysm may sometimes be seen bulging into the trachea, as in a case of innominate aneurysm which was examined by Mr Lane when house physician at Guy's Hospital. It must not, however, be supposed that a mere slight pulsation of the lower end of the trachea necessarily indicates a morbid condition, for Gerhardt and Schrötter have shown that such pulsation transmitted by the great arteries is present in many healthy persons.

Local diagnosis.—An important distinction between stenosis of the lower air-passages and that of the larynx was first pointed out by Gerhardt It

is that in the former affection the larynx does not during inspiration make the rapid and extensive vertical movements which occur when the larynx is itself the seat of obstruction to the entrance of air. According to this observer, if with severe stenosis the range of descent of the larynx is not more than one centimetre, one may confidently assert that the disease is either in the trachea or possibly in both bronchi, but not in the larynx. He also remarks that the position of the patient's head differs in the two sets of cases. When the obstruction is laryngeal, the head is thrown backwards as far as possible. When it is tracheal, the head is stretched forwards, and the chin slightly depressed, so as to relax the trachea.

The dyspnœa in stenosis of the lower air-passages is in the main inspiratory, like that in laryngeal stenosis. It is less often extreme in degree, on account of the greater calibre of the trachea as compared with that of the glottis. Consequently the breathing is not usually much altered in frequency; nor are the lower ribs and the other unsupported parts of the chest walls very much sucked in. But should the disease go on to actual suffocation, all these phenomena may be as marked as they possibly can be.

There is generally from an early period the most noisy stridor, heard not only through a stethoscope over the trachea, but also more or less on auscultation over every part of the chest, so that it drowns the normal breath-sounds. Indeed, it is commonly obvious to everyone standing near the patient. According to Gerhardt, the only cases in which any safe conclusion as to the seat of the obstruction can be drawn from observing the spot at which this sound is heard loudest through the stethoscope are those in which this spot is directly over the trachea in the neck. When there is stenosis of the lower part of the trachea, it often happens that the sound is audible with greater intensity over the larynx than over the sternum. Sometimes a râle is constantly discoverable over some particular point in the trachea. A sign to which Demme has drawn attention is that in prolonged cases of constriction of the lower air-passages the circumference of the upper part of the thorax becomes lessened.

In most cases of stenosis of the trachea, the dyspnœa undergoes aggravation from time to time; there are paroxysms of extreme distress attended with cyanosis, and generally one of them at length proves fatal. It was formerly supposed that the cause of such attacks was paralysis, or perhaps spasm, of the vocal cords from implication of one or both of the recurrent laryngeal nerves; but Dr Bristowe, in an admirable paper in the third volume of the 'St Thomas's Hospital Reports,' showed that this is not the fact. They are probably due either to acute swelling of the mucous membrane at the seat of pressure, or to an accumulation of mucus there which cannot be dislodged, or perhaps in part to spasm of the muscular tissue of the trachea itself. It is important to notice that no relief is to be expected from the performance of tracheotomy.

The patient commonly complains more or less of oppression of the chest, of soreness behind the sternum, or of actual pain. There may or may not be cough, with expectoration of mucus, perhaps tinged with blood, according to the nature of the disease which produces the stenosis.

In contrasting the physical signs of obstruction of one bronchus with those of stenosis of the entire lower air-passages, the first point to be remarked is that much depends upon whether the obstruction is complete or partial. In the former case there is absence of vesicular murmur over the corresponding side of the chest, with impaired movement of the ribs and of

the diaphragm, deficient vocal fremitus, and a normal percussion-sound. After a time the side may actually be found to have fallen in, and to measure less than the other side. In the latter case a snoring, whistling, or humming sound may be heard over the root of the lung between the scapula and the vertebræ, or there may be moist sounds there. A thrill may sometimes be felt with the hand placed upon the surface of the chest.

One clinical peculiarity of the obstruction of a main bronchus caused by a foreign body is that it is far more sudden, as well as more complete, than that due to any other cause. Consequently its effects may be supposed to approximate more closely than in any other morbid condition likely to be observed in man, to those of the plugging of a bronchus by wedges of laminaria which were studied by Lichtheim in a series of experiments on rabbits recorded in vol. x of the 'Arch. f. exp. Pathologie.' The opposite lung in these experiments became enormously distended. Very often it gave way, so that pneumothorax resulted; and even when this did not occur the animal usually died within twenty-four hours. What proved that the rapidly fatal issue was immediately dependent upon the state of this lung rather than of the one which was deprived of air, was that death did not follow when the pleura was laid open on the side of the obstructed bronchus. A bean or a pea is capable of swelling, like the laminaria plugs used by Lichtheim, although more slowly; and his results are worth bearing in mind, because it may be that in the failure of all attempts to extract a foreign body from a bronchus, it might sometimes be justifiable to admit air into the pleura; even if the operation did not prolong life it might greatly relieve the dyspnœa.

Sequelæ.—Every form of disease producing obstruction of the lower air-passages is liable to be followed by inflammatory changes in the pulmonary tissue, as well as in the walls of the air-passages themselves. Thus when an aneurysm has pressed upon the trachea, or upon a bronchus, the mucous membrane has repeatedly been seen ulcerated, and some of the cartilages exposed and partially detached, even though there may have been no indication of an approaching rupture of the sac. Stenosis of a bronchus, from whatever cause, is not infrequently accompanied by dilatation of its branches within the lung. Purulent fluid is apt to accumulate in them, and the result is the occurrence of more or less extensive pneumonia, which often goes on to gangrene. A foreign body fixed in a bronchus often sets up ulceration and sloughing of the part of the tube against which it presses. Sometimes this ends in perforation of the pleura, with pneumothorax, and the foreign body itself may become loosened and fall into the serous cavity. Sometimes it leads to pneumonia, which may spread from the root of the lung far into its substance. The occurrence of fœtid expectoration, and the development of the physical signs of hepatisation may reveal these various changes, but in some cases they are first detected in the *post-mortem* room. Even after expulsion or removal of the foreign body, it sometimes happens that the case nevertheless ends fatally as a consequence of the pneumonia that had been set up; but happily this sometimes subsides, and the patient makes a permanent recovery.

With regard to the *diagnosis* from one another of the several affections that may cause obstruction of the lower air-passages, it is worth remembering that the two diseases in the course of which stenosis of the trachea is most apt to occur without the presence of any other symptoms are *syphilis* and thoracic *aneurysm*. When the obstruction is limited to a bronchus,

especially perhaps on the left side, aneurysm is still probable; a mediastinal growth is a more likely cause than a syphilitic stricture. The possible presence of a foreign body must never be forgotten, especially if the symptoms have come on suddenly.

The *duration* of syphilitic disease of the trachea after symptoms have set in ranges, according to Gerhardt, from two months to four years. That of stenosis from compression would probably be found to be confined within comparatively narrow limits of time. Foreign bodies sometimes remain for a very long period—for months, even for years—in the lower air-passages, and yet are after all expectorated.

As to the *treatment* of the various affections that may cause stenosis of the trachea or of the bronchi, there is little to be said. Whenever there is a possibility that it may be due to syphilis, mercury and iodide of potassium should be actively employed. Gerhardt relates, in vol. ii of the 'Deutsches Archiv,' the case of a man, aged thirty-six, who had had constitutional symptoms after a hard chancre eight years before, and who consulted him on account of cough with scanty muco-purulent expectoration, a tickling sensation behind the sternum, a little alteration of voice, and slight interference with the breathing. These symptoms had been present for about six months. The patient had lost flesh, and his face was somewhat puffy and livid. Nothing could be discovered with the laryngoscope, and only râles behind the manubrium with the stethoscope. A permanent cure was effected by the administration of full doses of iodide of potassium during several weeks. Unfortunately, however, the cases which are usually seen, where cicatricial bands and ridges have already been formed, appear not to be amenable to anti-syphilitic remedies.

When there is a foreign body in the air-passages, the only proper course is to perform tracheotomy at once. Until this has been done, it is not safe to place the patient head downwards on the chance that the body may fall out through the glottis, as happened (but after tracheotomy) in the case of Mr Brunel, which is so graphically told in Watson's 'Lectures.' Nor does it appear to be prudent to administer an emetic; for the body, if dislodged from its position in a bronchus, may become impacted in the larynx and cause suffocation.

DISEASES OF THE LUNGS

SYMPTOMS AND PHYSICAL SIGNS

"Who knows but that, as in a watch we may hear the beating of the balance, and the moving of the wheels, and the striking of the hammers, and the grating of the teeth, and multitudes of other noises—who knows, I say, but that it may be possible to discover the motions of the internal parts of bodies (whether animal, vegetable, or mineral) by the sound they make; that one may discover the works performed in the several offices and shops of a man's body, and thereby discover what instrument or engine is out of order."—ROBERT HOOKE, 1667.

PERCUSSION—*History—Methods—Terminology—Physical theory—Tympanitic resonance—Amphoric and cracked-pot sounds—Diagnostic significance.*
AUSCULTATION—*History—Methods—The respiratory murmur—Bronchial and tubular breathing—Physical explanation—Râles and rhonchi—The vocal resonance in health—Bronchophony—Pectoriloquy—Ægophony.*
Palpation—Tactile vibration—Inspection and measurement of the chest.
COMMON SYMPTOMS—*Dyspnœa—Varieties—Phrenic dyspnœa—Cheyne-Stokes breathing—Cough: reflex, faucial, gastric, and cerebral—Pleurodynia.*

THE modern era in medicine was begun in the seventeenth century on its theoretical side by Harvey, on its clinical side by Sydenham; but just as anatomy and physiology languished from the close of the seventeenth till the third decade of the nineteenth century, so medicine made slow and uncertain progress until the close of the war which followed the French Revolution. Indeed, certain facts known to the Greek physicians were unknown at the beginning of the present century.

In the fifty years between 1815 and 1865 three great advances were made in the clinical investigation of disease—the discovery of mediate auscultation by Laennec, the recognition of Bright's disease, and the invention of the ophthalmoscope and laryngoscope. To these may perhaps be added the discovery of the reaction of degeneration and the application of electricity to the diagnosis of nervous disorders—not to their treatment, for that was much earlier and far less important.

Of these advances the earliest and the most considerable was the introduction of the physical examination of the chest. This was not entirely unknown to the ancients, and Auenbrugger had made important steps in this direction.

The mere invention of a mode of listening to sounds within the chest might have remained a curiosity, and has more than once led to learned trifling; what made it fruitful was its association with morbid anatomy. The great merit of Laennec (as of Bright afterwards) was that he constantly followed up his researches in the wards by dissection in the deadhouse. Hence, notwithstanding his premature death (by one of the diseases which he elucidated), he left the main outlines of the detection of pulmonary diseases with few omissions to supply and fewer errors to correct.

PERCUSSION.—This method was discovered in the middle of the last century by a Viennese physician, Auenbrugger, who published in 1761 his '*Inventum novum, ex percussione thoracis humani, ut signo, abstrusos interni pectoris morbos detegendi.*' This method, however, seems to have been adopted by Stoll alone among contemporary physicians of eminence, and it had passed into complete oblivion when Corvisart, in 1808, brought out in Paris a translation of Auenbrugger's work, with commentaries of his own, based upon extensive practice at the Hôpital de la Charité. Percussion was adopted by Laennec, and introduced into this country in 1825 by Sir John Forbes.

The way in which Auenbrugger performed percussion was by striking the chest directly "with the tips of the fingers, brought close together and stretched out straight." He also directed that either a glove should be worn, or that the patient's shirt should be kept on, so as to avoid a slapping noise. Even now the bent fingers of one hand, with their tips brought to a level, are sometimes used to ascertain roughly the state of the back of the lungs; but for more delicate percussion such a method is inadequate.

In 1828 Piorry, who was afterwards physician in the Charité at Paris, published a work upon what he termed "mediate percussion." This consisted in the use of a thin plate of ivory called a *plessimètre*, to be held by the left hand in contact with the surface of the chest, while a tap was given to it with the tip of the right forefinger, or with the tips of the fore and middle fingers. A hammer, which is known as a *plessor*, was afterwards added. On the Continent these instruments are more or less employed, and they are sometimes useful for teaching a class without hurting the patient. But in this country the usual practice is to simplify Piorry's method by using the left fore or middle finger in the place of a plessimeter. It is curious that in his original work Piorry himself speaks of that plan as having been already adopted by certain English and American physicians who had attended his lectures.

It must not be supposed that percussion is easy. On the contrary, a great deal of practice is necessary to enable one to obtain correct results; and clinical clerks commonly continue to make blunders in percussing long after they have mastered the difficulties of auscultation. The blow should come from the wrist; it should be sharp and sudden, so as not to damp the sound which is produced, and the fingers at the moment of striking should be as nearly vertical as possible. The amount of force that should be employed, and the extent to which the finger should be raised before striking, vary with the thickness of the soft tissues over the part of the chest which is to be struck; and every physician, even without being aware of it, modifies his manner of percussing in different patients and upon different regions of the chest as experience has taught him, in order to elicit the best possible sound under various conditions. As a rule, percussion can be practised by a skilled observer without causing any sensation that is complained of by the patient as being painful; but in delicate women, and even in some very thin men, the sternum and the ribs may be so exquisitely tender that a satisfactory sound can be elicited only with great difficulty. Sometimes cough is excited by every attempt at percussion, and a forcible blow may even lead to blood-spitting, so that it is well to abstain as far as possible from this method of examination when there has been recent hæmoptysis. In such cases, and generally with children, it is better to begin with auscultation and inspection.

We must not imagine that in different persons one can always elicit the same sound by percussion of the chest if the organs are healthy; nor, again, that the sound ought to be the same over different parts of the same chest. But, in health, the range of percussion-sounds is almost limited to a simple series. One extreme is heard where a thick mass of solid tissue lies behind the ribs, as over the centre of the heart just below the fourth left costal cartilage, or over the liver between the right seventh and eighth ribs. This sound is said to be "flat" or "dull."* It is, however, less absolutely dull than the note which is heard on percussion on the fleshy mass of the forearm or thigh, and seldom so dull as the note obtained by percussion over a solid lung or liquid effusion in the pleura.

The other extreme is heard where the ribs cover a thick substance of lung, as in front below the clavicles, in the axillæ, or behind below the scapulæ. This sound is commonly called "clear" or "resonant."† Both sounds are difficult to describe, but they are easily recognised in practice.‡

Between the extremes of dulness and resonance there are all gradations, for which the expressions "partial dulness," "incomplete resonance," "muffled resonance," are employed. These varieties of percussion-sounds are heard at the borders of a resonant or dull area, especially where the lung overlaps a solid organ with a thin edge. At such points many different sounds are obtained, according to the amount of force used in striking. A gentle blow elicits a sound only from the parts immediately below the spot which is struck; a more forcible blow affects deeper parts as well. Thus it is usual to speak of "superficial" and of "deep percussion." But it must be remembered that in "deep percussion" the sound is also modified by the structures which are laterally adjacent, so that a resonant area interferes with the sound yielded by a dull area on forcible percussion, although a dull area does not interfere with the sound yielded by a resonant one. For example, by "deep percussion" over the heart one can often elicit a more or less resonant sound even when it is not covered by lung; but on deep percussion over the lung it is not possible to detect any degree of dulness, however slight, as the result of the presence of the heart or of the liver by the side of the lung. When a solid organ is overlapped by lung, it is often necessary to employ deep percussion as the only means of detecting the distance to which the solid organ extends. But under other circumstances superficial percussion should be used when the object is to map out the relative positions of the viscera. It is otherwise when one wishes to determine the presence or absence of disease of the pulmonary tissue. In such cases one has to ascertain by repeated trials what amount of force is necessary to bring out an altered percussion-sound most distinctly.

As a rule, it is by comparing the two sides of the chest together in the person under examination, rather than by an absolute standard, that one judges of the results obtained by percussion. But if both sides are diseased this method may altogether fail; and even when only one side is diseased it is necessary to have some kind of standard, since without it one could

* Dulness on percussion: *Sonus obscurus; Fr.* Matité; *Germ.* Dämpfung.

† Resonance on percussion: *Sonus clarus; Fr.* Son clair; *Germ.* Sonorer oder heller Schall.

‡ It is important to bear in mind that "resonance on percussion" means altogether a different thing from what is called *resonance* in acoustics. This conventional way of using a scientific term would no doubt have been better avoided, since it is apt to lead to confusion; but in England it has been universal, and can scarcely now be altered.

not tell, by percussion alone, on which side the disease lay. Now, as already stated, the percussion-sound in health is different in different individuals. The thinner a man is, the more "resonant" his chest is likely to be; in very muscular or very fat persons it is sometimes difficult to elicit a clear sound anywhere, particularly over the backs of the lungs.

The differences in percussion-sound at different parts of the chest in health depend upon obvious anatomical conditions, but they nevertheless require careful study in actual practice. In front the sound is modified by the position of the liver and of the heart on the right and left sides respectively; and immediately below the clavicles it is more resonant near the sternum than it is further outwards. Behind, in the supra-scapular regions, one sometimes has to use a good deal of force in order to elicit anything but a dull sound and even between the scapulæ the sound generally becomes gradually more resonant as one passes downwards. Below the scapulæ the sound is generally almost as clear as under the clavicles; the resonant area on the left side extends about a finger's breadth lower than on the right side where the liver encroaches on the thorax. While the back is being percussed the patient should have his shoulders drawn forwards as much as possible, crossing his arms over the chest. In this way the "interscapular region" is increased and the "scapular" regions are much diminished.

Since the infra-clavicular regions are frequently the seat of phthisis and the basal regions of pneumonia or pleural effusion, the axillæ are the safest regions for obtaining the normal resonance of the lungs in any individual case; and the right side is preferable, because a distended stomach sometimes gives a tympanitic quality to the axillary note on the left.

We said that the variations of the percussion-sound in health are *almost* limited to a simple series. The qualification was necessary for two reasons. When the sternum, the clavicle, or one of the ribs is struck, a high-pitched tone is added to the sound, giving it what is termed an "osteal" character. In an infant, especially when it is screaming, one sometimes elicits what will presently be described as a "cracked-pot sound."

With regard to the theoretical interpretation of percussion-sounds there have been great differences of opinion. Even now scarcely any two writers seem to express the same views; and the variations in nomenclature are most confusing. Dr Walshe insists that the use of the terms "dull" and "resonant" (or "clear") is inaccurate, because they represent conditions which, instead of being "simple, are in reality made up of several elements, capable of separate analysis." But the analysis which Dr Walshe himself has attempted throws less light on the subject than we might expect. The view which appears the most satisfactory—if we may assume its theoretical accuracy, as to which few are competent to form an opinion—is that stated by Dr Gee. He declares that the terms in question are capable of scientific definition. According to him, dulness means *absence of tone;* a part is dull when the sound which it yields on percussion is a mere noise, without that regular succession of vibrations which constitutes tone. On the other hand, varying degrees of clearness or resonance correspond with the admixture, in different proportions, of noises and tones. No percussion-note is ever perfectly pure; and thus, beyond the resonance yielded by the healthy chest of even the thinnest person, there are "over-resonant" sounds which may accompany certain diseases.

Another question upon which writers differ is as to the seat of the vibrations causing the tone which is elicited by percussion over the healthy chest.

Dr Gee refers this tone to the middle-sized and largest bronchia; following Wintrich, he thinks that the pulmonary vesicles and the bronchioles are too small to yield it. But it certainly seems to be a great obstacle in the way of the acceptance of that view that in bronchitis resonance is seldom, if ever, impaired, however completely the tubes become filled up with pus or mucus. At least the *noise* or toneless part of a percussion-sound must, one would think, be due to vibrations of the thoracic walls, seeing that its amount, in proportion to that of the tone, varies with their thickness and with the extent to which they are loaded with fat; and Dr Bristowe believes that the whole of a percussion-sound is "mainly due to the vibration of the thoracic walls alone." He assumes that "so much of each half of the thorax as bounds lung-tissue vibrates bell-like when any part of that half is struck; and that the impure musical sound which is elicited comprises a fundamental tone due to the vibration of the whole or a large portion of the side, and harmonic tones due to the vibration of aliquot parts of it." It is a strong argument in favour of this doctrine, that deformity of the chest, without any apparent thickening of the parietes, may give rise to absolute dulness on percussion, notwithstanding that the lung beneath is quite healthy: when there is lateral curvature of the spine, for example, the rounded projection formed by the ribs on one side of the back generally yields a dull sound. Another argument is afforded by a peculiar alteration in the percussion-sound beneath the clavicle, known as Skoda's resonance, which is observed when a certain quantity of fluid is present in the corresponding pleura. For no explanation seems so satisfactory as that suggested by Dr Bristowe, namely, that the vibrating area is diminished, and that consequently it yields a fundamental tone which is raised in pitch. But the subclavicular percussion-sound is not affected in the same way when there is pneumonia of the lower lobe of the lung. So that one must assume, on Dr Bristowe's view, that hepatisation of the lung, notwithstanding that it gives rise to a more or less dull sound when percussion is made directly over it, does not, like pleural effusion, completely damp the vibration of the corresponding part of the chest wall and prevent it from joining with the rest of the side in emitting a tone when a distant part is struck.*

We have already spoken of the chest as being sometimes *over-resonant*—a better term than *tympanitic*, which has often been employed as meaning the same thing. Originally a percussion-sound was called tympanitic (*i. e.* like the note of a drum) when it was such as would be yielded by an abdomen in which the intestines are distended with gas; for tympanites has, since before the days of Hippocrates, been a name for that condition. And, as a matter of fact, the sound generated by percussion over an emphysematous (or over-distended) lung, when the chest walls are rounded and thin, often approaches, if it does not actually reach, this tympanitic quality.

Dr Gee classifies percussion-sounds containing adventitious tones as follows, according to their *pitch*. Those which are highest pitched he calls *Osteal*, after Piorry, because they are yielded by the hard solid tissues, cartilage,

* One point, however, in which it is less easy to agree with Dr Bristowe is his rejection of the distinction between "superficial" and "deep" percussion. He evidently thinks that his doctrine is incompatible with the admission that a percussion-sound can in any way be modified by the presence of solid matter within the chest, except in so far as it is in direct contact with the inner surface of the chest wall. But about the reality of this distinction there is surely no doubt.—C. H. F.

and bone, as has already been mentioned. Those which are somewhat lower were termed by Dr C. J. B. Williams *Tracheal* or *Tubular*, being more or less like the note yielded by the trachea on percussion when the mouth is a little open. Lower still are tones which Dr Gee proposes to call *Subtympanitic;* they are such as are usually yielded by percussion over healthy lungs in their natural state of distension. The lowest pitched tones of all are the *Tympanitic*, and are heard on percussing the healthy abdomen.

Certain modifications of the percussion-sound are noticed under special circumstances; they are the "amphoric note" and the "cracked-pot sound."

The *amphoric percussion-note** is described by Dr Gee as consisting in an "overtone existing either apart and alone, or as an harmonic superadded to the fundamental tone, which itself may either be clear or muffled." It is commonly heard when percussion is made over a stomach distended with air. It may also be elicited by giving a sharp fillip to the cheek when it is blown out to a certain point. It is higher in pitch than an ordinary tympanitic note.†

The *cracked-pot sound*‡ is exactly like the chinking of coins, or the sound produced by clasping the hands loosely together and striking them upon the knee. Probably it always depends upon the propulsion of air out of a space through a more or less narrow opening. It was originally noticed by Laennec, who gave it the name of *bruit de pot fêlé.*

Lastly, it must be stated that, besides the sound which is yielded by percussion and is audible to bystanders, the physician is himself conscious of differences in the tactile sense of resistance offered to his fingers as he taps, and that this is often of considerable assistance in enabling him to draw correct inferences from his observations.

As to the *practical significance* of the various modifications of sound elicited by percussion of the thorax, it is not necessary to say much in this place, since the subject must be considered in discussing each of the diseases of the bronchi, of the lung, and of the pleura. But, in general, it may be laid down that *dulness* on percussion may mean either consolidation of the pulmonary tissue, or its compression and solidification by fluid or otherwise. As a rule, the diagnosis between the causes of a dull note is based upon several considerations independent of percussion. But the dulness resulting from the presence of liquid in the pleura has the peculiarity of being more complete or absolute than that which arises in almost any other way; and the sense of resistance is also greater, especially when the quantity of liquid is large.

A *tympanitic sound* is either due to extreme emphysema of the lung, or, if typical, depends on the presence of air in the pleural cavity. There is, however, a very rare affection, diaphragmatic hernia, which may simulate left pneumothorax by escape of the stomach through the diaphragm into the chest. Moreover, a distended stomach may push up a shrunken lung until what appears to be thorax yields a tympanitic note.

The various combinations of dulness with tympanitic, subtympanitic,

* *Fr.* Son argentin, son métallique, son humérique; *Germ.* Metallklang, Amphorischer (d. h. krugartiger) Schall, metallish-klingender Schall.

† As a drumhead is tightened, its note when struck becomes shorter and shorter and also higher and higher in pitch, but it never loses its resonant ("tympanitic") character which distinguishes it from that produced by striking a solid object.

‡ *Fr.* Bruit de pot fêlé; *Germ.* Zischender oder klirrender Schall (Geräusch des gesprungenen Topfes).

tracheal, and osteal tones are met with chiefly when there are cavities or vomicæ in a part of the lung which is separated from the surface by a thickened and adherent pleura or by a layer of consolidated pulmonary tissue, or when air is present in a pleural cavity of which the walls are indurated. The significance of the amphoric ring and of the cracked-pot sound will be fully considered elsewhere.

Before leaving the subject of percussion it is well to repeat that the student who aims at more than educating his ear to appreciate differences of sound, and learning their empirical significance, must remember that "dull," "resonant," and "tympanitic" are conventional terms, best defined, practically, as those elicited by percussion over the forearm, the right axilla, and the stomach respectively; but that each of these sounds, and the many others to be heard by percussion of the healthy as well as of the diseased body, consist of a fundamental tone with endlessly varied harmonic or discordant overtones, and that each note may vary as follows: (1) in loudness or amplitude of vibrations; (2) in pitch or number of vibrations per second; (3) in duration; (4) in quality or *timbre*.

A clear sound (*son clair, heller Klang*) is opposed to a non-resonant or toneless one (*mat, dumpf*), and a high or treble to a low-pitched or bass; a tympanitic note, on the one hand, to an osteal or sternal sound which, though resonant, is high and very short, and, on the other hand, to a long but completely toneless one; and lastly (according to Skoda), a loud and long percussion-sound (*voller Schall*) to a short and feeble one of somewhat high pitch (*leer*).

This is the most convenient place to mention an effect of "immediate" percussion, which is not infrequently seen in emaciated persons, namely, the production of contractions in muscles which receive the tap, especially in the pectoralis major. A rounded knot rises up at the spot which is struck, and from this a smaller ridge spreads away, wave-like, along the fibres in both directions. This "idiomuscular contraction" has been supposed to be characteristic of phthisis; but it occurs in most wasting diseases, when the muscles are in a condition of physiological "exhaustion."

AUSCULTATION.—As Dr Gee remarks, it is an interesting fact that this, a still more important method of physical examination than percussion, arose directly out of Corvisart's researches. Among those who followed the practice of that professor at the Paris Charité were two friends, Bayle and Laennec. In endeavouring to distinguish between active and passive enlargement of the heart when percussion showed its size to be increased, they were in the habit of carefully observing the character of its impulse; and they became accustomed to apply the ear to the cardiac region for this purpose rather than the hand. One day (about 1815) Laennec was consulted by a young woman who had the general symptoms of disease of the heart, but in whom, as she was fat, he was unable to feel the impulse satisfactorily. For reasons of delicacy he was hesitating to put his head to her chest, when he remembered the fact that by applying the ear to one end of a plank one can hear the scratch of a pin at the other end. So he took a quire of paper, and rolled it very tight. And then, placing one end on the precordial region, and leaning his ear on the other end, he found, to his surprise and pleasure, that he could actually hear the beating of the heart more plainly than when the ear was in immediate contact with the thoracic wall. He soon began

to employ the new method of investigation, which he termed *l'auscultation médiate*, in phthisis and other pulmonary diseases, read a memoir on the subject in 1818, and published his great work in the following year. In 1826 he died of phthisis, at the age of forty-five, having almost worked out the subject so far as concerns affections of the lung.

Appreciating the importance of combined percussion and auscultation, Laennec was also well acquainted with the morbid anatomy of the diseases to be recognised. He showed how to distinguish by physical signs pneumonia, emphysema, pleuritic effusion, and pneumothorax, and he classified and defined the most important variations of the respiratory murmur as well as the chief adventitious sounds to which he gave the general term *râles*. He, like other pathologists at that time, was not aware of the importance of bronchitis (*catarrhe pulmonaire*) as a separate disease, and the very term was still unused; but he described the signs of œdema of the lungs and of phthisis almost as we should describe them now, and pointed out the existence and significance of the curious sound called "metallic tinkling."

It is remarkable that of the very few references to sounds heard by listening over the chest which have been traced in medical writings of an earlier date, two are in the works of Hippocrates, the father of medicine; he mentions the leather-like creaking of pleural friction and the splash obtained by succussion. The only other notice of any sound produced in the lung is, according to Dr Gee, one of pneumonic crepitation in van Swieten's 'Commentaries,' published in 1774.

The stethoscope.—In examining the backs of the lungs we still sometimes apply the ear directly to the surface of the chest, or rather so that only a towel or the patient's night-dress separates one's ear from his skin. In this way one can rapidly judge of the state of a large part of each lung; and it is often more convenient than using an instrument when the person is very ill and has to be lifted up in bed by attendants. But in most cases we employ some form of stethoscope. That which was originally designed by Laennec (*le cylindre*) has long ago been discarded; it was long and heavy, in shape like a general's leading staff or a thick and short ruler, and it was needlessly elaborate in construction.* What is now used is generally a light hollow stem of wood or metal, having a flat ear-piece at one end and spread out into a concavity at the other end, which is placed upon the spot to be examined. In applying it one must be careful not to hurt the patient by pressing too hard or by bearing unevenly upon one side of the rim. The ear must always be moved to the stethoscope, and not the stethoscope to the ear. There is no object in the perforation except for lightness; the sound is conducted by the solid substance, not by the column of air. A very light stethoscope and one made in two parts is so far faulty; a somewhat massive form made in cedar is acoustically better.

Many years ago a stethoscope was introduced in which the chest-piece and an ear-nozzle were connected by a flexible tube, and it is still used by some good auscultators. It is extremely portable, and as convenient as the binaural form for auscultation of the sides and back of the chest without raising the patient.

Recently, however, it has become the fashion to employ a stethoscope with a flexible stem and with two long metal and ivory ear-pieces, one of which is

* The specimen in the Royal College of Physicians in London is about ten inches long and rather more than an inch thick. It is perforated and divided into three parts, so as to make it half the length if desired, and to allow the "obturator" to be taken out or left in.

introduced into the meatus of each ear, and is held in position by an elastic band. To the use of this double or binaural stethoscope, as it is called, everyone should accustom himself, because of the facility with which the back and sides of the chest can be explored by it in persons who are too ill to sit up. Students like the instrument on account of the loudness with which sounds are transmitted through it. But it is not certain that what reaches the ear is always as clear and distinct as when a single stethoscope is employed. And the slightest movement of the flexible part of the binaural stethoscope produces noises which are apt to be perplexing. Other disadvantages of the instrument are that it is cumbersome to carry about, that the least contact with the patient's dress causes a loud noise, that it is almost useless unless applied directly to the skin, and that the natural sounds, as estimated by immediate auscultation, are exaggerated. Its advantages are its convenience of application to many parts of the chest, the exclusion of other sounds by both ears being occupied, its allowing the auscultator to see while he is listening (not always an advantage), and its making both respiratory and cardiac murmurs easier to hear.

The double stethoscope is best used to determine the existence and character of slight or doubtful cardiac murmurs; it is useful for listening to the trachea and vessels of the neck, to the lungs above the scapulæ, and to the abdomen, and it or the single flexible stethoscope is almost indispensable for listening to the lungs of a recumbent patient.

For examining the heart and the front of the chest without removing a patient's underclothing, the ordinary wooden stethoscope is the most suitable instrument.

For listening to the back of the lungs while the patient is standing or sitting up in bed, no form of mediate auscultation is so convenient as the application of the ear to the patient's chest covered by a thin towel.

Whatever form of stethoscope may be employed, one must take care that the patient's clothes do not rest against it nor against the surface of the chest near the spot to which it is applied. And another point to be kept in mind is that if the instrument is placed on the hairy part of a man's chest a crackling sound is often produced which may be very like what will be presently described as crepitation. This difficulty may be avoided by wetting the surface or smearing a little oil over it.

AUSCULTATION OF THE BREATHING.—*The respiratory murmur and its modifications.*—On listening over the lungs of a healthy person, one hears each time he breathes a soft, rustling, breezy sound, followed by a second one of similar quality but of shorter duration and fainter. Of this no further description is needed, since the only way of really learning to know it is to hear it again and again and in many different individuals. It is commonly called the "*vesicular murmur*," having been so named when the idea that it arose in the air-vesicles of the lung was accepted without question; but such an association with a theory which is disputable is an objection to the term. Dr Walshe proposed "pulmonary respiration-sound" instead. "The normal *respiratory murmur*" is perhaps a still better term.* The former part of this sound accompanies the act of inspiration; expiration is either altogether noiseless or attended by the shorter and fainter murmur, almost continuous with the inspiratory.

In some persons the respiratory murmur is much louder than in others.

* *Fr.* Souffle respiratoire, souffle vésiculaire; *Germ.* Normaler Lungengeräusch.

In children it is particularly loud, so that when under morbid conditions an equally intense murmur is heard in an adult, this is sometimes called "puerile breathing." But in thin adults it is often scarcely less loud, even when all parts of the lungs are healthy. On the other hand, there are some people in whom the act of breathing is attended with scarcely any sound, even when one tries to make them breathe deeply. One must therefore listen over different parts of a patient's chest before one draws conclusions from the degree of loudness of the murmur at a single spot. It is naturally louder where the thoracic walls are thin than where they are covered with thick muscular masses, and over the edges of the lung it is less loud than elsewhere; in other words, its intensity in health is generally proportionate to the degree of resonance on percussion at various parts of the chest. In the same way it is feeble when the walls of the chest are thickly covered with fat.

At the bases of the lungs, especially in persons who are confined to bed by whatever cause, the vesicular murmur is sometimes found to be mixed with a crackling sound, which may be mistaken for crepitation, but which disappears when the breath is drawn deeply two or three times in succession, so that it must be due to slight collapse from disuse, and has no clinical significance. On the other hand, there are certain spots at which in many healthy persons the breathing is attended with a sound that differs from the vesicular murmur. They are (1) the space between the scapulæ over an area of variable extent, but somewhat lower in situation on the left side than on the right; (2) the region below each sterno-clavicular joint, especially on the right side and in females, and the part corresponding to the spines of the seventh cervical and first dorsal vertebræ. The sound heard over these parts is harsh, or, as it is often called, "*bronchial* in character," because it is transmitted from the main bronchi. It differs from the vesicular murmur in having a blowing character, in the expiratory part being as loud as the inspiratory, and in there being an interval between them. A similar sound is heard more loudly and constantly on auscultation over the trachea, and still more loudly over the larynx; here it is of a more "whiffing" quality and harsher, and is distinguished as "*tubular*," a term, however, which some writers use as synonymous with bronchial.

To be able, in practice, to distinguish a loud or harsh vesicular murmur from true bronchial breathing is the most essential step in auscultation. For the latter sound, with certain modifications of it, may be heard, in disease, over any part of the chest, and constitutes one of the chief signs of the most important pulmonary affections. In bronchitis, indeed, bronchial breathing does not occur, but it may accompany any disease in which the lung is either compressed, or consolidated, or hollowed into cavities. In other words, its range is generally conterminous with that of percussion-dulness. The modifications of bronchial breathing concern its quality. A blowing character belongs to them all; and they all consist of an inspiratory part and of an expiratory part, separated by an interval. What distinguishes them is that the blowing sound is more or less hollow. In its most extreme form it has an echoing character, and resembles the sound produced by breathing into a large empty glass bottle; it is then named *amphoric*.* When it is whiffing but without a "hollow" quality it is by

* A less marked degree of the same quality of sound is often called *cavernous*, because it is commonly heard over vomicæ, which are sometimes spoken of as caverns in the lung. The term is superfluous and misleading. A vomica may often yield consonating râles, not

most writers termed *tubular*, although others, as above mentioned, employ "tubular breathing" and "bronchial breathing" indiscriminately.*

It must be understood that between these several modifications of bronchial breathing all gradations exist, so that it is often difficult to know whether to call a sound "tubular" or bronchial, tubular or "amphoric." But it is well to maintain as far as possible the distinctions between them. If true tubular (or amphoric) breathing is heard, at whatever part of the chest, there can be no doubt that disease is present, even though the spot is one in which bronchial breathing is sometimes audible in health. It is a good rule to spare the more striking epithets. The usual errors of beginners are to mistake a loud or harsh respiratory murmur for bronchial breathing, to call bronchial breathing tubular, and tubular breathing amphoric.

With regard to the physical causes of the vesicular murmur, of bronchial breathing, and of its various modifications up to amphoric, there has been much speculation. Of late the tendency has been to apply to them strictly the theory of the *veine fluide*. According to this theory, a blowing sound is generated whenever a fluid (whether liquid or gas) passes from a narrow space into a wider one. Now, during inspiration this condition is fulfilled at two points in the respiratory tract: (1) when the air enters the trachea from between the vocal cords; (2) when it emerges from the extremity of each bronchiole into the ampulla formed by the air-sacs around. On the other hand, during expiration, the only point at which a *veine fluide* can be formed is at the upper orifice of the larynx; but since the false vocal cords form a lip on each side, the resulting sound should be audible not only above the spot at which it is formed, but also below. It is obvious that these facts accord perfectly with what has been stated with regard to the characters of the vesicular murmur and of the sound heard on listening over the trachea and primary bronchi. The former belongs mainly to the act of inspiration; the latter is divided into more or less equal parts, of which one attends inspiration and the other expiration. That the vesicular murmur is generated somewhere below the larynx has indeed been demonstrated in a series of experiments, of which an account was given by the author in the 'Med.-Chir. Review' for July, 1873, by certain French observers, especially Bergeon, Chauveau, and Boudet. They cut through the trachea of a horse and drew the lower end of the tube outwards through the wound in the skin, so that no *veine fluide* could possibly arise in it; after this operation they found, on listening over the animal's chest, that the vesicular murmur still remained audible, and that its intensity was little if at all diminished. On the other hand, by dividing the pneumogastric nerves in another horse they succeeded in abolishing the vesicular murmur. The explanation of this is supposed to be that the muscular walls of the bronchioles were paralysed, so that they opened into the air-sacs by wide funnel-shaped mouths—which would yield no *veine fluide*. In this last experiment the laryngeal sounds heard over the trachea are of course still persistent.

amphoric breathing at all. Moreover, the names of physical signs should refer to their physical characters, and not to the anatomical conditions they denote, especially when their significance is not constant.

* *Fr.* Souffles bronchique, bronchillaire, tubaire, glottique, soufflante, caverneuse, amphorique; *Germ.* Bronchialgeräusch, amphorisches Athemgeräusch. *Unbestimmtes Athemgeräusch* answers to the "vesiculo-tubular or subtubular" breathing of some English and the *respiration rude broncho-vésiculaire* of some French authors.

The theory of the *veine fluide*, however, in the form in which it is stated by the French experimenters, does not account for the fact that in most persons expiration is attended with a murmur, although a faint one. Moreover, in pulmonary emphysema the expiratory murmur becomes greatly prolonged and very noisy, while the inspiratory murmur is much diminished.*

The French observers whose views have been quoted suppose bronchial breathing, whether in health or in disease, to consist of sounds generated in the larynx, and transmitted downwards with more or less modification until they reach the ear of the auscultator. Indeed, they seem to have proved this experimentally. A horse was affected with pneumonia, and an intense *souffle tubaire* was audible. Chauveau and Boudet cut through the trachea, so as to allow air to enter the lungs without passing through the glottis, and the *souffle* at once disappeared. It would be of great interest to observe the same point in man, either in cases of cut throat or after tracheotomy; but the requisite conditions can scarcely ever be satisfactorily fulfilled, for unless the orifice into the trachea is as wide as the calibre of the tube itself a *veine fluide* may always be generated, at least during inspiration. Stokes, however, long ago pointed out that when the larynx is diseased it is often difficult or impossible to determine whether the lungs are or are not healthy. And it certainly seems very probable that extensive ulceration, destroying the attachments of the vocal cords, may prevent the formation of a *veine fluide* in the air which passes the glottis, and so render impossible the development of bronchial breathing in diseased lung below.

Of the modifications of bronchial breathing, from tubular to amphoric, all that need be said in this place is that the more marked the hollow quality the more one is justified in asserting that a space filled with air, of larger size than even a main bronchus, has been formed in the substance of the lung—unless, indeed, the pleural cavity itself should contain air. But even in acute pneumonia, when there has not been the slightest destruction of pulmonary tissue, it is surprising how hollow the bronchial breathing sometimes is; and Dr Walshe speaks positively of having heard sounds to which he would assign the name of "cavernous" in cases, whether of pneumonia or of pleurisy, in which there was no excavation, and in which the lung was only consolidated or compressed over large bronchial tubes.

Adventitious respiratory sounds.—The sounds derived from auscultation hitherto described have been sounds which are identical with, or more or less modified from, those that can be heard on listening over the healthy lungs or air-passages.

There are other sounds which are adventitious, and have no physiological representatives. Thus in pleurisy a *friction-sound* is heard, which has its origin on the surface of the lungs; this will be discussed in the chapter on pleurisy. With regard to the adventitious sounds which are found within the lungs there has unfortunately been much confusion of names. Some writers describe them all indifferently, either as "Râles" or as "Rhonchi;"

* These difficulties may be met as follows. It is a very slight extension of the results obtained by Bergeon, in experimenting with a tube provided with a lip or rim where it was narrowed, to assume that a lip, at the orifice of a contracting cavity, would generate a *veine fluide* in the cavity itself. Now, in emphysema it is fair to suppose that the mouth of the bronchiole projects some distance forwards into the space formed by the dilated air-sacs around it; and even under normal conditions it may do so sufficiently to produce the faint expiratory murmur which is heard in healthy persons.—C. H. F.

the former term was introduced by Laennec, and rendered into English as "rattle" by those who introduced his views into this country; the latter is used as synonymous in Germany. According to this use of the words, each is applicable indifferently to two kinds of sounds, which are very unlike one another, and which may be distinguished by those of the one kind being *continuous* and blowing, whereas those of the other kind are *interrupted*, and "crackling" or "bubbling" in character. Very often the two kinds are spoken of as being respectively "dry" and "moist;" but this is better avoided, for reasons which will presently appear.

There is, however, another meaning of the term *rhonchus*,* which seems to have been given it by the late Dr Latham, of St Bartholomew's Hospital, and which afterwards was sanctioned by Sir Thomas Watson. In that sense it is limited to continuous or "dry" sounds, or even to a single variety of dry sound. This is what is called sonorous rhonchus, the other dry sound being aptly termed *sibilus* or sibilant rhonchus. Both these sounds belong mainly to bronchitis. *Stridor* is a rhonchus so loud as to be audible at a distance.

If the two continuous (or "dry" and blowing) sounds are to have each a name of its own, the term *râle*† may conveniently be confined to interrupted ("moist" or crackling and bubbling) sounds. They occur in almost every disease to which the lungs or the air-passages are liable. They are generally attributed to the disturbance of fluid lying in the bronchial tube by air bubbling through it; and on that account they are called moist sounds. Each crack is supposed to denote the bursting of a bubble. But there have long been observers who maintain a different opinion, and ascribe them to the sudden separation of surfaces that had been in contact. In 1871, Traube, in the 'Berliner med. Wochenschrift,' applied this view to explain the râles heard in the larger tubes by the momentary detachment of portions of viscid mucus from their sides, by the current of air in respiration. It is difficult to conceive that in such thick secretion as is often found in the air-passages bubbles can be made and broken with sufficient frequency to account for the abundant râles that are often heard; and, as Traube remarks, râles are often to be heard in cases of pleuritic effusion, when there is not the least reason to suppose that any fluid is present in the tubes, and when therefore they can only be attributed to the separation of the sides of tubes that had been forced against one another in compressed parts of the lungs. He says that he has several times determined the absence of fluid in such cases at an autopsy. A further point to which he draws attention is that a râle can be produced by pressing gently with the stethoscope upon the surface of a healthy lung of a recently killed animal. Wintrich has shown that a like result may also be brought about by inflating the collapsed lungs after death ('Virchow's Hdbh.,' Bd. v, Abth. 1).

If Traube's explanation is correct, it will also apply to the moist pleuritic and pericardial rubs which closely resemble "mucous râles." There is, however, no doubt that the larger "bubbling" râles are produced by bubbles bursting either in the bronchi or the trachea.

Râles are somewhat arbitrarily distinguished according to the supposed size of the bubbles which produce them, or rather of the spaces in which they are found.

* Rhonchus (ῥόγχος or ῥῆγκος, snoring) is Laennec's *râle crépitant sonore; Germ.* Knarren, Schnurren.

† The French word râle or *rasle* corresponds to the German *Rasseln*, and our *rattle*.

The gradation is sometimes made thus: *fine* (or small)—*submucous*—*mucous* râles, and then *gurgling;* but "mucous" is a bad epithet, for the fluid causing the râle may be pus or blood. "Small," "medium," and "large" râles are sufficiently descriptive terms.

In some cases râles, like rhonchi, are audible without the aid of the stethoscope. In fact, the "rattle in the throat" is only a large mucous râle.

Consonance.—There is another distinction between different kinds of râles which is of far greater importance than that of their apparent size, for it indicates whether the tubes in which they are found are surrounded by spongy or by solid lung. In the latter case they have a peculiar quality which in the former is wanting, and which the ear easily recognises, although to describe it in words is difficult. We may call the râles heard when the lung is solid "bright," "clear," "musical," "ringing," or "metallic." Skoda called them "consonating" râles, but we must not assume that their peculiarities depend upon *consonance* in the strict acoustic sense of the term (p. 840, *note*).

Consonating râles are associated with bronchial or tubular breathing, with dulness on percussion, and with the equivalent vocal sign called bronchophony.

The sounds which are termed crepitant râles (or crepitation) and subcrepitant râles are "smaller" (*i. e.* the interruptions are more frequent and shorter) than those of gurgling; and they differ from toneless or non-consonating râles of the same "size" or degree by their "bright" or metallic quality. One particular kind of râle, almost (if not quite) peculiar to an early stage of acute pneumonia, is termed *fine crepitation.* This sound is consonating and typically bright or musical, and it is never perfectly heard except when the lung is hepatised. It sounds less "moist" than any other râle. It will more fully be described and its origin discussed hereafter.

Non-consonating râles, chiefly heard in bronchitis, need no further designation than large, small, or medium.*

AUSCULTATION OF THE VOICE.—If, while listening to the chest of a healthy man, we tell him to speak, we generally hear an indefinite humming or buzzing noise. In a woman the sounds come to the ear more sharply, but one is still unable to distinguish the words that are uttered. Only over the upper part of the interscapular region on each side is there in some persons a space in which one can hear the voice clearly, and in which the separate words are perceived almost as they are when one places the stethoscope over the larynx and trachea, though less loudly.

* To every simple classification of râles and rhonchi that has as yet been proposed there are two insuperable objections. One is that in endeavouring to convey to other persons definite ideas as to the auscultatory phenomena observed in a case of pulmonary disease, or in receiving from them the same kind of information, we are at once baffled by uncertainty as to the sense in which we or they employ different terms, unless we are familiar with the various meanings that are assigned to them by practical men. The other objection is that in studying medical literature, as soon as we pass beyond the scope of a single text-book we fall into the gravest mistakes if we imagine that the language used by different writers has always the same sense. And as for the future, all experience goes to show that if the most distinguished physicians of the day were to meet week after week until they adopted a uniform terminology, and agreed to impose it on others, nearly every one of them would within a year forget in his own practice to make use of it.—C. H. F.

The attempt was made at the International Medical Congress at Copenhagen in 1884, with what degree of success remains to be seen.

Now, in disease the voice may be carried to any part of the chest thus distinctly, and may be heard far more loudly than is ever the case in health. For this "increased vocal resonance," as it is often termed, Laennec invented two names according to its degree of intensity. When the voice, however distinct, gave the impression of still coming from a distance he spoke of "*Bronchophony;*" when it appeared as though it were spoken from the chest immediately into the stethoscope he used the term "*Pectoriloquy.*"*

Bronchophony, as a rule, is associated with bronchial or tubular breathing or with consonating râles; pectoriloquy with tubular or amphoric breath-sounds, or with metallic gurgling. In other words, bronchophony attends those conditions in which the pulmonary tissue is solid; pectoriloquy is heard when, in addition to solid lung, there is a large empty cavity. Occasionally tubular breathing and bronchophony may be heard over a part of the lung consolidated by pressure of fluid in the pleura.

Laennec laid more stress upon auscultation of the voice than of the breathing; the first section of his great work is headed "Exploration de la Voix." Pectoriloquy was the firstfruits of his great discovery, and he did not introduce the term bronchophony until after the first edition of his work; hence, as Dr Gee remarks, it is not surprising that he always clung to this sign with peculiar affection. It became his object to define it in such a way that it should become an unerring indication of a vomica. For this purpose he added to the characters of what he termed perfect pectoriloquy that of being limited to a very small part of the chest. But subsequent experience has shown that, like other "hollow" sounds, the most typical pectoriloquy may in exceptional instances be heard when there is no large cavity within the thorax. Thus pectoriloquy no longer carries the exaggerated importance which Laennec attached to it; indeed, one modern writer, Guttmann, omits it altogether, and includes all degrees of increased vocal resonance under the term bronchophony.

An attempt has of late been made to confine the term pectoriloquy to the sound of the whispering voice. It has long been known that an increase in the vocal resonance is often better appreciated when the patient whispers than when he speaks aloud.†

* "En me livrant à des recherches comparatives relativement à la résonnance de la voix chez plusieurs sujets sains et malades, je fus frappé par un phénomène tout-à-fait singulier. . . . . Lorsque, tenant le cylindre appliqué au dessous de la partie moyenne de la clavicule droite, je faisais parler la malade, sa voix semblait sortir directement de la poitrine et passer tout entière par le canal central du cylindre. Ce phénomène n'avait lieu que dans une étendue d'environ un pouce carré. Dans aucun autre point de la poitrine on ne trouvait rien de semblable" ('De l'auscultation Médiate,' tom. i, § 22).

† Dr Bristowe proposes by bronchophony to understand that tones generated in the larynx—by pectoriloquy that the articulate sounds formed in the cavity of the mouth—are conveyed downwards to the stethoscope with abnormal distinctness. According to this way of using the terms, when the patient speaks aloud, and when the words he utters are clearly perceived by the auscultator, both bronchophony and pectoriloquy are heard at the same time. Dr Bristowe's proposal has the advantage of introducing a real distinction into the meanings of the two terms in question, whereas hitherto the difference has been only one of degree. But its adoption would render useless, except to those who kept themselves well informed of changes in the sense of words, all the literature of the subject during the last sixty years. Moreover, it is not suggested that transmission downwards of the whisper enables any conclusion as to the state of the lung to be drawn which cannot be drawn from ordinary bronchophony. The practical importance of whispered bronchophony lies in the fact that its presence is so easily recognised, and is thus a valuable sign when there is difficulty in detecting increased resonance in the ordinary voice.

The answer to the question *why* bronchophony and pectoriloquy should be heard when the lung is solid and not when it retains its spongy structure, involves also the explanation of bronchial breathing, of consonating râles, and of the loud transmission of the cardiac sounds to distant parts of the chest. The complete physical explanation has not yet been given,* but the differences in the sounds under discussion are readily appreciable by everyone who takes the trouble needful to educate his ear; they mutually check and confirm one another; and long experience has given us confidence in assigning to them a diagnostic significance on which we can depend.

One modification of vocal resonance was named *ægophony* by Laennec, on account of its resemblance to the bleating of a goat.† It will be discussed under pleuritic effusion, of which it is a sign. Its physical explanation has been thus stated by Dr Stone.

In a course of experiments with a pitch-pipe placed between the lips of various patients and made to utter a musical note by their drawing a deep inspiration, he found that when the spoken voice, or even the whisper, yielded marked ægophony to a stethoscope placed over the affected part of the chest, there was no transmission of the sound of the pitch-pipe. Moreover, if the patient could be made to sing or to intone a good musical note, no ægophony was to be heard. Among spoken words a difference was found according to the vowel sounds they contain. The French A yielded hardly any ægophony; it was more marked with the E, still more so with the I, and most of all with U. Now, as is well known, Helmholtz showed that the different vowel sounds are formed by the addition of certain harmonic overtones in varying degrees of intensity to a fundamental tone which may be the same for all of them. And according to Dr Stone the cause of ægophony is that the fundamental tone is intercepted in its passage through a layer of pleural exudation, while the overtones are allowed to pass and, being heard by themselves, give the peculiar character to the sound. In some further experiments he succeeded in imitating ægophony. This was effected by transmitting the voice through a wide india-rubber tube, over which was placed a bladder containing water.

* Laennec was content to assume that spongy lung was a bad conductor of sound. Skoda, as the result of experiments on the dead tissues, declared the conductivity of hepatised lung to be *less* than that of the healthy organ; but he could not reproduce the conditions under which auscultation is practised during life, and it is difficult to believe that a homogeneous material does not convey sound better than a series of spaces containing air. Thus almost all recent writers reject Skoda's conclusion. He maintained that *consonance* is the cause of bronchial breathing and bronchophony. In acoustics, however, consonance has been understood to mean a power of vibrating in unison with some particular tone, or of producing sounds in harmonic relation to it. And in his work on the subject Skoda does not really limit his application of the term to consonance in a strict sense. He alludes to the effect of the sounding-board of a violin, and to the increased sound produced by a tuning-fork when it is placed upon a table. But these are examples of what writers on acoustics have termed "resonance" rather than of "consonance." In Tyndall's 'Lectures on Sound' nothing is said about consonance.

† "*De l'égophonie ou de la pectoriloquie chevrotante.*—La voix plus aiguë, plus aigre que celle du malade, et en quelque sorte *argentine*, produit seulement une illusion telle qu'il semble que quelqu'un parle dans la poitrine du malade. Elle a, d'ailleurs, un caractère constant d'où j'ai cru devoir tirer le nom du phénomène; elle est en quelque sorte tremblotante et saccadée comme celle d'une chèvre, et son timbre se rapproche également de celui de la voix du même animal. Ce caractère ne présente que des variétés légères dont on peut se faire une idée exacte en se rappelant l'effet que produit un jeton placé entre les dents et les lèvres d'un homme qui parle, celui de la voix transmise à travers un roseau fêlé, ou le bredouillement nasal des bateleurs qui font parler le fameux personnage de tréteaux connu sous le nom de *polichinelle*" ('Ausc. Méd.,' tom. i, § 154).

When a stethoscope was applied to the upper surface of the bladder an ægophonic twang became exceedingly distinct.*

PALPATION.—By applying the hand to the chest we are able to compare the extent of movement in respiration of the two sides and of the upper and lower parts of the same side. This use of the sense of touch corrects or confirms the observations of the eye.

Moreover, when the hand is placed upon the bare chest of a healthy man a tremulous sensation is felt when he speaks, especially if the voice is powerful and low in pitch. The sensation is more "voluminous" (because felt over a large space), but in quality closely resembles that of the *frémissement cataire* to be described as a sign of cardiac disease. In a woman or a child it is often not to be perceived, and on the right side it is almost always more distinct than on the left.

This normal sensation is known as tactile vibration of the voice, or *vocal fremitus*. It is increased when the lung is solidified, and is diminished or abolished when the lung is separated by liquid from the thoracic walls. In other words, it is increased under the same conditions as vocal resonance on auscultation, and diminished or absent when the vocal resonance and respiratory sounds are rendered feeble or entirely lost. In estimating tactile vocal fremitus the whole hand should be laid flat on the patient's chest, and he should be directed to speak loudly, slowly, and in as deep a tone as the compass of his voice will allow. As with other physical signs of the chest, comparison of the two sides is invaluable for bringing out the diminution or exaggeration of tactile vibration.

The practical importance of loss of tactile vibration seems to have been first pointed out in the 'Journal Hebdomadaire' for 1829 by the French physician, Raynaud, who also discovered pleuritic fremitus.

INSPECTION.—A rough estimate of the shape, size, and movements of the chest is naturally made when one first glances at the patient after he has stripped. But accurate observations are often postponed until after one has listened to the breathing over different parts of the lungs.

It is, however, better, as a rule, when a complete examination is to be made, to begin by carefully inspecting the thorax as the patient sits upright and in as easy a posture as may be before the physician. The light should be made to fall first full, and then obliquely on the chest. The shoulders should be covered by a shawl while the front is examined. The number of respirations in a minute should be counted, their rhythm, and especially the length of expiration and the degree of pause between expansion and contraction noted, and the relative movements of the chest and abdomen (denoting the predominance of thoracic or phrenic respiration) should be observed. Next the chest should be looked at in front, behind, laterally,

* Dr Stone's theory of ægophony seems to me to be fully established. And it is of the more interest because it brings into complete accord with the auscultatory phenomenon another physical sign, which (so far as I am aware) had never been thought of in connection with it, but which has long been known to be one of the chief indications of pleuritic effusion, namely, loss of tactile vibration. When the pleura contains fluid the fundamental tone, according to Dr Stone, is intercepted; and this is the one which would be felt under normal circumstances, whereas the overtones consist of waves too rapid to be perceptible to the touch. I am not sure, however, that loss of tactile vibration is to be detected in every case when the voice has an ægophonic character.—C. H. F.

When the effusion in the pleura is large, tactile vibration is absent, and also ægophony; the harmonic overtones, as well as the fundamental tone, of the voice are both cut off.

and by looking down upon it from above the patient's head as he sits, in order to measure its relative dimensions, and to compare the movements of the two sides.

Bulging or flattening of one of the infra-clavicular regions is more easily detected by standing behind the patient while he is sitting, so as to look downwards over his shoulders. A general enlargement of one side is often easily appreciable by the eye. But one must make quite sure that the patient is sitting or standing perfectly upright, especially if the case is that of a child or of a young woman with a thin flexible spine. In infants one may grasp the chest with the two hands from behind, placing the thumbs tip to tip upon one of the vertebræ. In adults a measuring tape is often used.

But, as Dr Gee remarks, circumferential measurements are apt to be fallacious, because considerable increase in the sectional area of one side of the chest may leave the length of the periphery unaltered, by "the passage of the elliptical form into the circular." It is this which renders his *cyrtometer* so useful an instrument.* It is made of two long pieces of very narrow metal gas-tubing, of an eighth of an inch in diameter, which are fastened together by a short piece of caoutchouc tube slipped over their ends. The central caoutchouc piece is placed over the spinous process of a vertebra, and the hollow metal rods are then carefully bent round the patient's body, so as to meet over the sternum. It is now easy to remove them without altering their shape; and by laying them upon a sheet of paper one can obtain an accurate tracing, which shows exactly the configuration of the two sides of the chest, and enables them to be compared. One must not forget that the half-circumference of the chest on the right side is in many healthy persons greater than that on the left side, the difference being sometimes as much as an inch.

Various instruments, called *stethometers*, for the measurement of the movements of the chest have been devised by Gibson (1848), Quain (1858), and other physicians. One of the best was Dr Arthur Ransome's, of which a description may be found in the 'Medico-Chirurgical Transactions' for 1873. But although they have yielded information as to the exact degree of impairment of mobility of different parts of the chest wall in various diseases, it seems doubtful whether any one of them has been employed in actual practice by other observers than their inventors, the reason being that they are troublesome to use, and that they bring to light no facts that may not be ascertained without them.

A similar remark applies to the exact mensuration of the several diameters of the chest carried out by means of calipers, with a graduated quadrant, devised by Woillez, Wintrich, and other physicians.

Nor does it appear that in clinical practice any results worth speaking of can be attained by the use of an instrument invented by Dr John Hutchinson many years ago for the purpose of measuring the amount of air that can be expelled from the chest by the fullest possible expiration. This instrument, which is called the *spirometer*, may possibly be of value in examining recruits for the army, or "lives" for insurance. But there are great practical difficulties in obtaining correct results; very few persons succeed in "blowing" their full amount of air into the instrument until they have had some practice.

Common symptoms.—There are certain symptoms which belong to most

* Somewhat similar cyrtometers were invented by Woillez and by Björnström, and are much used in France and Germany.

diseases of the Chest, and which are most conveniently treated of in general here. They are Dyspnœa, Cough, and Pain.

DYSPNŒA.—The use of this term is commonly limited to cases in which a sensation of "shortness of breath" is experienced, with more or less discomfort or distress. But it is better to follow the physiological use of the term, and to understand by it that the respiratory movements are deeper than natural, or more frequent, or both deeper and more frequent, without regard to whether the patient is conscious of the disturbance.

It is a remarkable fact that persons affected with extensive disease of the lungs, provided that such disease develops itself gradually, may continue to breathe as slowly as in health, and with no more effort, so long as they are at rest. The amount of oxygen supplied to the blood is no doubt much reduced; but it suffices for the wants of the system. It might have been supposed that the oxydising processes of the tissues would be checked; that sugar would be likely to appear in the urine, and that the excretion of urea would be diminished, and that of less perfectly oxidised bodies, as uric acid, be increased. But a series of experiments on animals recorded by Senator in 'Virchow's Archiv' for 1868 appears to show that this is not the case; and his conclusions are quite in accordance with clinical experience. The body adjusts its requirements to its necessities. One reason why persons affected with chronic bronchitis or other pulmonary disease almost always grow thin is that they instinctively learn to take very little food. But a far more important method of adjustment seems to be the avoidance of all bodily effort. So soon as such a patient begins to walk, especially on rising ground, dyspnœa sets in. Muscular exertion at once involves a demand for more oxygen than is contained in his arterial blood. So, again, the supervention of pyrexia in a case of this kind necessarily leads to a disproportionate increase in the rapidity and in the depth of the breathing. In illustration of this principle Cohnheim instances the remarkable subsidence of dyspnœa which often occurs immediately after the crisis in acute pneumonia, before the affected lung has even begun to recover itself.

Sometimes, however, dyspnœa of a very marked kind arises without reason to suppose that the oxidation of the blood is at all defective. This is the case, for example, in *diabetic coma:* and a distressing shortness of breath may be the earliest symptom of which the patient is conscious in the course of chronic *Bright's disease.* Of this a striking instance once came under the author's notice. He was seeing his out-patients, when the attendant nurse asked him to listen to her chest, because her breathing had become so difficult and laboured that she felt unfit for any exertion. After the most careful investigation, nothing amiss with either the lungs or the heart could be detected. Then, as she said she was thirsty, her urine was examined for sugar, and the result being still negative, it was tested with nitric acid, which brought down a large quantity of albumen. A few months later dropsy set in, and her case soon ended fatally.

Another form of dyspnœa, independent of any disease of the thoracic organs, is of *nervous* origin. It is often ascribed to hysteria; but, like other hysterical symptoms, it is not always accompanied by other signs of that disease.

Phrenic dyspnœa.—There is a peculiar form of dyspnœa which depends upon *paralysis of the diaphragm.* Its characters were first recognised by

Duchenne, and they deserve careful study, because its origin is very likely to be overlooked. So long as the patient is at rest his breathing is perfectly easy. But the slightest effort at once begins to distress him and to increase the frequency of his respirations; when he walks he experiences a sense of suffocation as soon as he has made a few steps; in mounting a staircase, and even in speaking, he is obliged to stop every instant to take breath. When he sighs he feels as though the abdominal organs were being drawn up into his chest. The act of defæcation is much embarrassed. His voice is weak; there is more or less difficulty in coughing and sneezing, because he cannot take the deep full inspiration which is a necessary preliminary; so that even a slight attack of bronchitis is attended with great danger. If one looks at the surface of his body while he breathes, the characteristic indication of paralysis of the diaphragm is generally at once apparent. During inspiration, when the ribs rise and the chest expands, the epigastrium and the hypochondriac regions are drawn in; during expiration they are pushed forwards. In other words, their relation to the thoracic movements is exactly the reverse of what it normally should be. Sometimes it is not so easy to see the alteration as to feel it with the two hands placed just below the cartilages of the ribs. If only one side of the diaphragm is paralysed, as is sometimes the case, the corresponding hypochondrium may be drawn in while the other one protrudes in the natural manner.

Among the examples of this affection recorded by Duchenne, there are some in which it appeared at an advanced stage of progressive muscular atrophy, others in which it was associated with paralysis of many other muscles as the result of lead-poisoning, and one in which it was hysterical. Walshe says that he has seen it in a well-marked form as a sequel of diphtheria. Erb cites Oppolzer as having observed it at the age of puberty without any cause being discoverable. Another cause, which is mentioned by Duchenne on the authority of Aran, is the extension of inflammation from the peritoneum or from the pleura. And he gives a case of empyema in which the muscular tissue of the corresponding side of the diaphragm was of an orange-yellow colour, and in which the fibres microscopically had undergone complete fatty degeneration.

It appears hazardous to diagnose paralysis of the diaphragm in every case of thoracic disease in which one or both of the hypochondriac regions are drawn in during the act of inspiration. That is no infrequent occurrence in a great variety of circumstances, and is often due to a mere inaction of the muscle, which surely ought to be distinguished from paralysis. The application of electricity seems not to be likely to help in clearing up the difficulty, for in all cases in which the diaphragm has been observed to be paralysed it appears to have retained its power of responding to faradic stimulation of the phrenic nerves. The best method of stimulating these nerves is, according to Duchenne, as follows:—by two fingers, placed just outside the edge of one sterno-mastoid muscle, the skin is first drawn slightly inwards; they are then separated, leaving between them an interval, upon which a small conical metal rheophore is pressed, so as to be just over the spot where the phrenic nerve lies upon the scalenus anticus. The rheophore is now given to an assistant to hold, and the same procedure is repeated on the opposite side of the neck. When both rheophores are fixed the operator takes one in each hand. He passes through them an interrupted current, which should instantly give rise to a contraction of the diaphragm, shown by the abdominal walls being pushed forwards, while

the lower ribs are separated from one another. Sometimes, however, the platysma interferes with this result, contracting with such force as to jerk the rheophores out of position. And sometimes it is necessary to shift them a little from spot to spot before one can succeed in acting on the phrenic nerves. Erb recommends a different method; he places one pole upon the neck, and the other over the attachment of the diaphragm to the costal cartilages.

Whatever position may be adopted for the rheophores, the stimulus should be so used that the resulting contraction of the diaphragm may fall in with the natural respiratory movements. The current should be stopped as soon as the muscle has acted, and a few seconds after it should be re-applied.

This procedure seems to possess considerable therapeutical value. By means of it Duchenne succeeded in completely restoring the functions of the diaphragm in a man named Bonnard, who had advanced progressive atrophy of other muscles, but in whom the paralysis of the muscle in question was as yet recent and incomplete, as was shown by the hypochondriac regions receding only when he breathed deeply. After a few weeks of treatment he became able to ascend stairs, and to walk long distances without discomfort.

Orthopnœa.—There is a kind of dyspnœa in which there is no distress and little acceleration of the breath so long as the patient is either standing or sitting up in bed; but so soon as he lies down difficulty in breathing is apparent.* That the diaphragm descends more freely when the weight of the liver and other viscera draws away from it, and that both sides of the chest act better when the weight of the body does not hamper them, either on one side or behind, is readily understood. It is as natural as the increase of dyspnœa which follows movement, and particularly ascent of rising ground. But it is not easy to explain why the advantage of an upright position is so much more marked in cases of cardiac dyspnœa than in those of bronchitis, or phthisis, or anæmia. As a clinical fact, however, while some degree of orthopnœa accompanies short breath from whatever cause, the marked form in which a patient sitting up in bed is able to converse easily, but is quite unable to lie down, and can only sleep propped up with pillows or in an arm-chair—this typical orthopnœa—is rarely seen except in cases of organic disease of the heart. It may sometimes be observed in emphysematous patients, and as a mere complication of dyspnœa it is seen in spasmodic asthma, in ascites and renal dropsy, in grave anæmia, in advanced phthisis, and in cases of pleural effusion and of pneumothorax.

Cheyne-Stokes respiration.—This is the title given by German writers to a curious variety of dyspnœa first noticed by Dr John Cheyne, of Dublin; and afterwards described by Professor Stokes, who regarded it as a symptom of fatty degeneration of the heart. An attack usually, but not always, begins with dyspnœa; this is followed by a gradual slackening of the respiratory movements until they fail altogether, and a complete pause ensues for half a minute or even longer. This condition of apnœa is succeeded by slow and shallow breathing, which gradually quickens and deepens until it resumes its former character. The whole process is of variable length, and is usually repeated several times within a few hours;

* "Ægri recto corpore residere cupiunt, qui habitus est ad eam rem aptissimus." 'Aretæi de causis et signis acutorum morborum,' lib. ii, cap. 1.

often it recurs during several days or even for weeks and months. The total interruption of the breath gradually led up to, and in like manner gradually recovered from, is the essence of the phenomenon. Its physiology has been much discussed by Traube, Schiff, Filehne, Luciani, and Wertheimer.* It may be observed in rabbits and other animals after considerable hæmorrhage; and is seen clinically not only under the condition described by Stokes, but in cases of apoplexy, meningitis, and cerebral tumours, in the uræmic state, and in the *coma vigil* of extreme anæmia and of exhaustion from fevers. It may occur during chloroform narcosis, or as a result of frequent administration of morphia. It often comes on while the patient is asleep or comatose, but sometimes when he is in possession of his faculties, walking about, and fully sensible of the peculiarity of his breathing. The pupils become contracted during the height of the fit, as in other conditions of apnœa, but this is not constant; the pulse is quickened in the ingravescent stage as the number of respirations is increased, and subsides with them, until the pause follows, when it becomes slow and incompressible.

There is little doubt that the immediate cause of this curious phenomenon is some change (probably due to anæmia) in the respiration-centre of the bulb. It is seldom or never observed in uncomplicated diseases of the heart or lungs, and rarely accompanies marked orthopnœa or severe dyspnœa. It is a symptom of nervous origin, and probably connected with deficiency in oxyhæmoglobin of the blood supplying the medulla oblongata. Its relation to fatty degeneration of the heart is perhaps rather that both are results of anæmia than that a feeble circulation causes it, for it is very rare in cases of cardiac failure from valvular disease.

As a prognostic symptom Cheyne-Stokes breathing is very grave. Undoubted cases of recovery after it has appeared have been recorded, but as a rule it only comes on in the last stages of cerebral disease, of anæmia, uræmia, or cholæmia, and points to exhaustion of the nervous centres and to approaching death.

COUGH, as is well known, is produced in the following manner:—A deep inspiration is first taken, the glottis is then closed, and, a sudden expiratory effort being made, the glottis is allowed to open, causing a loud sound, and allowing a blast of air to pass out, which may carry with it any secretion or other substance present in the air-passages. In describing laryngeal diseases we have already seen how they may modify the characters of cough, giving it a hoarse, or rough, or metallic quality, or rendering it almost noiseless. But an ordinary cough may almost be taken as an indication that the larynx is healthy.

The nervous mechanism by which cough is effected is reflex in its action. As a rule, the irritation which gives rise to it starts from the respiratory mucous membrane, as is evidenced by the consequent expulsion of mucus or pus in greater or less quantity. But sometimes the most violent and repeated efforts of coughing bring away nothing. The cough is then said to be *dry;* and in the last century the distinction between a "dry" and a "humid" cough seems to have been regarded as one of the most fundamental points in regard to chest complaints. It is, however, quite possible for the air-passages to contain mucus which is too viscid and too firmly adherent to be expectorated; and probably what is still more frequent is

* See also Dr Samuel West's remarkable case ('Lancet,' 1890, p. 545), and one related by Dr J. D. Mann ('Brain,' 1890, p. 178), which he observed for fourteen months.

that some part of the respiratory mucous membrane is affected with slight catarrh, and that this condition either itself constitutes an "irritation" or else renders the surface sensitive to the passage of air over it, or to the disturbance produced by the laryngeal movements in breathing or speaking. But, on the other hand, there is no doubt that the starting-point of cough is sometimes altogether outside the air-passages, and, as may well be supposed, the recognition of this fact is of great importance in medical practice. The question has been worked out in experiments upon animals by several physiologists, one of whom was Kohts, of Strasburg, whose observations appeared in 'Virchow's Archiv' for 1874. In regard to cough, as to all other reflex phenomena, although positive experimental results are of great clinical value, negative results prove very little. For under morbid conditions afferent nerves may transmit impressions with more than usual energy, or reflex centres may be unduly excitable, so as to be stimulated by impressions which normally should not disturb them.

The following appear to be the chief ætiological varieties of cough which have to be recognised, apart from affections of the respiratory organs.

1. *Throat cough.*—Kohts found, both in animals and in man, that irritation of the pharynx had the effect of producing cough in many individuals, but not in all. There is therefore no theoretical difficulty in admitting that catarrh of the fauces may be attended with cough, without there being a corresponding affection of the larynx; but the parts being continuous it must always be difficult, if not impossible, to say that this is actually the case, especially as Kohts showed that the glosso- and arytæno-epiglottic folds and the lateral edges of the epiglottis were among the most sensitive structures of all, so far as the production of cough is concerned. It is a somewhat different question whether a relaxed and elongated uvula frequently gives rise to cough by coming into contact with the parts behind the base of the tongue. Mackenzie speaks of this as giving rise to a "distressing tickling cough continuing all day," and some surgeons have adopted the practice of snipping off the uvula whenever a patient has complained of such a cough, for which no other cause could be discovered. But while this treatment sometimes succeeds (as, for instance, in cases recorded by Dr Garrett, of Hastings, in the 'Lancet' for 1872), its failures are at least as frequent.

2. *Ear cough.*—That cough can be excited by irritation of the external auditory meatus had been known long ago, but it was generally forgotten until Dr Cornelius Fox drew attention to it in the 'Lancet' for 1867. He examined a number of persons, and found that this peculiarity existed in about one among every five or six. He mentions the case of a gentleman who experienced a feeling of irritation of the larynx and had a violent suffocating cough whenever he introduced a toothpick into the left ear; in him, too, a somewhat similar action was capable of being exerted in the reverse direction, for long-continued singing would cause him pain in the ear. Dr Fox shows that the ear may sometimes be the starting-point of a cough under such circumstances that the relation may be overlooked. Thus a healthy-looking woman, aged fifty, had for eighteen months had a most distressing cough. As she was deaf in the right ear the meatus was examined, and was found to contain a hard plug of cerumen, and to have a small ulcer in its floor. Almost immediate relief to the cough followed extraction of the wax and the application of nitrate of silver to heal the ulcer. In a patient of Mr Toynbee's a cough was cured by the removal of a piece of necrosed bone from the external ear. It is obvious that a foreign body,

such as a bead, might have a similar effect. Dr Fox is no doubt right in maintaining that the afferent nerve in all such cases is the auriculo-temporal branch of the fifth, and not (as had been suggested) the minute auricular twig of the vagus.

3. *Tooth cough.*—Dr Fox incidentally mentions that it is well known to dentists that the stump of a tooth may be the starting-point of a cough, and he also refers to cough in infants during the first dentition as ceasing when the gum lancet is used. In investigating an obscure case, therefore, one must not fail to examine the teeth.

4. *Stomach cough.*—In the last century it was a favourite dogma that dry cough, and even humid cough, are very often produced by disorder of the digestive organs. The most recent exposition of such a view is to be found in 'Copland's Dictionary.' But, as so often happens in like cases, what has long since ceased to be taught by the faculty has become an article of faith among the public. Thus mothers still commonly refer to the stomach coughs in their children which are really due to catarrh of the upper air-passages. Or, committing a fatal error, they set down to the same cause the dry cough of early phthisis, attended (as it often is) with nausea and loss of appetite and pain in the side. It has been stated that the sign of a cough due to gastric irritation is either that it comes on when the stomach is loaded with a full meal, and disappears after the completion of digestion, or else that it occurs chiefly when the patient is in bed at night. The second of these criteria corresponds well with the fact that persons in whom intermission of the pulse and palpitation of the heart are caused by irritation of the stomach experience these symptoms when they lie down more than when they are sitting or standing. Kohts, however, in his experiments, failed altogether to excite cough by irritating the stomach. He cites from 'Brücke's Physiology' a case in which a boy coughed day and night with the utmost violence and obstinacy until he vomited, whereupon the cough at once ceased; but he adds that Brücke, who himself made the observation, believed the starting-point of the affection to have been, after all, something in the air-passages, which became dislodged when the stomach expelled its contents. Another instance, quoted from Professor Leyden, is that of a patient who had repeated attacks of biliary colic, and who every time became affected with dry cough and with pain in the right hypochondrium twenty-four hours before the jaundice set in. Walshe says that he has known the trifling irritation due to the presence of an *Ascaris lumbricoides* keep up reflex cough for several weeks.

5. *Centric cough.*—Kohts found that he could sometimes excite cough in animals by mechanical or electrical stimulation of the floor of the fourth ventricle, and he thinks that the centre for this reflex act is situated rather above that for respiration. In hysteria, as is well known, a hard, dry, barking cough is common, and this may be supposed to be centric in its origin. A remarkable instance of this was recorded by Dr John Harley in the 'Med. Times and Gazette' for 1863. The patient, a girl aged fourteen, uttered a short bark seventy times a minute without intermission, so that, according to calculation, she must have coughed 40,000 or 50,000 times in the course of the day. She had had the cough a fortnight when she came under observation. She was treated with valerianate of zinc and with a cold douche and frictions to the spine, and in three days the cough ceased. A very similar case, in a child aged eight, was described by Dr Whytt more than a century ago under the name of nervous cough. A

remarkable feature in each of these cases was that the cough ceased instantly when the patient lay down. Dr Whytt made an elaborate series of investigations into the effects of posture upon his patient, finding (for example) that the cough did not return when she sat up in bed so long as the feet were extended straight out, but that as soon as they were inclined at an angle she began to cough. He also observed that putting the feet in hot water at once arrested the cough.

PAIN is a symptom of various thoracic diseases, but it may also occur in the same places when it is the sole indication that anything is the matter with the patient, and when therefore it can only be regarded as a substantive affection.

Sometimes, perhaps, the seat to which pain is referred is the interior of the lung itself. Walshe speaks of "pains deeply felt within the chest, and shooting in the direction of the pulmonary branches of the vagi and sympathetic," as existing "independently of any other deviation from health not only local but general." He also refers to "various anomalous and more or less painful sensations, felt deeply within the chest by phthisical patients."

But in the large majority of cases thoracic pain is referred to the chest walls, and especially to one or both of the infra-mammary or infra-axillary regions. Various names are given to pain in these situations, according to the views held with regard to its nature.* The term "pain in the side," *pleurodynia*, is the best of these.

It is sometimes difficult to determine whether a pain in the side is due to pleurisy, anæmia, mitral disease, or gastric disturbance. The stomach seems to be frequently its starting-point when it is on the left side, which is the case in most instances of pleurodynia. The same is true of the pleurodynia of cardiac disease and of chlorosis. Another frequent cause is ovarian irritation, especially in women who are hysterical. The spine, too, must be thought of, even when the pain is unilateral; and we must remember that a pain in the side, if recent, may be the precursor of an attack of shingles.

Again, it is necessary to bear in mind the possible presence of disease or injury of a rib. In July, 1877, the author was consulted by a lady, the wife of an old schoolfellow, who told him that, having had a cough all the previous winter, she had one night felt something crack in her left side while she was coughing. Ever since then she had suffered from a continuous gnawing pain there. On examining the side there was considerable enlargement of one of the lower ribs, which seemed to be clearly the callus of a fracture. Under suitable treatment she got well, but for as long as six months afterwards she still experienced some pain in coughing, which, however, was no longer limited to one spot, and extended as high as the shoulder. She also said that she sometimes felt pain in the side towards

* Walshe describes in succession three separate affections, which he terms "pleurodynia," "thoracic myalgia," and "intercostal neuralgia." The distinctions which he would draw between them seem to be chiefly in reference to the intensity and duration of the pain, to its being accompanied by superficial tenderness, and to the presence or the absence of the "points douloureux" of Valleix (cf. p. 385). He limits the term *pleurodynia* to attacks of pain of extreme severity, generally setting in suddenly and lasting only a short time. Of it he says first that it is "an actual rheumatism of the walls of the chest, affecting their muscular and fibrous textures;" and then, a little further on, that "nerve-fibres are implicated, and that rheumatic neuralgia of the intercostal nerves forms an element of it." Is it not clear that the distinction is only arbitrary, and the hypothesis of rheumatism equally so?

night when she was fatigued, and that changes of weather seemed to increase it. Probably such fracture of a rib in coughing is extremely rare. In a lecture reported in the 'Lancet' for 1882, Mr Marshall relates the case of a woman, aged thirty-five, who in the severe weather of the spring of 1881 caught cold, shivered, and was attacked first with pain in the left side and then, a month later, no less severely in the right. In the previous year she had had acute rheumatism, and her case was regarded as neuralgic. At length she came to Mr Marshall, who found two firm oblong swellings, one along the lower border of the right fifth rib, and the other at a corresponding spot upon the eighth rib. When they were pressed upon she experienced acute pains shooting through to her back. They gradually softened into abscesses and were opened, when parts of each rib were found to be eroded and softened. Ultimately some pieces of dead bone came away, and she did perfectly well. In another instance, also recorded by Mr Marshall, an abscess, evidently connected with disease of a rib, arose in a patient who had phthisis. Syphilitic periostitis is another affection that must be borne in mind, although it is much less common in the case of the ribs than of the sternum. Mr Marshall speaks of it as almost confined to women.

BRONCHITIS

" Forte si tussire occæperit, ne sic tussiat,
Ut cuiquam linguam in tussiendo proferat."
PLAUTUS, 'Asinaria,' iv, 1.

General symptoms: cough, dyspnœa, pain—Physical signs: rhonchus, sibilus, râles—Morbid anatomy.
Acute Bronchitis—Capillary form—Its symptoms and prognosis—Pulmonary collapse—Its production and relation to pneumonia.
Chronic Bronchitis—Varieties—Sequelæ—Emphysema—Its anatomy and origin—Atrophic emphysema—Symptoms and signs of emphysema—Bronchiectasis, uniform and saccular—Fœtid bronchitis—Ætiology, prognosis, and treatment of bronchitis generally.
Plastic bronchitis—Its rarity—Anatomy, course, and symptoms—Treatment.

THE disease which is termed bronchitis is very common in our climate, and the word is now familiar, but it does not appear in medical literature before the publication of works in 1812 by Peter Frank in Germany, and in 1814 by Badham in England. Up to that time the disease was known as "catarrh," or "defluxion on the breast," while the more severe forms were designated by the cumbrous name of "peripneumonia notha," invented by Sydenham. Bronchitis is generally understood to include inflammations of all parts of the air-passages below the larynx. When the windpipe is very obviously affected, the term tracheitis may be used; but, as might be expected, the boundary lines recognised by the anatomist find no application in clinical practice. In a large number of cases the morbid action reaches to a greater or less extent above the bifurcation of the trachea, yet we need not speak of the disease as anything more than bronchitis.

Bronchitis as the term is usually applied refers to *catarrhal* inflammation, and does not imply either plastic exudation or ulceration.

There are few affections of which there are so many varieties as of bronchitis; and these differ, both in symptoms and in course. The most convenient plan will be to enumerate such of the symptoms and physical signs as belong to all alike, and afterwards to give separate accounts of the more important varieties.

Common symptoms.—Foremost among the general symptoms is *cough*. This is never absent, and it is often exceedingly severe, and of a loud, barking, or ringing character. It may consist of isolated explosions, succeeding one another more or less regularly, and sometimes with extreme frequency. Or it may occur in paroxysms, which sometimes end in retching or actual vomiting. It may be worse when the patient lies down, or it may come on especially when he first gets up in the morning, being excited by an accumulation of mucus or pus in the air-passages during the

night. Sometimes the irritation which sets it up is definitely referred to some one spot along the course of the trachea, which is felt to be raw or tender; sometimes there is a vague tickling sensation, which cannot be localised. The characters of the sputum differ so widely in different forms of bronchitis that it is useless to speak of them generally.

Another symptom in all severe cases of bronchitis is *dyspnœa*. It depends, in the main, upon the mechanical obstruction to the entrance of air into the pulmonary vesicles, which has its seat in the small tubes.

A curious circumstance, to which Riegel seems to have first drawn attention, is that in all affections of the bronchioles the dyspnœa is expiratory rather than inspiratory. Sometimes the act of inspiration is quite short and easy, while that of expiration is difficult and much prolonged; sometimes they are both embarrassed; but inspiration appears never to be alone impeded in bronchitis, as it so often is in affections of the larynx or trachea. This special tendency to expiratory dyspnœa, when the smaller air-tubes are inflamed, perhaps depends on paresis of the muscular fibres of the bronchia.

Another peculiarity of the breathing, which may often be noticed in children, is that each expiration is instantly followed by an inspiration, the pause in the act of breathing taking place at the end of each inspiration and not at the end of expiration, as it does normally.

In severe cases there may be *orthopnœa*, so that the patient must be propped up with pillows all night, but this is usually an indication that there is emphysema beside bronchitis.

When dyspnœa is considerable there is more or less lividity or *cyanosis*. This shows itself in the face and hands. In extreme cases the face becomes turgid, flushed, and bloated. The veins of the neck are dilated and throb with each pulsation of the right ventricle, and the superficial veins generally are fuller than natural. This condition of venous congestion contrasts with the pallor which usually accompanies the dyspnœa of phthisis, of diphtheria, of cardiac, and of renal disease.

Pain is by no means constantly present in bronchitis. Many patients, however, complain of a sore feeling behind the sternum, or in the upper part of the chest on either side. Or, again, the harassing cough may give rise to more or less severe myalgia in some part of the thoracic walls. Not infrequently this muscular pain is felt at the epigastrium. But another cause of epigastric pain is fulness of the liver, from obstruction to the venous circulation.

Physical signs.—The signs of bronchitis are less numerous than those of almost any other disease of the respiratory organs. They are mainly auscultatory, for the percussion-sound is quite unaltered unless the case is complicated with pleural effusion or with some affection of the pulmonary tissue such as emphysema, or collapse, or broncho-pneumonia.

The vesicular murmur is more or less altered in character, or is replaced by adventitious sounds. In some cases, and those not the least grave, the change in the vesicular murmur is that it is *faint* and *indistinct*; it may be absent occasionally over a part of the lung, from the corresponding bronchial tube being plugged by mucus. In this case one can usually bring it back by making the patient cough vigorously two or three times.

As a rule in bronchitis the inspiratory murmur becomes *rough* and *hard* in quality, and the expiration may be accompanied by a very similar sound. It is then sometimes difficult to distinguish this from bronchial breathing.

That, however, has a more blowing character; and it is strictly limited to certain parts of the chest, whereas in bronchitis the sound is heard over both lungs alike and very widely; moreover bronchial breathing, except over the sternum and between the scapulæ, is accompanied by more or less dulness on percussion. It cannot be too strongly impressed on those who are learning the use of the stethoscope that neither bronchial breathing nor any of its modifications occurs in bronchitis except when some complication is present. Nor is the vocal resonance in any way altered.

The adventitious sounds which occur in bronchitis are those that have been already discussed, under the names of "rhonchus," "sibilus," and "râles." *Rhonchus* (or "sonorous rhonchus") is a loud snoring or cooing noise, often audible by the patient himself and by those about him, and due to vibrations that can be felt by the hand placed upon the surface of his chest. It is formed in the larger tubes, and in bronchitis its cause is the presence of a mass of viscid mucus partly obstructing the entrance of air, and producing a *veine fluide*. The proof of this is that it can very generally be made to disappear, at least for a time, by the patient's coughing once or twice, and, indeed, it comes and goes of its own accord, being heard first in one part of the chest and then in another, as mucus happens to accumulate in different branches of the bronchial tree.

Sibilus (or "sibilant rhonchus," as some prefer to term it) is a high-pitched whistling sound. It is formed in the bronchioles, and is of much graver import than rhonchus, since inflammation of the smaller air-passages is far more dangerous than that of the larger. It seems to be due to the narrowing of the calibre of the affected tubes which results from swelling of their lining membrane. Consequently, it cannot be got rid of by coughing, and it usually remains in the same spot for hours or days together.

Many terms have been employed to denote the particular quality of these continuous "dry sounds." Sometimes they resemble "snoring," as the term *rhonchus* would imply, sometimes the hoarse "cooing" of a wood-pigeon; often they are high-pitched and musical like a box of pipes, sometimes deep in tone, like the notes of certain stops of the organ, and sometimes "wheezing," "squeaking," or "whistling."

The *râles* which accompany bronchitis may be of every variety of size. They are not of "consonating" quality, inasmuch as the lung-tissue round the tubes in which they are formed still remains spongy. If râles are not universally distributed through the lungs, they are, as a rule, most marked over the lower lobes, and behind rather than in front. Signs that might suggest the presence of bronchitis in the upper lobes only—especially if limited to the upper lobe on one side—should always arouse a strong suspicion of phthisis. A point which is worthy of mention is that it is sometimes impossible to detect any râles in cases in which the profuse expectoration would certainly have led one to expect them—only dry sounds are audible.

Diagnosis.—The symptoms and physical signs above enumerated are not sufficient in themselves to justify a diagnosis of bronchitis. It is necessary to add certain negative points by which the presence of other affections of the air-passages or of the lungs is excluded. Particularly in cases of which the clinical history is such as to render it possible that the pulmonary parenchyma may contain scattered tubercles, whether of acute or of chronic

development, great caution must be exercised in forming an opinion. Sometimes, but very rarely, the occurrence of secondary nodules of a malignant new growth in the lungs offers another chance of error. The difficulty is not so much in saying that bronchitis is present as in determining whether it is the principal affection, or only a complication. For it is exceedingly apt to arise in the course of a great variety of diseases, as measles, enteric fever, rickets, phthisis, organic lesions of the heart, and Bright's disease.

Morbid anatomy.—In mucous membranes, as in the skin, the morbid appearances produced by inflammation are far less conspicuous after death than during life; and a reason why this is pre-eminently the case with the bronchial mucous membrane is afforded by the abundance of elastic fibres in its structure. It is sometimes far from easy to determine the presence of bronchitis at an autopsy, even when it was the sole disease. A good method of detecting puriform secretion in the smaller tubes is to slice off the edge of the lung, and then to press the tissue towards the cut surface, when a yellow bead appears at each little orifice. But in many cases there is extreme redness and swelling of the mucous membrane, which may have a velvety appearance, while every part of the air-passages, up to the trachea, may be full of a yellow or brownish opaque fluid.

The histological changes in bronchitis have been in recent times particularly studied by Socoleff ('Virchow's Archiv,' vol. lxix), and by Dr Hamilton ('Practitioner,' 1879). The former examined artificial bronchitis in dogs and in rabbits produced by insufflation of bichromate of potass or of a weak solution of chromic acid; the latter studied cases that presented themselves in the *post-mortem* room of the Edinburgh Royal Infirmary. Both observers are agreed that a very early change is the detachment of the ciliated epithelium, which seems to be thrown off in flakes, and which remains absent during the whole course of the disease, to be regenerated when recovery takes place. In a young man who died of opium-poisoning, in from ten to sixteen hours, the ciliated cells were already to a great extent shed, although Dr Hamilton speaks of the morbid process in that case as having been rather acute congestion than actual inflammation. He says that the cells themselves undergo fatty degeneration, which probably destroys many of them. Others, no doubt, are expectorated; others are inhaled into the smaller air-tubes, where they may be seen lying in large detached masses among the other catarrhal products. There is an obvious analogy between this exfoliation of the columnar layer of the bronchial epithelium and desquamation of the cuticle when inflammation affects the skin. During the further progress of the attack the basement membrane is covered only by a layer of flat cells, from which there project here and there pyriform or oval corpuscles, of transitional character, which are covered by a more or less abundant mass of leucocytes, embedded in a mucoid fluid. A point on which Dr Hamilton lays great stress is that the basement membrane itself becomes thickened and swollen, apparently as the result of œdema. Both he and Socoleff are convinced that the leucocytes which appear in such large numbers upon the free surface of the mucous membrane are not derived by emigration from the blood-vessels, but are formed by germination from the flat cells that lie immediately in contact with the basement membrane. Socoleff's chief reason for maintaining this opinion was that in animals killed twenty-four hours after the commencement of the morbid process he found leucocytes on the free surface of the mucous membrane, although its

substance was at that time entirely free from them. Dr Hamilton insists especially on the difficulty which leucocytes derived from the blood would have in traversing the thickened basement membrane, and on the fact that in his preparations he could discover no indication that this was taking place. But it is perhaps worthy of notice that Socoleff himself figures ciliated epithelial cells having in their interior red blood-discs, which must have made their way through. And one naturally hesitates to accept observations upon deep-seated tissues as overthrowing the results of investigations made upon the cornea and other superficial structures, for the special purpose of determining the nature of the inflammatory process (cf. p. 45).

In all but very early and very slight cases of bronchitis the mucous or submucous tissues are, in their whole substance, more or less thickly filled with leucocytes, which are collected in lines along the lymph spaces between the fibrous bundles and around the vessels. Dr Hamilton is satisfied that these also are in great part derived by germination from the flat endothelial cells of the lymph spaces or from connective-tissue corpuscles. Another very important change occurs in the mucous glands. They become swollen so as to be sometimes as large as hemp-seeds, according to Riegel. Their epithelium undergoes very active proliferation, and the newly-formed cells become distended with mucin, and appear to be the source of the mucus that often forms so large a part of the expectoration. This mucus, however, becomes mixed with leucocytes and epithelial cells of "transitional" form, as well as with saliva and air-bubbles.

When bronchitis has existed for a great length of time before death the changes found *post mortem* are somewhat different. The mucous membrane is often pale and grey, with but few vessels visible. In many cases it presents a number of delicate longitudinal ridges, which Rindfleisch has shown to consist of an overgrowth of connective tissue, containing very numerous cells, and bundles of elastic fibres running in various directions. According to Dr Hamilton the muscular coat is sometimes found to be hypertrophied, sometimes atrophied. The cartilages shrink and disappear, undergoing the same change which occurs in articular cartilage, namely, the absorption of the matrix from the periphery inwards, with the formation of "medullary spaces" filled with leucocytes. In many cases the mucous glands also are destroyed. At an earlier period their orifices are widely dilated, giving the mucous membrane a minutely pitted appearance when looked at with a good light; and sometimes they become inflamed, forming minute funnel-shaped ulcers.

Of the various forms of bronchitis some run an *acute*, others a *chronic* course.

ACUTE BRONCHITIS.—This often affects mainly the larger air-passages, so that it may fairly be called a "tracheo-bronchitis;" and in such cases the inflammation is sometimes derived by extension from the nose and throat. The cough is often very distressing, and especially violent when the patient attempts to lie down. He may complain greatly of a sore sensation along the sternum; and pressure upon the trachea may be painful and may at once excite cough. This form of the disease, however, is not dangerous.

Capillary form.—Very different is the course of acute bronchitis when it attacks the bronchioles throughout the lungs, for this may be one of the

most rapidly fatal of all diseases. It is distinguished as "Capillary Bronchitis," which answers to what was formerly called "Suffocative Catarrh."

It usually sets in with a sensation of chilliness, or less frequently with a rigor which may be repeated. The degree of pyrexia varies; the temperature may range up to 104°, especially in children; more often it is at a lower level; and it does not run any typical course. The head and the upper part of the body become covered with sweat. The hands and the surface generally feel hot and the face is more or less flushed. The pulse is frequent, sometimes so rapid that it cannot be counted. It is often tense and full, perhaps as the result of contraction of the systemic arterioles.

But the most prominent symptom of this form of bronchitis is the dyspnœa. The patient sits up, with chest heaving and with nostrils quivering, unable to utter more than two or three words at a time, using his shoulders and arms in violent efforts to breathe. On carefully inspecting the thoracic movements, one finds that there is a great obstacle to the entrance of air into the lungs. The epigastric and the hypochondriac regions of the abdomen recede at every inspiration; in children all the lower ribs and lower part of the sternum may be forcibly sucked in. The supra-clavicular and the suprasternal spaces also recede, but, on the other hand, as Seitz has pointed out, the upper ribs often remain almost motionless in a position which is that of a forced inspiration, giving to the corresponding part of the chest a vaulted shape.

The cough of capillary bronchitis is often exceedingly harrassing. At first it is usually dry, there being nothing in the air-passages to be expectorated. Afterwards it is accompanied by more or less abundant sputum. The secretion of the inflamed bronchial mucous membrane, goes through stages very similar to those that may be observed during the progress of a cold in the head. It begins by being swollen and dry, then it pours out a transparent mucous fluid; after a time this becomes mucopurulent, and finally almost pure pus. These changes are what the older medical writers described as "concoction." The dry stage sometimes lasts several days, or even throughout the whole duration of the disease. Thus Dr Latham in his 'Lectures on Subjects connected with Clinical Medicine,' narrates the case of a boy, seven or eight years old, who for six days remained in a condition of extreme suffering, with shrill sibilus audible all over his chest, and then gradually recovered without expectorating anything. A point, however, which must not be forgotten is that infants and children as old as this patient commonly swallow whatever they cough out of the air-passages into the mouth. In older patients, when sputum first appears, it is as a rule scanty and dislodged with great difficulty, the patient perhaps coughing a number of times in rapid succession, until he is purple in the face, before he can get relief by bringing up a translucent pellet of mucus. But in other cases the spitting-jar becomes filled in a few hours with a considerable quantity of a greyish-white glairy liquid, which has numerous air-bubbles entangled in it. Under the microscope this kind of sputum is found to contain remarkably few formed elements. As already remarked, in bronchitis the tubes cease for the time to be lined with columnar epithelium. It is therefore probable that when a few cells of that type are seen in the matters expectorated (except at the very commencement of the disease) they have been derived from healthy and not from inflamed parts of the air-passages, just as flat epithelial cells are often seen which come

from the throat or the mouth. Cells of transitional form, however, and mucous corpuscles are present in small numbers, and as the case advances pus-cells abound more and more until the sputum becomes opaque and of a greenish-yellow colour. It now comes freely, and with little effort, so that the cough is said to be "loose."

Event.—In many cases, after acute bronchitis has lasted for some time, the quantity of expectoration begins gradually to diminish from day to day; the other symptoms become less and less severe, and presently the patient recovers from his attack. But in other cases the inflammatory exudation accumulates in such large amount as to threaten death by suffocation. Râles then become audible all over the chest, and are so loud that no trace of vesicular murmur can be anywhere detected. Indeed, they are often heard at a distance from the patient. Still more important as a warning of danger is the supervention of cyanosis; the flushed cheeks, the lips, and the hands assume first a faint lilac, and finally a leaden colour; the blood is no longer duly aerated, and a condition of asphyxia has begun.

Another very grave symptom is the failure of effort on the part of the respiratory muscles; the breathing gradually becomes more and more shallow, until at last it may be represented only by a slight flickering movement of a few of the ribs, or by a faint jerking contraction of the diaphragm. With this, too, the patient ceases to be conscious of the necessity for active breathing. Instead of remaining upright he sinks down in bed, with his head in any position in which it may happen to be placed. His mind may wander for a time and then he becomes unconscious. Sometimes death is preceded by convulsions.

As a rule, if acute bronchitis is to end fatally, it does so in the course of the first fortnight, and in some rare cases the patient succumbs within twenty-four or forty-eight hours. But it not infrequently happens that when the disease has apparently been subsiding favourably, a relapse occurs which puts an end to all hope of recovery. It need not be said that the patient's muscular strength is one of the most important points that one has to take into account in attempting to form an estimate of the probable issue in a state of acute bronchitis. In very old persons the prognosis is always doubtful; it is so likewise in those who are very fat, or who are already weakened by previous illness, or who have progressive muscular atrophy affecting the shoulder or trunk muscles, or any considerable deformity of the spine or of the chest.

In infants, the chance of recovery is better in proportion to the age; while the gravity of the disease is greatly increased by the presence of rickets. We must be cautious, however, in giving an unfavourable prognosis in the case of children. It is surprising how rapid may be both the pulse and the breathing, for two or three days together, in those who ultimately recover.

Complications.—In acute bronchitis the digestive organs are often disturbed in a manner that is not readily accounted for, since the degree of pyrexia is but moderate. The tongue is often coated with a thick whitish-yellow fur. There may be nausea and vomiting, and the bowels may be obstinately constipated. In children it is sometimes difficult to say whether the abdominal or the bronchial symptoms are primary.

But the most important complications of acute bronchitis, when it attacks the smaller tubes, concern the substance of the lungs. One of them

is known as *collapse* of the pulmonary tissue; the other is an inflammatory affection known as *broncho-pneumonia*. This is distinct in its origin, pathology, and histology from true or acute pneumonia; but since, unfortunately, that term is applied to at least three separate diseases, it will be more convenient to consider it in the next chapter.

PULMONARY COLLAPSE is identical with a state of lung which is seen in infants as the result of imperfect respiration, and which is nothing else than a persistence of the fœtal condition of the tissue. That, however, is properly termed Apneumatosis or Atelectasis (ἀτελής = imperfect, ἔκτασις = expansion). It affects the whole of both lungs if the child has never breathed at all, or parts of the lungs (especially the anterior edges) if it has breathed incompletely, from having been prematurely born, or being weakly, or having its air-passages obstructed by mucous.

A German writer, Jörg, is generally credited with having been the first to point out, in the year 1832, the real nature of the atelectasis, which before was supposed to be congenital pneumonia; and it is commonly said that the patches of collapsed lung also were up to that time confounded with red hepatisation. But Bright in 1828, writing of the morbid appearance found in the lungs of two children who had died of whooping-cough, showed that he clearly recognised the difference; and Dr Alderson also is said to have drawn attention to it. The distinction between these two affections is unmistakable. A collapsed part of the lung is, indeed, reddened, and the colour of its cut surface is reddish brown, or, when covered with pleura, reddish purple or violet. A section of it, however, looks perfectly dry, smooth, and homogeneous; it has not the dull, lustreless, and granular appearance of hepatisation. Moreover, its surface lies below the level of the adjacent air-containing parts of the lung, whether seen in sections or on the pleural surface; if it reaches the free edge of the organ it forms a notch there. Lastly, inflation from the bronchus will usually restore to it its normal appearance.

Sometimes, however, collapsed pulmonary tissue is at the same time œdematous, and then its characters are less marked, its cut surface being moist and emitting serum when gently squeezed.

Collapse after bronchitis must be distinguished from the airless condition called *carnification*, which is caused by compression from pleural effusion; here the tissue is bloodless as well as airless, and the colour is slaty or mouse-coloured instead of being reddish brown.

The way in which collapse arises was well illustrated in a case which occurred at Guy's Hospital in 1874. A child, aged two years and two months, died four days after the performance of tracheotomy for a chronic laryngeal affection. Upon the under surface of the left lung there was a narrow red line of collapsed lung-tissue. This had running through it a tube which (like all the other tubes in the same part of the organ) happened to have become dilated as the result of the chronic obstruction to the child's breathing. That tube was plugged at its upper part by a piece of sponge, about a quarter of an inch long, which had evidently fallen into the trachea at the time of the operation. The limitation of the collapse to the part of the lung served by the obstructed tube was perfect. As a rule, collapse is secondary to closure of the corresponding tube by viscid mucus.

How this brings about the affection has only lately been well under-

stood. Gairdner, in 1850, suggested that the plug acted like a ball-valve, allowing air to escape during expiration, but preventing its entrance during inspiration. But the explanation always seemed unsatisfactory, and would not account for the complete disappearance of the air, inasmuch as the elastic force of the pulmonary tissue and that of the confined air must soon become inadequate to raise the valve. Lichtheim, of Berne, in an important research recorded in the 'Arch. f. exp. Path.' for 1879, showed that in rabbits collapse follows within twenty-four hours after a bronchus has been plugged by a piece of laminaria, which becomes swollen, so as to prevent passage of air in either direction. It is therefore evident that the affection must depend mainly, if not entirely, upon absorption of the air by the blood which circulates in the walls of the alveoli; and Lichtheim gives reasons for believing that the several gaseous constituents of atmosphere are taken up with different degrees of rapidity, the carbonic acid and the oxygen first, and afterwards the nitrogen. A further result of his investigations is the proof that the elasticity of the pulmonary tissue is not exhausted until it has become completely devoid of air. For without the aid of this elasticity, absorption by the blood must cease before collapse would become complete.

The state in which a lung is found when there has been slight narrowing of the space in which it lies (whether from pleural effusion or enlargement of the heart or pushing up of the diaphragm) is inexplicable, unless it be admitted that whenever even a small part of the organ fails to be acted on by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent. This, it must be admitted, is a hard doctrine to accept; but there seems to be no doubt about its truth. Now, in bronchitis, at least in children, it often happens that large portions of the lower lobes of the lungs are unacted upon by inspiratory forces, for (as we have seen) the lower ribs and even the sternum are commonly drawn inwards, instead of rising, during the act of breathing. And the same thing occurs in croup and in other diseases attended with laryngeal obstruction. It therefore appears probable that collapse of the lower and anterior edges of the lungs, which is so often seen under such circumstances, is generally, if not always, due directly to the cessation of inspiratory traction upon those parts of the organs. And Bartels, as far back as 1860, showed by dissection that it is in fact often impossible to demonstrate any plugging of the tubes passing to collapsed areas of pulmonary tissue. So, again, we may refer the collapse of the bases of the lungs, which is so commonly seen in the bodies of those who have died of enteric fever, to the shallowness and imperfection of the respiratory movements. Even when collapse depends upon obstruction of tubes, an important factor in its production is a deficiency of power in the muscles of the thorax. For, with strong muscles, there is not only the chance that a fit of coughing may expel a plug of mucus, but also that a vigorous inspiratory effort may succeed in drawing air into the tissue in spite of it. Accordingly, in adults collapse scarcely ever occurs as the effect of primary bronchitis. Even in children its development is greatly favoured by a rachitic state of the ribs, and perhaps also by the muscular weakness resulting from measles or any other acute disease; and the younger the child the more likely is it to show collapse of the lungs under a bronchial attack.

The view that inspiratory retraction of the lower part of the chest is

generally the cause of collapse of the lungs, is not incompatible with the opinion that an extensive collapse due to obstruction of the corresponding bronchial tubes may sometimes in its turn lead to a falling in of the thoracic walls. Dr Gee, for example, describes unilateral shrinking of the chest as resulting in some cases from collapse of the whole of a lung in consequence of plugging of its main bronchus. Generally speaking, however, the space in the pleural cavity vacated by a collapsed portion of pulmonary tissue is filled up by over-distension of other parts of the organ. Moreover, if an entire lung should become emptied of air, the opposite lung undergoes enlargement and displaces the mediastinum.

Some writers believe that pneumonia is apt to arise in collapsed portions of lung tissue, in which case the anatomical distinctions between collapse and hepatisation would of course fail. Lichtheim found in his experiments that acute œdema of the affected lung sometimes occurred, so that, although airless, it was bulky, soft, and moist. The retention of secretion in obstructed tubes is suggested by Jürgensen as a probable cause of inflammatory irritation; and Lichtheim, in some instances in which an animal survived for a considerable time after obstruction of the bronchus, describes the lung as looking almost like a sacculated kidney, full of dilated channels distended with pus. But, as a rule, pulmonary tissue, when collapsed as the result of disease in the human subject, remains uninflamed. If there are patches of broncho-pneumonia in the same organ, their presence is merely a coincidence.

So far as appears, collapse, whether arising from bronchitis or from laryngeal obstruction, is always a temporary condition; for the affected parts of the lung again receive air if recovery takes place from the primary disease. The author has never seen in the *post-mortem* room any unmistakable results of collapse at a former period.

With the exception of the collapse which occurs at the extreme bases of the lungs as the result of inspiratory retraction of the chest walls, this condition, when secondary to bronchitis, seldom affects more than small portions of each organ, lobules or groups of lobules scattered here and there, some on the surface, others in the interior. Hence it does not often give rise to very definite physical signs; though, if many patches should exist in close proximity to one another, it is possible that there would be more or less dulness on percussion, deficiency of vesicular murmur, and perhaps bronchial breathing. As regards symptoms, all that can be said is that collapse aggravates the dyspnœa already produced by the bronchitis.

Chronic Bronchitis.—This, like the *acute* form, varies widely in its degree of importance and of severity in different cases. Some of its mildest forms are seen in children and in young adults, who from time to time have attacks of what is termed bronchial catarrh, until, as they get older, they perhaps ultimately "grow out" of them. Other cases, chiefly in persons advancing in age, take the form of what is called a winter cough. Year after year, during the cold season, they become troubled with a cough, which leaves them entirely in the summer, and which is attended with more or less muco-purulent expectoration. Sometimes it occurs only in the morning, when they rise from bed; sometimes it goes on at intervals throughout the day; sometimes it is very bad at night, so as to disturb their rest. For a time there is not the least dyspnœa. Gradually, however, they find that in muscular exertion, as in walking uphill, or in

mounting stairs quickly, the breath becomes short and hurried. Still it is surprising how little heed is paid to such symptoms, which, among the poorer classes, seem to be taken almost as a matter of course. In making autopsies we repeatedly find the tubes in the lower parts of the lungs filled with pus, and even dilated, and the lungs themselves markedly emphysematous, in the bodies of patients who had perhaps died in the surgical wards of the hospital, and who had never made any complaint of pulmonary symptoms. But after a few years this cough continues even during the summer; and there is constant dyspnœa, the breathing being hurried and wheezing, especially during any exertion. For a long time there is no loss of flesh; but at length wasting occurs, and it may reach an extreme degree.

It is only during the early stages of chronic bronchitis that it is possible for the patient to recover, so as to remain henceforth free from the liability to its return when exposed to cold or damp. But even in advanced cases the disorder may often be kept at bay if the patient is able to avoid changes of temperature, and to spend every winter in a warm climate, or else to remain indoors throughout that season of the year. In this way life may not infrequently be preserved to its natural term.

When death occurs, it is sometimes as the result of an intercurrent acute attack. For those who suffer from chronic bronchitis are exceedingly liable to exacerbations which always cause more or less anxiety, but which may pass off, leaving behind only increased susceptibility for the future.

In other cases chronic bronchitis ends fatally by the supervention of dropsy, exactly like that which attends primary cardiac disease. The right chambers of the heart are then dilated and hypertrophied; and the trunk and branches of the pulmonary artery are thickened, just as in cases of mitral stenosis. In one instance the wall of the pulmonary artery was actually thicker than that of the aorta, and it was also atheromatous. Such changes probably never take place until emphysema of the lungs has developed itself; and the obstruction to the pulmonary circulation is sufficiently explained by the defective aëration of the blood which occurs under such circumstances, and by the diminution of the capillary area in the pulmonary system of vessels. Traube laid stress upon a third factor, namely, the deficiency of the movements of expansion and retraction of the lungs in breathing, which under normal conditions further the flow of blood through the pulmonary capillaries. Ultimately the liver may become "myristicated," the spleen and the kidneys indurated, and the stomach congested. It is, however, remarkable how rarely these results of long and extreme venous congestion are seen in cases of chronic bronchitis with emphysema, compared with their constancy in those of mitral disease. One important element in bringing about these more remote changes is the occurrence of granular degeneration in the muscular substance of the right side of the heart. Another, which has not yet received its due share of attention, is a like degeneration of the diaphragm, as pointed out by the late Mr Callender in the 'Lancet' for 1857, and by Zahn, in vol. lxxiii of 'Virchow's Archiv.'

Certain varieties of chronic bronchitis demand separate mention. One of them is that which Laennec called *catarrhe sec.* A like form of acute bronchitis has been already mentioned. But the cases now referred to are described by Riegel as having a duration of several months, and as often

ending fatally, at least in children. The most prominent symptom is a paroxysmal cough, which is so violent that the face becomes purple and the veins of the neck swell out. Yet there is no sputum, except perhaps a little tough mucus. Pyrexia is very slight, or altogether absent.

Another variety of chronic bronchitis is attended with a remarkable flow of a thin watery albuminous liquid from the mucous membrane, so that the name of *bronchorrhœa serosa* has been given to it. Laennec relates the case of an old man of seventy, who for ten or twelve years spat up about four pints of this watery secretion every day, and yet remained not ill-nourished. But in other instances, as Andral pointed out in his 'Clinique Médicale,' extreme emaciation occurs, with weakness and pallor, almost like what might have been caused by profuse hæmorrhage.

Chronic bronchitis, if it lasts long, gives rise to certain secondary affections of the pulmonary parenchyma and of the air-passages themselves, which add greatly to its gravity. They are known as "pulmonary emphysema," and "bronchiectasis" or "dilatation of the bronchi."

EMPHYSEMA—pulmonary or vesicular emphysema—must not be confounded with that condition of the subcutaneous and other connective tissues which also bears the name of emphysema, and which depends upon infiltration with air as the result of injury to some air-containing structure. What adds to the confusion is that in the lung itself infiltration of air into the sub-pleural connective tissue does sometimes, though rarely, occur, and may go on until, passing along the root of the lung, the air diffuses itself through the mediastinum, and reaches even the superficial fascia of the neck and of the chest; this last affection is technically known as "interlobular" or "interstitial" and "sub-pleural emphysema."

A very complete description of emphysema as regards both its anatomical character and its symptoms was given by Laennec; before then it had almost escaped notice. Its real nature was first pointed out by Rokitansky, who showed that it consists in part of an over-distension of the pulmonary alveoli, but also in part of an atrophy of their walls, causing their cavities to run together into regular spaces, sometimes of very large size.

Over-distension of the alveolar tissue does not in itself justify the lung being called emphysematous. In children who have died after a few days' illness of laryngeal diphtheria, or croup, or acute bronchitis, it is common to find the lungs very bulky, and looking far more open-textured than usual. One is apt to speak of this condition as emphysema. But it is clear that there has been no time in such cases for the occurrence of atrophy of the alveolar walls, and in all probability if the patients had recovered the lungs would quickly have returned to their normal state. Even when some amount of emphysema seems to be really present, it may happen that immediately after an attack of dyspnœa a great apparent increase of the condition can be made out by physical signs, which yet subsides again within twenty-four hours. Hertz, in 'Ziemssen's Handbuch,' mentions the case of an asthmatic patient, aged thirty, in whom he observed such a transitory over-distension of the lungs on several distinct occasions.

Anatomy.—The presence of emphysema of the lungs in the dead body is recognised partly by touch and partly by sight. The substance has a peculiar soft and silky feel; it scarcely crepitates, if at all, when squeezed between the finger and thumb; and pressure upon its surface readily causes

a deep pit, which remains after the pressure is removed, proving the loss of the normal elasticity of the pulmonary tissue. Another effect of the same cause is failure of the lungs to collapse when the chest is opened. They often remain fully distended, and the left lung covers the heart, so that scarcely anything is to be seen of that organ. In a case which was observed at Guy's Hospital in 1868, one lung overlapped the other behind the sternum by an inch and a quarter. A similar condition, which must have been pathological, was found in a body which the writer, when demonstrator of anatomy, froze for the purpose of making a transverse section; a wax model of that preparation, which is now in the museum, shows one lung covering the other for some little distance; there was advanced phthisis, and it is very likely that emphysema also existed, although this was not proved to be the case. In extreme instances the lungs bulge in all directions, both during life and after death, displacing the structures around them. Their apices protrude far above the clavicles; and their bases bulge so that the diaphragm instead of being arched upwards is flattened. In two cases the pericardial sac was pouched inwards at its lower part, so that Dr Moxon described the heart as resting upon and as being separated from the surface of the diaphragm by cushions of lung.

Sometimes there are large bullæ or blebs containing air, of all sizes up to that of a walnut or a pigeon's egg. These are seen chiefly along the anterior borders of the lungs, but sometimes also along their inferior borders or near their roots. Not infrequently the ear-shaped process of the left lung shows a more marked degree of emphysema than any other part. But in some instances, even when the lungs are very highly emphysematous, no large cavities are to be seen.

The tissue if closely inspected is found to be full of spaces of the size of small shot or of millet-seeds. One noticeable appearance is a rounding off of the free edges of the lungs, and their outer surfaces often show marks of the ribs, the intercostal spaces having yielded so as to allow the lungs to bulge outwards. Emphysematous lungs are of a grey colour, mottled with spots and lines of pigment; they are soft and inelastic, but not friable, and their cut surface is dry and bloodless.

The earliest change in a lung that is becoming emphysematous is, according to Rindfleisch, a dilatation of the infundibular cavities into which the alveoli open; according to Hertz, a nearly uniform dilatation of the infundibula, and also of the alveoli themselves. Gradually the alveolar walls waste, until nothing is left of them but small ridges projecting a little way into the interior of an oval or rounded space, into which each infundibular cavity and its alveoli have now become resolved. After a time the septa between these spaces in their turn thin away and become perforated. Thus the result is a progressive increase in the size of the spaces with a diminution of their number. An aggregation of fatty granules round the remains of the nuclei of the alveolar epithelium is commonly present; and in the 'Med.-Chir. Transactions' for 1848 Mr Rainey maintained that the morbid process concerned in emphysema is primarily and essentially a fatty degeneration; but there is no reason to suppose that this is the case. There is, of course, an enormous destruction of capillaries when the affection is at all extensive. Rindfleisch speaks of the vessels as collapsing until "only a narrow ribbon-like band is left, which may be recognised as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like

itself to form the usual anastomotic network." He goes on to say that relatively wide communications are opened up between the pulmonary artery and the pulmonary and bronchial veins. These anastomoses appear in well-injected lungs as peculiar elongated unbranched channels of the same diameter throughout, strikingly contrasting with the far more numerous, extremely tortuous, and dilated arteries, for the contents of which no such supplemental mode of escape has been provided.

Pathogeny.—With regard to the mode of origin of emphysema of the lungs there have been many different opinions.

Laennec's idea was that the tubes in cases of bronchial catarrh being obstructed by swelling, or by an accumulation of mucus, the air which found its way into the alveoli during inspiration became unable to escape during expiration, inasmuch as the expiratory force was less than the inspiratory. In other words, he thought that emphysema was the result of a process the exact converse of the ball-valve action which, as we have seen, was supposed by Gairdner to be the cause of collapse. Louis objected that the ordinary seat of catarrh is the base and lower part of the lung, whereas the parts most apt to be affected by emphysema are the apex and the anterior margin.

In 1851 Gairdner advanced the theory that emphysema arises solely during inspiration. His view was that collapse or reduction in bulk in one part of a lung is a necessary antecedent to the development of emphysema in another part. During inspiration, when the chest becomes enlarged, if each and all of the lobules cannot expand to fill it, some of them must be stretched unduly; and this produces emphysema.

For a few years Gairdner's view was widely accepted, and emphysema was held to be essentially "complementary" or "compensatory" either to collapse, or to retrogressive tubercular disease, or to some other contracting lesion of the lung. But in 1856 Sir William (then Dr) Jenner addressed to the Royal Medical and Chirurgical Society a powerful argument, in proof that the development of emphysema occurred during expiration. And it is now known that this same doctrine had already been taught in Germany, as far back as 1845, by Mendelssohn, in a work entitled 'Der Mechanismus der Respiration und Circulation.' Gairdner urged that it is impossible for emphysema to be produced by the act of expiration, even with a closed glottis, because the force by which the air becomes compressed within the lung opposes exactly as much resistance without as it creates pressure within. Jenner now pointed out (as Mendelssohn had done before) that certain parts of the thoracic walls are yielding, and consequently incapable of maintaining this resistance. Both these observers indicated the apices of the lungs as being devoid of adequate protection against an expanding force from within; and we have seen that Louis had long before shown that the apices were especially apt to become emphysematous. Jenner remarked that during a fit of coughing the supra-clavicular regions may be seen to bulge, and that by placing one's hand upon them one can feel that they are distended by a considerable force. If the apices are the seat of emphysema this bulging under violent expiration is extreme, and percussion over the bulging parts may elicit an almost tympanitic sound. He further showed that the upper costal cartilages are to some extent yielding, and that therefore the alveoli of the anterior margin of each lung become affected with emphysema as well as those of the apex. Other parts which he also named as apt to become emphysematous were—

the margin of the base of the lung, the part of the lung near its root below the entrance of the bronchus, and the little ridge of lung which, on the right side, projects behind the trachea. The base of the left lung generally he showed to be less firmly supported than that of the right, the liver being more unyielding than the stomach; and he cites Louis as having found the left lower lobe emphysematous twice as often as the right one.

The correctness of Jenner's theory has since been supported by observations made in certain cases of congenital malformation of the chest walls. Thus in the case of Groux, who had a fissure of the sternum, the anterior part of the lung protruded through the fissure in the act of coughing. Ziemssen met with an example of absence of the *pectoralis minor*, and of the entire sterno-costal part of the *pectoralis major*, so that the intercostal muscles of the four upper spaces were covered only by fascia and by integument. During forced expiration these spaces bulged from 1 to 1½ mm. above the level of the ribs; when the muscles of one space were faradised that space for the time remained flat, the others bulging as before. Further evidence is afforded by cases in which, after the cicatrisation of penetrating wounds of the chest, the affected parts have ultimately become the seat of hernia of the lung, as the result of weakening of the thoracic parietes. Many such instances may be found collected in a little work published by Desfosses in 1875. It may be noted, too, that horses are liable to an affection of the lungs identical with emphysema, as the result of the straining efforts which they are called on to make, during which they keep the glottis closed. As Sir William Jenner says, in vol. iv of 'Reynolds' System,' "no one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort the horse holds its breath, having previously inflated the lungs. No sooner, however, does this animal cease its effort than the glottis is opened, and the air suddenly expressed from the lungs. The degree to which the air was compressed may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapours is seen to be driven forth." In his paper in the 'Med.-Chir. Transactions' Jenner had shown that the parts of the lungs that are emphysematous in a "broken-winded" horse are those which are so placed as to be least able to resist pressure. It is curious that a capital description of this affection of horses was given before the end of the seventeenth century by Sir John Floyer, in a treatise on asthma; the passage is cited in full in 'Watson's Lectures.'

Emphysema, then, is the result of expiratory pressure with a closed, or partially closed, glottis. The expiratory muscles forcibly compress the air within the chest, and if all parts of the thoracic parietes were equally unyielding no harm would result. But as certain parts can and do yield, some of the compressed air is driven into the corresponding alveoli of the lungs, and gradually breaks down their structure in the manner already described. No doubt the resistance of the chest walls in different regions fails progressively, more and more, as the affection advances. Thus the sternum and the upper cartilages become arched forwards, a change which probably is due to the frequently repeated application of an expansile force from within the thorax. It is only as the result of long-continued pressure that the diaphragm can become flattened, and that the lung can protrude inwards beneath the heart, as described above.

Even when a part of the lung (generally the apex) is shrunken by retrogressive tubercular disease, most pathologists now agree with Jenner in

thinking that the development of emphysema in the tissue around, particularly along the anterior edges, is due to the pressure of air driven into the alveoli by coughing. Others, however, still hold that to such cases Gairdner's theory remains applicable, and that the emphysema is "complementary" in the strict sense of the term.

Although a frequently repeated cough is one of the chief causes of emphysema, yet in man, as in horses, other actions beside coughing may increase the expiratory pressure so as to produce the same result. This is the case, for example, with all violent efforts in which the glottis is kept closed to fix the chest. Dragging or lifting heavy weights, straining at stool, even the act of parturition, may be mentioned as possible causes of emphysema. Thus Waldenburg is cited by Hertz as having seen the affection develop itself in a medical student who, having come from a country place where he had no occasion to ascend the stairs, occupied in Berlin an apartment on the fourth floor, up to which he ran without stopping several times daily. Hertz himself met with a similar case in a young shopman, whose lungs became emphysematous in about a year, without any cough or bronchial catarrh, as the result of his having to carry heavy goods up a high staircase in haste a great many times every day. For persons who have suffered from bronchitis, and in whom the distending process has already begun, it is most important to avoid all occupations or amusements that involve repeated expiratory efforts. Playing a wind instrument may sometimes be exceedingly injurious; for, although the glottis is not closed, the air within the chest is kept under great pressure, while it is being slowly allowed to escape.

Atrophic emphysema.—Of late, German observers have been disposed to attribute emphysema in part to primary changes in the pulmonary tissue independent of the mechanical conditions just described. A *senile atrophy of the lungs*, bearing a close resemblance in its characters to emphysema, is generally admitted by pathologists; it was originally described by Dechambre in 1835, from observations made at the Salpêtrière. Sir William Jenner speaks of it as "*small-lunged emphysema*," in contrast with the ordinary form of the disease, which he calls "large-lunged emphysema." As he says, the small size of such lungs, their lightness, and the very small space into which they may be compressed, are often most remarkable. When the thorax is opened after death they "fall in like an inflated bag of wet paper." The subjects of senile atrophy of the lungs are commonly thin, shrivelled, and withered-looking. Their chests are very small and narrow, the lower ribs being so obliquely placed that they almost reach the crest of the ilium, and so closely packed as nearly to come in contact with one another. The lungs are so reduced in size that the extent of præcordial dulness may be increased, notwithstanding that the heart partakes of the general wasting. Yet there is commonly little distress of breathing, because the volume of the blood is at a minimum, and because the deficiency of muscular power forbids active exercise.

It has always, however, seemed to the author that the supposed likeness of this senile atrophy of the lungs to emphysema is really due to the fact that a slight degree of emphysema resulting from the bronchial catarrh to which aged persons are so liable is commonly mixed up with it. Hertz, in his chapter on atrophy of the lungs in 'Ziemssen's Handbuch,' speaks of bronchitis as a frequent "complication;" and he also mentions that the bronchioles are very thin and "generally uniformly dilated, seldom irregularly sacculated." But brochiectasis can hardly be otherwise than mechanical

in its origin; and it seems reasonable to take the same view of the pulmonary rarefaction, which Hertz describes as being most marked at the apices and along the anterior edges.

Predisposition.—That in younger persons some cause for emphysema must exist, beyond expiratory pressure upon the alveoli, is argued by Hertz from the fact that in certain families several members are found to suffer in succession, as the result of comparatively trifling affections of the air-passages. Schnitzler, for example, saw three brothers, whose parents were still alive and well, but who all became the subjects of emphysema at the age of thirty, without definite cause. Walshe cites Dr Jackson, of Boston, as having upheld the view that the disease is transmitted by inheritance; he found that "of twenty-eight emphysematous persons, eighteen had either a father or a mother, or both, similarly affected; whereas of fifty non-emphysematous people, three only sprang from emphysematous parents." Dr Greenhow once argued for a relation between emphysema and the "gouty diathesis."

So far as concerns the mere occurrence of this affection in different members of the same family, it is important not to overlook the fact that they may all have been alike exposed to the causes of bronchial catarrh, and perhaps all alike unduly susceptible of taking cold. But Cohnheim and other recent German writers have looked for an explanation of emphysema, apart from mechanical causes, in a varying physical state of the pulmonary tissue, as regards its degree of elasticity. In this connection certain observations of Perls may be cited of which there is a record in vol. vi of the 'Deutsches Archiv.' By means of a pressure gauge he determined in a large number of cases the degree of force with which the lungs retracted when the pleural cavities were opened in the dead body; and he found that after death from enteric fever, or (in one case) from phosphorus-poisoning, their elasticity was reduced almost to nothing. Cohnheim regards it as an established fact that in a very large proportion of cases emphysema depends upon a congenital defect of development in the elastic tissue of the lungs. But the reference which he gives to a paper by Eppinger in the 'Prag. Vierteljahreschrift' for 1876 does not seem to bear out this assertion; for although Eppinger found that even in slightly emphysematous lungs there was a great reduction of the network of elastic fibres in the alveoli, the smallest fibres having completely disappeared, he yet appears to have regarded this as a change occurring in the course of the development of the disease, and not as an antecedent malformation.

In this connection a case recorded by Hertz is of great interest. It is that of a regimental cornet-player, aged thirty, who had always been able to use his instrument without any difficulty, even on the march. He was attacked with double pneumonia, which, however, subsided in a week; he felt quite well, had no cough, and began again to practise with his cornet. But in the course of the next seven months he discovered that he was no longer able to take sufficient air into his lungs to maintain a long note for the proper period, that he was short-breathed on exertion, and could no longer play while marching. Hertz found on examination that the lungs were markedly emphysematous, which had not formerly been the case; and his supposition is that the pneumonia had damaged their texture, so that they were not able to resist expiratory pressure as before.

Clinical symptoms.—The recognition of emphysema is based mainly upon physical signs. The chief subjective symptom of the disease is *dyspnœa*. The patient first notices that he is short of breath when he exerts himself,

as in running upstairs; but after a time difficulty of respiration becomes a permanent condition from which he is never entirely free. As Dr Walshe says, he feels as if his chest were never emptied of air as it naturally should be; and he is conscious of an annoying sense of inflation or distension. It is true that many emphysematous persons affirm that their dyspnœa is only occasional; but Dr Walshe says that in all such cases which he has seen, the patient has been deceived, a moderate amount of dyspnœa having become to him a second nature—a thing unperceived and giving rise to no discomfort.

It does not necessarily happen that the affection advances; it may continue stationary, and life may be maintained until extreme old age, provided that the risk of intercurrent attacks of bronchitis can be obviated. Hertz supposes that the existence of emphysema necessarily involves an increased liability to bronchial catarrh, by leading to congestion of the mucous membrane of the air-tubes; but it would be difficult to prove this, seeing how very common bronchitis is as an antecedent condition.

On the other hand, in the majority of cases emphysema becomes more and more marked every year. After a time the noisy hurried breathing may become so short that the patient cannot utter a sentence without stopping in the middle of it. At night he has to be propped up by pillows. His distress becomes aggravated from time to time, sometimes by an exacerbation of bronchial catarrh, sometimes by the supervention of asthma, sometimes by mere pushing upwards of the diaphragm, as the result of distension of the abdomen with flatus, or with undigested food. The face, the hands, and at last the whole body become livid, as if from capillary bronchitis. In fact, chronic bronchitis never goes on for any length of time without being complicated with more or less of emphysema, so that it is impossible to separate the effects of each. Cough, on the other hand, may be entirely absent in cases of emphysema unless there is bronchial catarrh. But many persons, whose main disease is emphysema, constantly have cough, and expectorate a frothy liquid, or pearly-grey masses of mucus.

Hæmoptysis is not generally counted among the symptoms of this affection. Sir Dyce Duckworth, however, in the 11th volume of the 'St Bartholomew's Hospital Reports,' declares that it is of not infrequent occurrence. In 1869, a woman, aged forty-nine, was brought dead into Guy's Hospital after an attack of severe hæmoptysis, and at the autopsy the only disease that could be discovered was an extreme degree of emphysema of the upper lobes of the lungs, with some excess of fibrous tissue forming the interlobular septa. The air-tubes were full of clots. She was said to have suffered for three months from wheezing and short breath; on the morning of her death she woke up at 4.25 a.m. with "coughing and vomiting of blood through the nose and the mouth, and was suffocated in ten minutes."

Ultimately emphysema gives rise to great wasting, and to extreme enfeeblement of the muscular strength. A noteworthy circumstance, mentioned by Walshe, is that the over-distension of the chest renders the body unnaturally buoyant in water, so that the patient is astonished to find himself able to swim more easily than before, at the very time when he is growing more and more incapable of other kinds of exertion.

Physical signs.—Of these the most important are derived from *percussion*. Even very slight degrees of emphysema may be detected by carefully mapping out the areas of the heart and of the liver. Instead of beginning

at the upper border of the fourth left costal cartilage, the cardiac dulness begins only over the fifth or even the sixth cartilage. Instead of beginning at the upper border of the sixth right rib, the hepatic dulness begins only at the level of the seventh or of the eighth. If in a case of bronchitis one finds that percussion over these two organs yields normal results, one is generally safe in declaring that no appreciable amount of emphysema has yet developed itself.

As the disease advances, the heart becomes so completely covered by the lungs that no cardiac dulness at all can be detected, the pulmonary resonance above meeting the tympanitic note of the stomach below. At the same time the apex-beat ceases to be felt in the normal position, in consequence of the downward displacement of the diaphragm carrying the heart with it; and the cardiac pulsations can often be felt in the epigastrium. On the right side pulmonary resonance now extends down to the margin of the thorax. The edge of the liver may sometimes be felt in the hypochondrium. But very often this is not the case, and there may be a marked reduction in the area of the hepatic dulness, so that one may be tempted to suspect that the organ is atrophied or cirrhosed when this is not really the case. This seems to be due to the great increase in the antero-posterior diameter of the thorax preventing the liver from coming in contact with the parietes of the chest and abdomen to the normal extent. Another noticeable feature of well-marked cases of emphysema is the clear character of the percussion-sound over the sternum as high as its upper border; and over the bases of the lungs behind a clear percussion-sound is elicited to a much lower level than normal.

Moreover the percussion-sound, over parts of the chest where it ought naturally to be resonant, is over-resonant, and according to Walshe and Gee there is often a fall in its pitch. The change in the percussion-sound is often very conspicuous over the back of the lungs, a sonorous drum-like note being readily produced where normally there is often difficulty in eliciting a clear resonant sound.

Next to percussion, *inspection* affords the most valuable indications of emphysema. As already explained, the sternum and the upper ribs arch forwards as the result of their yielding to the frequently repeated expiratory pressure which is the cause of the affection. The sternum not infrequently becomes convex in a vertical plane, with an angle, known as the *angulus Ludovici*, at the junction of the manubrium with the body. The clavicles also are more bent than under normal circumstances. The curve of the dorsal vertebræ becomes greatly increased, so that the back is rounded. The effect of all these alterations in the parietes of the chest is to give it a cylindrical form. It is often aptly said to be "barrel-shaped." By the cyrtometer its horizontal circumference is shown to be almost perfectly circular, as is well illustrated in a diagram given by Dr Gee. Sometimes the rounding of the ribs and of their cartilages continues to the very bottom of the thorax, and the hypochondriac regions are permanently expanded to the fullest possible extent. One result of this is, as Hertz has pointed out, a transverse groove, which crosses the abdomen horizontally from one twelfth rib to the other; it is due to the stretching of the upper part of the transversalis abdominis muscle, which is fixed to the rib cartilages, as compared with the relaxed condition of the lower part, which has no such attachment. This groove may form a conspicuous, and at first sight puzzling feature of a case, when there is at the same time a considerable accumula-

tion of fluid in the peritoneal cavity. But in other instances the lower ribs and their cartilages are flattened, or even hollowed inwards; this occurs especially when the pulmonary affection began in bronchitis or whooping-cough at an early period of life, so that the bases of the lungs became collapsed.

The "subcostal" angle at the ensiform cartilage is far more open than usual, as obtuse as in deep inspiration.

The upper intercostal spaces in emphysema are either unaffected, or narrowed, while the lower ribs are widely separated. Stokes declared that he had never seen the spaces otherwise than depressed; but Walshe and others say that it is not uncommon for them to be prominent or bulging when the parts of the lungs beneath are highly emphysematous.

The high shoulders, increased depth of chest, short and full neck, raised ribs, closer together above and wide apart below, with the obtuse substernal angle, are the physiological characters of the chest during deep inspiration, and can be imitated by filling the lungs and holding the breath. The same fulness of the chest may be the result of distension of the pleura by fluid on one side or the other, but when bilateral it is characteristic of emphysema alone. It is the exact opposite of the "expiratory" form of chest seen in advanced phthisis.

Further indications as to the presence of emphysema, and as to the extent to which it is advanced, are yielded by observation of the act of breathing. During inspiration the chest in well-marked cases is seen to be almost motionless. It is, indeed, impossible for the upper ribs to rise and expand, as they normally should do, because they have permanently assumed a position in advance of what could have been reached in health. There is, however, a jerking movement upwards of the thorax as a whole, produced mainly by contractions of the sterno-mastoid and scalene muscles, which start into unnatural prominence, and appear to be hypertrophied. If the lower ribs are thrown outwards as much as the upper ones, the chief agent in inspiration must be the diaphragm. But in many cases the state of affairs is reversed. The lower ribs still retain a certain degree of mobility, but the diaphragm is pushed downwards, so that it can do little towards enlarging the thoracic cavity. It may then be observed that the epigastrium becomes hollowed during the act of inspiration.

Violent cough causes bulging of the supra-clavicular spaces; and in cases of emphysema Sir William Jenner warns against confounding this condition at the root of the neck with prominence of the same part due to distension of the veins. A still more important source of error is the sudden protrusion of an aneurysmal sac during the act of coughing. Another point to which Jenner has drawn attention is, that if one is feeling the pulse of an emphysematous patient while he coughs violently one perceives the artery to become suddenly full and tense, and it ceases for a moment afterwards to beat.

Auscultation gives comparatively little information in cases of emphysema, apart from signs of the bronchitis that is so commonly associated with it. The most marked sign of the emphysema itself that is given by the stethoscope is enfeeblement or nearly complete absence of the vesicular murmur. The bronchial breathing which may normally be heard over the roots of the lungs behind, and on the sternum, is often wanting in emphysematous patients; but even in those who are healthy this is not infrequently the case. The expiration is exceedingly prolonged owing to the

loss of elasticity in the lungs. Walshe says that, instead of being only one third the length of the inspiration, it may be four times as long; in other words, its relative duration may be increased twelvefold. The growling, squeaking, or wheezing expiratory sound of emphysema is very commonly spoken of; but it seems doubtful whether this sign does not belong rather to a concomitant bronchitis than to the pulmonary lesion itself. In one case, at Guy's Hospital, as the tubes became free, this sound entirely disappeared, although the extremely feeble state of the inspiratory murmur and the over-resonance of the percussion-sound clearly showed that emphysema still persisted.

In cases of emphysema the *vocal resonance* is much diminished, and tactile vocal *fremitus* is often entirely lost.

In the latter stages of pulmonary emphysema a tricuspid regurgitant murmur may often be detected; and even at an earlier period increased pressure in the pulmonary artery may be indicated by an accentuated second sound. Epigastric pulsation and pulsation of the veins of the neck with each beat of the heart may also be observed. These symptoms are all the result of obstruction in the pulmonary capillaries having led to dilatation of the right side of the heart and incompetence of the tricuspid valve.

When air has escaped into the subpleural connective tissue, constituting what is known as "interlobular" or "interstitial emphysema," there is sometimes heard a friction-sound very like that which occurs in pleurisy. This sign was originally noticed by Laennec. Most writers since then have been disposed to doubt the correctness of the observation; but it has been recently confirmed by Dr Gairdner; and Dr Hudson, in his edition of Stokes's works, says that he also met with a case in point.

The *treatment* of emphysema will be considered with that of bronchitis at the end of the present chapter.

Hypertrophy of the lungs.—The barrel-chest above described, *i. e.* the thorax when filled by the ordinary or "large-lunged" emphysema, is closely simulated by a condition which is physiological rather than diseased, and may be described as uniform overgrowth of the lungs. It occurs to some extent in all healthy persons who are engaged in hard manual labour or in athletic sports. The chest of the youth who improves his wind by rowing, or of the workman who is accustomed to carry heavy weights, becomes expanded, and the lungs increase in volume and capacity. When the efforts made with the glottis closed are too great, rupture of the air-vesicles is apt to occur, and thus true emphysema may be mingled with physiological hypertrophy; but this is far from a necessary complication.

Ascending ladders and climbing hills, especially when a weight is carried on the shoulders, has a similar effect. Greater efforts of inspiration are made, the lungs are expanded more perfectly, and the breathing capacity of the chest is increased.

Habitually breathing rarefied air produces similar but exaggerated effects. In persons who live in mountainous regions both causes of hypertrophy of the lungs combine; they are frequently ascending heights, often with burdens on their shoulders, and they are constantly breathing air at a low pressure, so that each cubic inch of lung gives them less oxygen to aërate their blood. The result is that the thorax becomes enlarged in all its dimensions. Instead of a yard's girth for a man of moderate stature, and a metre's

for a tall man, the chests of men of five feet six inches or even less measure more than forty inches in circumference.

This condition may be observed in Swiss guides; but it has hitherto been found most constantly and highly developed among the natives of the lofty table-lands of the Andes, in Bolivia.

DILATATION OF THE BRONCHI; BRONCHIECTASIS.—This condition was first described by Laennec. It is seldom a primary disease, and is often found associated with fibroid induration of the tissue between the enlarged tubes; such cases will best be discussed in the account of Cirrhosis of the lung. In other cases the pulmonary parenchyma is either healthy or emphysematous or collapsed: the bronchiectasis is the result, not of interstitial pneumonia, but of bronchitis.

Anatomy.—All writers divide bronchiectasis into cylindrical and sacculated, and the definition is of pathological and clinical importance.

In the *cylindrical* form the tubes sometimes run through the substance of the lung, with but little diminution of calibre, until they end abruptly beneath the pleural surface; their appearance is often compared to that of the fingers of a glove, but it rarely happens that the dilatation is so uniform throughout their whole length. In one instance, which occurred at Guy's Hospital in 1873, the bronchial tubes were so enlarged as to be conspicuous at the root of the lung, "pushing the lobes apart from one another," as it is expressed in the report of the autopsy. More often the medium-sized and smaller tubes are alone affected. The existence of bronchial dilatation may then be obvious on the cut surface of the lung, far too many large orifices being visible, from which pus wells up in great quantity; or, in order to detect the enlargement of the tubes, especially if they are empty, it may be necessary to open them up with scissors. Judging from the author's own observations, slight forms of bronchiectasis are much more often met with in the extreme bases than in any other parts of the lungs. Not infrequently he has seen enlargement of the principal tube passing into the ear-shaped process of the left lung when no such change could be made out elsewhere. This was when that process itself was emphysematous; and, indeed, these less-marked examples of bronchiectasis are very often found in association with emphysema. On the other hand, when the bronchial affection reaches an extreme degree, there is seldom a corresponding amount of rarefaction of the pulmonary tissue. If the tubes in any one part of the lung are universally dilated, running to the surface side by side, and perhaps as large as quill pens, or even larger, the parenchyma between them is necessarily reduced to a small space. In these cases it is sometimes difficult, at the first glance, to say whether the affection is or is not secondary to a fibroid change in the lung itself. But in the most marked and typical cases of cylindrical bronchiectasis, such as occur in children after whooping-cough or measles, the tissue between the dilated tubes is often quite free from induration or other morbid changes.

It must be understood that cylindrical bronchiectasis is by no means absolutely uniform in diameter at different points. Sometimes the tubes gradually widen as they approach the surface. Sometimes they have fibrous bands or trabeculæ projecting from their walls here and there, and rendering their calibre very irregular.

Sacculated dilatation of the bronchial tubes, also, varies widely in character in different cases. The most typical form is one which presents

appearances that have scarcely yet been exactly described. When a section is made of the lung the cut surface appears to be covered with an immense number of shallow, smooth-walled depressions, like so many minute saucers. Each of these has in its floor a very small rounded orifice, and it is obvious that they are all sections of small bronchiectases, which probably were spherical before they were cut across, but have become flattened by their own elasticity, and by that of the adjacent pulmonary tissue. In all likelihood, if the lung in such a case could be inflated and dried, and the parenchyma then cleared away so as to expose the tubes in their continuity, each would be found changed into a regular series of globular dilatations, so as fairly to deserve the epithet moniliform. In other instances sacculated bronchiectases are more unequal in size and irregular in form; but it is probable that most of such cavities are really not dilated tubes, but smooth-walled vomicæ which have been formed by ulceration. It is to these latter sacs that in all probability the statement of Biermer applies (in vol. v of 'Virchow's Handbuch'), that the majority of the sacculi of bronchiectasis have openings only towards the trachea, and are closed on the distal side. Still less are true bronchial sacs ever converted into shut cysts.

Dilated bronchial tubes often have exceedingly delicate walls, being apparently thinner than they were before they began to increase in size. But sometimes the tissues are thickened, the lining membrane being velvety and showing the changes above described as occurring in bronchitis.

Pathology.—As to the way in which bronchiectasis arises, there can be little doubt that it is the mechanical result of expiratory pressure, being in fact produced by exactly the same cause as emphysema. To explain satisfactorily why in one case the alveoli should yield, and in another case the tubes, would perhaps be impossible. But we may fairly suppose that it depends upon the degree to which the walls of the tubes have been softened by inflammation, and also, it may be, upon the amount of elastic resistance originally possessed by the lungs and bronchi in each individual. (See Dr Grainger Stewart's paper, 'Edin. Med. Journ.,' July, 1867.)

A more frequent cause of dilated bronchi is chronic interstitial pneumonia, but with this form we are not at present concerned.

Independently of previous organic changes in the lung, bronchiectasis is not frequent either in children or adults. It sometimes complicates emphysema, but more frequently takes its place.

In children it is most often a sequel of whooping-cough, and sometimes comes on very early, and so severely that the patient grows up in a state of permanent cyanosis, with cold extremities and short breath.

In adults bronchiectasis occurs in middle rather than advanced life, and is almost always preceded by bronchitis, seldom by tubercular disease, and never by acute pneumonia.

Diagnosis.—It is only when bronchiectasis has reached an extreme point that it is characterised by definite *physical signs*. As a rule, the chief indication of this affection is the presence of râles which appear to be too large and coarse to be formed in the undilated tubes of the part of the lung in which they are heard, as, for instance, at the extreme base, or along the anterior edge. If, however, a number of tubes cylindrically dilated are arranged side by side, while the lung tissue between contains but little air, there may be more or less marked bronchial breathing, bronchophony, and even dulness on percussion. But such a case could not be clinically distinguished from primary chronic pneumonia, with bronchiectasis as a result.

Again, the question of the diagnosis of a saccular dilatation of a bronchial tube from a phthisical vomica, to which stethoscopists formerly devoted themselves with much ardour, applies to the dilatation which attends cirrhosis of the lung, not to cases arising out of bronchitis alone. One circumstance which is strongly indicative of bronchiectasis is for the physical signs over a certain part of the lung to undergo more or less regular variations from time to time, being now well marked, and now again indistinct or absent.

As for its *symptoms*, dilatation of the tubes is of course in part concerned in causing cough, dyspnœa, and lividity in patients affected with it. But one can never clinically separate its share in producing these effects from that due to the bronchitis which is always associated with it, and perhaps also to concomitant emphysema. The only thing that enables one to diagnose bronchiectasis with confidence is a peculiar way of expectorating which may in some cases be observed. For some hours, perhaps, there is no cough at all. During this time liquid is accumulating in the dilated parts of the air-passages, the sensitiveness of which appears to be blunted, so that they do not resent its presence. At last perhaps some runs over into a tube which is still healthy. The result is a more or less violent fit of coughing, by which all the liquid that has collected is suddenly expelled, pouring out of the patient's mouth, and even through his nose, so as to half fill his spittoon. Sometimes this process is set in action by percussion of the chest. Sometimes it occurs, especially in the morning, when the patient rises from the recumbent posture. When this accumulation has been got rid of he usually feels much more comfortable than before, the breathing is easier, and the chest less oppressed. Such intermittent expectoration (or in children vomiting) of pus is a sure sign of dilated bronchi.

Fœtid or putrid bronchitis.—It is especially in cases in which the bronchial tubes are dilated that bronchitis becomes accompanied with the expectoration of foul-smelling sputa, and sometimes with horrible fœtor of breath. Traube has, indeed, recorded one or two cases in which putrid bronchitis occurred without there having been any bronchiectasis; but as a rule it is only when liquids have been long stagnant in some part of the air-passages, or in a space communicating with them, that putrefactive chemical changes occur.

The characteristic symptom of this rare disease, as was first pointed out by Traube, is the presence in the matters expectorated of certain soft, friable, smooth masses, of a dirty greyish-yellow colour and very fœtid odour, varying in size from a millet-seed to a bean. Such bodies had been originally noticed by Dittrich in 1850 as plugging the affected tubes in fatal cases; and in Germany they are commonly called "Dittrich's" or "Traube's plugs" (*Bronchial-pröpfe*). Microscopically they are made up of pus-cells and granules mixed with oil-globules. Later on, they also contain certain long, narrow, acicular crystals, of which Virchow gave a description long ago in the first volume of his 'Archiv,' as consisting of a fatty acid. These crystals are colourless, often sharply bent or twisted, sometimes collected together in sheaves or in thick bundles. Some of them may appear to be varicose, a condition which Traube has shown to be the result of pressure by the cover-glass. According to Guttmann they contain a combination of palmitic and stearic acids. In 1867 Leyden and Jaffé further pointed out, in vol. ii of the 'Deutsches Archiv,' that under high powers the granular detritus is composed of fungi, some round, others rod-shaped, others forming beaded chains or

filaments (*Leptothrix pulmonalis*). Their presence appears to be the reason why iodine often gives a purple or a violet, or even a blue tint to the whole mass, as Virchow and Gamgee independently observed. Chemical analysis of the sputum of putrid bronchitis, in different cases, has also shown that it may contain volatile fatty acids (valerianic and butyric acids), leucin and tyrosin, ammonia, and sulphuretted hydrogen.* It is also worthy of mention that Leyden and Jaffé succeeded in inducing in ordinary muco-purulent sputum, outside the human body, a putrefactive process closely analogous in its results to that which must be supposed to give its peculiar character to the expectoration of patients with putrid bronchitis.

In putrid bronchitis the sputum as a whole is generally very abundant. It often separates in the spittoon into three layers: of these the uppermost is muco-purulent, opaque, greenish yellow, and frothy; the middle is a transparent albuminous liquid like serum; the lowest is granular, and of a dirty yellow appearance, consisting of swollen pus-cells and their remains, together with Dittrich's plugs. Sometimes the sputum is of a uniform chocolate colour.

The odour of the patient's breath and sputum in cases of putrid bronchitis is often identical with that of gangrene of the lung. But in other cases it is of an altogether different character. There is, of course, great difficulty in defining the distinction verbally; Guttmann compares it with the smell that pervades a soap factory. Dr Laycock's statement is that in one of his patients the odour was like "that of the may-flower or of apple-blossom, with a kind of *arrière goût* of fæces." The author has often observed this kind of smell, especially when the dilated bronchial tubes were emptied with a gush of enormous quantities of fluid after the manner described above. Probably in such cases there is no active process going on in the walls of the affected tubes themselves. On the other hand, in many of those cases in which the odour is like that of gangrene of the lung the development of fœtor in the sputa indicates the abrupt commencement of a destructive change, both in the air-passages and in the pulmonary parenchyma, which rapidly brings about a fatal issue.

The credit of having first pointed out the clinical features of cases of this kind belongs to Dittrich (1850). His description is that it "commonly arises in persons of the middle period of life, who have suffered for years from bronchial catarrh, with abundant muco-purulent expectoration, and who may either have already begun to waste, or may still remain well nourished. Suddenly, and without apparent cause, the sputum becomes offensive, of a dirty grey colour; the breath also stinks, poisoning the air around. Thereupon follow severe dyspnœa, fever of typhoid character, rapid collapse, an earthy, dirty yellow complexion, and ultimately cessation of expectoration, coma, and death." At the autopsy the walls of some of the bronchial tubes are found intensely inflamed and sloughing. There are more or less extensive areas of pneumonic consolidation, passing here and there into gangrene. Other parts of the lung tissue are œdematous, exuding a fœtid liquid. The bronchial glands are swollen, soft, and of a dirty-grey colour. Several like cases were afterwards recorded by Traube.

* The authority for including methylamine and acetic acid in the list is Dr Gregory, of Edinburgh, as reported by Dr Laycock in the 'Med. Times and Gazette' for May, 1857. See, however, a valuable paper on the chemical characters of fœtid expectoration by Dr Gamgee ('Edin. Med. Journ.,' March, 1865), and one by Bamberger in the 'Würzburger med. Zeitschrift,' 1864.

The occurrence of hepatisation and gangrene of the lungs is by no means limited to those regions which were before the seats of bronchiectasis. Pneumonic patches may be scattered throughout every part of the organ on each side; and it seems obvious that many of them owe their origin to the inhalation into healthy tubes of particles of putrid *débris* derived from others which are already diseased.

But the issue of putrid bronchitis is not always thus serious. Slight cases sometimes end in recovery. We must then suppose either that there has been no ulceration of the walls of the tubes, or that the necrotic process has been limited, and that healing had taken place after detachment of sloughs of no great size. Other cases, again, run on for months with but little change in the symptoms, and without marked impairment of the general health. The general treatment is that of chronic bronchitis (p. 979). The special treatment of the fœtor is that of pulmonary gangrene (p. 1017).

To complete this account of the effects of dilatation of the bronchial tubes it must be mentioned that in vol. xv of the 'Deutsches Archiv' Gerhardt has recorded two cases in which painful swelling of some of the joints occurred as a sequela. He was inclined to regard this as analogous to gonorrhœal synovitis, or to that which sometimes follows dysentery.

The occasional supervention of abscess of the brain as a complication of suppuration in the air-passages or in the lungs has been referred to elsewhere (*supra*, p. 626).

Ætiology of bronchitis generally.—The chief cause of ordinary acute and also of chronic bronchitis is *exposure to cold*. In all probability cold air entering the air-passages through the mouth sometimes acts upon them as a direct irritant. The nasal mucous membrane warms the inspired air; but when persons venture out of doors in cold weather, with a bronchial surface already sensitive, there is often catarrh of the nasal mucous membrane which compels them to breathe through the mouth. The analogy of so many other inflammatory affections of internal structures, which contain no tubes communicating with the external atmosphere, is in favour of the view that bronchitis may also be set up by the action of cold upon the surface of the body; and this is borne out by the fact that in many cases there is no reason to suppose that cold air has been inhaled. A patient may, for example, "take a chill" by getting wet through, by sitting in a draught, by lying on damp grass, by merely remaining motionless out of doors when perspiring profusely after exertion. As a rule, it is especially when the body has been heated and is cooling that danger of catching cold exists. The reason appears to be that, whenever the body has more heat to dispose of than is required to maintain its temperature, the cutaneous capillaries become dilated to allow as much loss of heat as possible. This is equally true whether the heat is supplied to the organism from without or generated in its interior, as the result of muscular exertion. Accordingly, after a Turkish bath, one plunges for a few seconds into cold water, which causes contraction of the blood-vessels, before one ventures to sit in a room at an ordinary temperature. Rosenthal, and afterwards Riegel, have shown by direct experiment that if animals after exposure to great heat are removed and placed in air which is not warmed, they go on cooling until their temperature falls below the normal point. They therefore suggest that, when a person catches cold,

what occurs is that blood from the surface of his body, chilled by loss of its heat, is carried to deeper structures, until they also become less warm than natural. If now there be anywhere a weak spot, it suffers and becomes inflamed. In most cases probably the first effect of external cold is to set up sore throat and coryza, and then the inflammation spreads to the trachea and bronchi.

Some persons are far more sensitive to the action of cold than others. The risk of catching cold may, however, be often obviated to a great extent by "hardening" the skin, that is, by exposing it regularly to sudden changes of temperature, so as to accustom its vessels to contract promptly and vigorously. The best way of doing this is doubtless to sponge the surface with cold water, or to use a cold douche or a shower-bath every morning after a tepid bath. The cold bath by itself is probably less effectual for the particular end in view, although it is all that is required for robust persons with an active circulation, in whom it is followed by a good reaction. Even in young children a warm bath, especially in the morning, is best followed by rapid sponging with cold water.

Many *secondary* or *predisposing* causes of bronchitis are really conditions which favour the injurious action of cold. Thus the disease is very apt to attack young children on account of their feeble powers of resistance. A curious point that came out in some investigations made by Geigel as to the infant death-rate in Würzburg was that bronchitis was relatively less fatal to illegitimate than to legitimate children, the reason being in all probability that the latter are more coddled up and kept warm, so as to be rendered more sensitive to cold than if less care of them were taken.

The prevalence of bronchitis in Europe is, as might be expected, least in the hot season of the year, from June to September, and its proportionate frequency in different months is not the same as that of acute pneumonia. This is true also of the geographical distribution of the two affections. Bronchitis increases in frequency from the equator towards the poles, but the increase is not uniform in all longitudes, for it varies with the climatic conditions of each particular country. What favours it most is not a low mean temperature, but the occurrence of sudden and violent changes. In some parts of the tropics bronchitis is by no means uncommon at the end of the hot season. There are certain countries in which it is of very rare occurrence, particularly Egypt, the western prairies of North America, the plains of India, a part of the West Indies, and California.

Next to cold, the entrance of irritant substances into the air-passages during breathing is the most important cause of bronchitis. In discussing the ætiology of phthisis we shall have to consider the influence of various occupations, in which the inhalation of dust is almost inevitable, upon the production of that disease. Such occupations also cause a great liability to bronchitis, which may either in the course of time be followed by the development of phthisis, or may run a chronic course without complication until it ends fatally by the supervention of an acute attack, or by dilatation of the heart and dropsy. Pulverulent substances which happen to be coloured, such as carbon or oxide of iron, often tinge the sputum deeply when they have been inhaled. But, on the other hand, it may happen that a miner, whose lungs are black with coal-dust, spits up a yellow muco-purulent fluid, containing no carbon whatever. This accords with the fact that the bronchial mucous membrane never itself becomes the seat of anthracosis; even the peribronchial tracts of fibrous tissue derive the black

deposit which is found in them from the surrounding pulmonary alveoli, and not from the tubes themselves.

The inhalation of gases, especially nitrous or sulphurous acid, is exceedingly irritating to the air-passages, and not infrequently sets up acute bronchitis in workmen whose occupations expose them to it. But, according to Hirt, the chronic form of the disease is comparatively seldom traceable to this cause. After one or two acute attacks a tolerance seems to be established, and no further ill effects are observed. On the other hand, he speaks of the emanations from certain oil-works, from tar factories, and from the pans in which brine is evaporated to make salt, as having a beneficial influence on the bronchial mucous membrane.

The *treatment* required for bronchitis varies widely in different forms and in different stages of the disease, so that it is not easy to lay down general rules.

In the milder forms of acute *bronchial catarrh*—such as are called by some writers tracheo-bronchitis—little is necessary beyond placing the patient in an equal temperature, which should be at about 63°. Small doses of ipecacuanha, with neutral salts like nitre, are probably serviceable by favouring exudation from the inflamed mucous surface. The application of mustard plasters or of hot flannels sprinkled with turpentine to the throat and to the upper part of the chest often gives great relief to the sense of soreness along the trachea and behind the sternum. In slight cases a linseed poultice and inhaling steam are efficient and agreeable remedies.

Very different measures are necessary in cases of *capillary bronchitis*. Sometimes, if suffocation appears to be rapidly impending and the right side of the heart to be overloaded, it is advisable to bleed from the arm. Antimony is often the best medicine, and for a day or two it may be given in considerable doses, so as to produce decided nausea. Another drug which may be very successful is lobelia, the ethereal tincture of which is prescribed in half-drachm or even in drachm doses at frequent intervals. There are some patients, however, for whom all depressing remedies are obviously unsuitable. In these serious and often dangerous cases the administration of turpentine sometimes affords the best chance of arresting the fatal issue which is threatening. In one of the very worst cases which the author ever had to treat, life appeared to be saved by alternate doses of turpentine and of champagne. Ammonia in frequent doses is another most valuable drug.

It is important to maintain a moist state of the air round the patient. A kettle on the fire, with a long tube throwing steam out near the patient's bed, fulfils this indication better than anything else. But when it is used, one must never forget that the ceiling and the curtains necessarily become saturated with damp, and that if the temperature of the room should be allowed to fall a few degrees during the night, or in the early morning, a chill will result, which may probably be fatal to the patient. In some cases the inhalation of steam gives much relief, or a medicated spray may be employed, containing conium juice, or morphia, or salines such as chlorate of potass or ammonium chloride. Large poultices are commonly placed round the whole chest from front to back; and mustard or turpentine is applied until the surface is thoroughly reddened.

After a few days it is generally necessary to substitute for nauseant

drugs, like ipecacuanha or antimony, such remedies as carbonate of ammonia, squill, and senega.

In *chronic bronchitis* a great variety of medicines are useful; the difficulty is to formulate rules for selecting one rather than another. If the cough is dry and hard, ipecacuanha is most serviceable, and to this squills and nitre are often usefully added. If it is distressing by its frequency and apparent aimlessness, bromide of ammonium or of potassium often gives relief. In such circumstances morphia or opium may be taken with great advantage. The compound tincture of camphor is an excellent form to prescribe. After a few hours' sleep the patient may wake greatly refreshed, and in all respects better. It is, however, always necessary to consider, before prescribing opiates in a case of bronchitis, whether one is likely to do harm by checking cough, and so preventing the tubes being emptied of their contents. If there is lividity, stupor, or even drowsiness, such medicines must be carefully avoided. In many cases, especially if the expectoration is viscid and abundant, sal ammoniac is very useful; it may be given in doses of gr. xv to gr. xx, with a little syrup of lemon or extract of liquorice to conceal its disagreeable taste. Iodide of potassium is another salt which often does good service in chronic bronchitis.

In cases in which there is excessive exudation and secretion from the surface of the mucous membrane balsamic remedies are applicable—tolu, benzoin, Peruvian balsam, benzoic acid; or, again, one may prescribe copaiba, turpentine, ammoniacum, or one of the fœtid gum resins, such as assafœtida.

Of late years several physicians, in Germany especially, have made large use of compressed air, and also of rarefied air, in the treatment of various bronchial and pulmonary affections. In the earlier attempts recourse was had to pneumatic chambers, made somewhat after the fashion of the diving-bell, in which the patients sat for an hour at a time, under a pressure of $1\frac{3}{5}$ to $1\frac{3}{7}$ atmospheres. It is obviously in very exceptional circumstances only that such elaborate constructions can be available in practice; and therefore attention has been more recently devoted chiefly to the invention of portable forms of apparatus, by which the patient is made to inhale air of varying degrees of pressure without being himself immersed in it. Most of these instruments are upon the principle of the ordinary gasometer used to receive coal gas at gas-works; an air-containing cylinder, open below, is suspended in another cylinder, open above, so that the one can move freely up and down within the other. By pouring more or less of water into the outer cylinder, and then either pressing down the inner cylinder with weights or lifting it up to varying heights, the air inside it may be compressed; or it may be rarefied to any desired extent. The object is to make the patient *inspire* compressed air, or *expire* into rarefied air. A tube from the inner cylinder is connected with a mask, which can be fitted air-tight over the nose and the mouth. There is a stopcock, which is turned by the patient each time he breathes, so that the mask communicates with the cylinder either during inspiration or during expiration (as may be intended), whereas on reversing the movement it communicates with the external air. Waldenburg, who invented this machine, usually directs that compressed air should be inspired for five, ten, or fifteen minutes, and then, after a pause, that expiration into rarefied air should be practised for a similar period. The range of pressure variations employed is but small. In most cases two or three sittings a day are sufficient. It is obvious that, so far as emphysema is concerned,

the greatest degree of benefit is to be anticipated from expiration into rarefied air. The inspiration of compressed air, when the whole body is not immersed in a pneumatic chamber, might be expected to tend rather towards increasing the distension of the pulmonary alveoli. But, on the other hand, it is said that expiration into rarefied air may, in its turn, do harm by augmenting the flow of blood to the bronchial mucous membrane, whereas inspiration of compressed air has the effect of increasing the blood-pressure in the systemic vessels, and of unloading the pulmonary vessels and the right side of the heart. It is therefore best to alternate the two methods, as Waldenburg and others advise. There is strong testimony of the beneficial action of this mode of treatment, in augmenting (at least for a time) the activity of emphysematous lungs, and in relieving the symptoms of bronchial catarrh. (See Dr Theodore Williams' three lectures in the 'British Medical Journal,' April 18th, 1885; Dr Gamgee's paper, *ibid.*, December 18th, 1886; and the chapter in Dr Hale White's little book on non-medicinal therapeutics.)

In some cases of chronic bronchitis recourse may be had with advantage to certain Continental spas. According to Braun those waters are the best which contain a considerable amount of chloride of sodium, as well as of carbonate of soda, such as Ems in Germany and Mont Dore in France.

The diet of patients suffering from chronic bronchitis requires regulation. Rather free diluents are in most cases desirable, and probably the mineral waters just mentioned act chiefly in this way, promoting free and loose secretion from the bronchial mucous membrane. By common experience beer does harm, and even London labourers with a cough avoid porter, and drink gin instead. Cheese is also believed to be bad for a cough; and Pliny's dictum—*nuces tussientibus inimicæ*—still holds good, probably from the mechanical irritation of the fauces which they produce.

When it is thought desirable for a bronchitic patient to spend the winter and spring away from home, the choice lies usually between climates which are soft and "sedative" (such as Torquay, Falmouth, Penzance, Pau, Madeira), those which are stimulant without too much risk of exposure to cold winds (as Mentone and San Remo), and those which are surrounded by pine woods (as Arcachon and Bournemouth).

PLASTIC BRONCHITIS.*—This singular affection is certainly nothing else than bronchitis anatomically; but it has characters so peculiar that from a clinical point of view it would be absurd to group it with the ordinary catarrhal forms. It consists in the exudation of a fibrinous material from the walls of the air-passages which forms "casts" of their channels. A like exudation may occur by extension of a morbid process downwards from the larynx in diphtheria, and much less frequently by extension upwards from the pulmonary alveoli in pneumonia. But these are totally different in pathology and symptoms. The former has been described with membranous laryngitis in the chapter on Diphtheria. The latter will be again mentioned in that on Pneumonia.

The disease is one of the rarest that are known to physicians—"an affection of great rarity" (Walshe); "höchstselten" (Riegel); "aeusserstselten" (Biermer). The experience of Sir Thomas Watson was remarkable, in having had under his own observation five well-marked examples.

* *Synonyms.*—Fibrinous bronchitis—Croupous bronchitis or Bronchial croup of the German writers—Bronchial polypi of older authors—Angina polyposa.

These "bronchial polypi" were known very early. Dr Nicholas Tulp (the lecturer in Rembrandt's famous painting, "The Lesson of Anatomy") records and figures two specimens brought up by a Dutch sea-captain suffering from hæmoptysis: "*Effudit duos insignes venarum ramos, adæquantes singulos expansæ manûs magnitudinem*" ('Obs. Med.,' Amst. Elz., 1652, cap. xiii, p. 122, tab. iii, iv). Afterwards cases were recorded by the younger Bartholin, Cheselden, de Haen, Morgagni, Hunter,* Cheyne of Dublin, and Stokes. Most of the early cases (1690—1730) were published in the 'Philosophical Transactions.'

Anatomy.—In almost all cases of plastic bronchitis the patient soon begins to expectorate masses of the peculiar exudation. It usually appears rolled up into a sort of ball, with a good deal of mucus and blood covering it. All this is easily removed by floating it out in water, and one then sees that there is a complete cast of some part of the bronchial tree, extending perhaps down to its finest subdivisions, so that, according to Biermer, the minute terminal filaments may actually show bulbous ends moulded in the infundibula themselves. The colour of the cast is whitish yellow or grey; its consistence is tough and elastic; it is almost always made up of a number of concentric laminæ, separated here and there by narrow spaces and with a more or less definite central cavity, containing mucus or bubbles of air. The more delicate filaments are said to be generally solid.

The laminated structure affords a distinction from the branching clots which are sometimes formed in the air-passages as the result of hæmorrhage, and which are quite homogeneous. Biermer, indeed, is disposed to deny that blood ever coagulates so as to form casts of the bronchial tree. But Dr Walshe speaks of it in the most positive terms, and there could hardly be a better authority.

The casts in cases of fibrinous bronchitis, when examined microscopically, are seen to consist of a hyaline or slightly fibrillated base, in which are embedded large numbers of leucocytes. They seldom contain red blood-discs in any quantity. Several observers have noticed Charcot's crystals in them. In one case Waldenburg found that the thicker parts contained only a few formed elements, but very abundant fat-globules.

The length of a bronchial cast is commonly from one and a half to two and a half inches, but sometimes it may be four or five inches, or even (as in a case of Riegel's) six or seven inches. The diameter of the thickest part of it is seldom greater than that of a goose-quill, being in fact considerably less than that of the space in which it was formed. Biermer has pointed out that from its appearance one can sometimes draw an inference as to the part of the lung from which it came, whether from the short rapidly branching tubes of the upper lobe, or from the comparatively longer tubes of the lower lobe. The masses expectorated at different times by the same patient often resemble one another so exactly in size and in the arrangement of their subdivisions as clearly to show that they have all in succession been derived from the same tract of mucous membrane. For example, in a case recorded by Kretschy seven casts appeared one after the other, all of which came from the middle and lower lobes of the right lung. In fatal cases it is not usually found that the tubes which have poured out the fibrinous

* Hunter's case occurs as a short Appendix to his 'Treatise on the Blood, Inflammation, and Gunshot Wounds.' The patient was a man of twenty-two, who spat mucus often mixed with blood. He recovered. The figure agrees with those illustrating the cases of Fuller, Peacock, Salter, Tuckwell, and the author of the present work, in the 'Pathological Transactions' (vols. v, ix, xvi, and xxi).

exudation show any marked morbid changes. The mucous membrane is sometimes reddened, sometimes pale and healthy-looking. In two instances Biermer found the epithelium still remaining beneath loose casts; but he was himself disposed to think that these cases might be exceptional; and Kretschy has since stated that in his case there was no trace of epithelium in that part of the air-passages which contained the plastic material. The submucous tissue may be swollen and infiltrated with serum. The pulmonary alveoli are usually unaffected, but they have been found sometimes collapsed, sometimes over-distended.

Symptoms.—The expectoration of casts of the lower air-passages is generally attended with severe cough and dyspnœa, the occurrence of which may be the first indication that the patient is otherwise than well. But in many cases there is an antecedent stage during which he appears to be suffering from ordinary bronchial catarrh; and this may last for a long time. Sometimes the disease sets in with rigors, loss of appetite, thirst, oppression of the chest, and pyrexia, so that it may be supposed that an attack of pneumonia is impending. Presently a dry, hard cough appears, which may cause extreme suffering; the breathing becomes rapid, up to 40 or more in the minute; it may be attended with the greatest anguish, as of impending suffocation, with lividity, and with a small tense pulse. There may be some pain in the side, and a feeling of soreness within the chest, but on the whole the attack is more distressing than acutely painful. At first nothing is expectorated, or only a little mucus. The cough may even, it is said, go on for days before any fibrinous masses appear. More often a cast is detached and got rid of after a few hours, and by this the cough and dyspnœa are generally at once relieved, at least for a time. Hæmoptysis often occurs at intervals during the paroxysm, not only at the time when the cast is being expectorated, but also previously. The quantity of blood is not large; perhaps it amounts to a tablespoonful at a time. The case of the late Prof. Daniell, recorded by Watson, is exceptional in the fact that from two to eight ounces were spat on each occasion.

Physical signs.—Examination of the chest throws little light upon cases of plastic bronchitis. If a large tube is blocked, absence of vesicular murmur may be made out over some part of one of the lungs. The fact that the violent cough fails to clear away the obstacle might perhaps suggest to a keen observer the presence of something more than a plug of mucus such as may prevent the entrance of air in ordinary cases of catarrhal bronchitis, and the diagnosis as to the cause of the obstruction would then lie between fibrinous casts, a foreign body, and stenosis of the walls of the tube. In practice, however, it scarcely ever happens that any suspicion of the real nature of the case arises until a cast has actually been expectorated. There is not usually any change in the percussion-sound, but Dr Walshe says that he has had repeated occasion to observe dulness, as complete as that of pneumonic consolidation, dependent upon collapse of the lung-substance. He also says that local pneumonia now and then occurs, attended with crepitation and with bronchial breathing, as well as with rusty sputa. When there is extensive blocking of tubes the movements of the corresponding side of the chest may be distinctly impeded, and the lower ribs may even be drawn in during inspiration. Râles are sometimes audible over the affected part of the lung, especially when the cast is becoming loose; some writers have described special sounds as thus produced, but it does not appear that they are really characteristic.

Course.—The expectoration of a single cast very rarely brings an attack of plastic bronchitis to an end. As a rule the relief is only temporary. After some hours the cough and the dyspnœa return, and are followed by the appearance of another cast. This process is usually repeated about once in twenty-four or in forty-eight hours for several days, and then the affection slowly subsides. Smaller pieces may be spat up at very frequent intervals; being embedded in mucus, they sometimes remain unnoticed unless specially looked for.

Prognosis.—It may well be supposed that the expulsion of such large masses as sometimes come from the air-passages in this disease is not unattended with danger. In 1865 the author showed to the Pathological Society a cast which was taken from the body of a girl aged seven, having been found lying across the bifurcation of the trachea, with its branches extending into the ramification of the right bronchus, but with its broad end occluding the left bronchus. She had been expectorating similar masses for three days; and on the very day on which she died she had already, at 3 a.m., brought up a cast of about the same size as that which killed her at 3 p.m. in a violent fit of cough and dyspnœa. It is therefore clear that Dr Walshe and others go too far in giving a favourable prognosis in cases of plastic bronchitis without reservation. Lebert, in a paper in the 'Deutsches Archiv' for 1869, divides 44 cases (collected from various sources) into acute and chronic: of seventeen of the former, four ended fatally; of twenty-seven of the latter, only three. The distinction, however, appears to be rather artificial; and one of the four fatal acute cases, that recorded by Nonat, was, there is reason to suspect, a case of diphtheria. As a rule, when death occurs, it is caused by extension of the disease into so large a part of the bronchial tree that due aëration of the blood can no longer be effected, and is preceded by stupor and somnolence. Riegel, however, relates a case in which, although the patient died in an attack of asphyxia, after spitting up large casts nearly every day for three weeks, the air-passages were all found empty after death.

Lebert placed in a separate category cases in which plastic bronchitis has run on to a fatal termination without any fibrinous masses having been expectorated. As they occurred chiefly in children, and generally in association with broncho-pneumonia after measles, they should probably be regarded as different from plastic bronchitis.

Recurrence.—When an attack of fibrinous bronchitis has passed off, leaving the patient apparently well, it by no means follows that the disease is really at an end. One of the most curious points about it is its liability to return again and again at irregular intervals, sometimes during a very long period. Dr Walshe met with an instance in which the expectoration of casts continued, with occasional brief intermissions, from the spring of 1843 to June, 1857, when he lost sight of the patient. In the course of this time she married, and lived at Buenos Ayres for four months. Other observers have recorded cases which were scarcely less protracted; in many of them the general health seemed to remain unaffected.

Ætiology.—With regard to the causes of plastic bronchitis, scarcely anything can as yet be said. It is much more common in males than in females.

The period of life at which it is most frequent is between ten and thirty. One case, recorded at the advanced age of seventy-two, had lasted seven or eight years.

Adding to the 31 cases tabulated by the late Dr Peacock in the fifth volume of the 'Pathological Transactions' 24 additional ones collected from various sources, we find that of these 55 cases, 42 occurred in men and 13 in women. (Biermer's figures are 39 male to 19 female patients.) Of 37 patients whose ages are given, 5 were between five and ten, 12 between eleven and twenty, 10 between twenty-one and thirty, 8 between thirty-one and fifty, and 2 between fifty-one and sixty.

The writer has had only two typical cases under his care, one a boy of twelve, about 1870, who spat up repeated casts with some amount of blood, and recovered; the other a man of forty-three, who died of acute pneumonia (May, 1890) in the course of one of repeated attacks of bronchitis with expectoration of large fibrinous casts. A third case (published in the 'Pathological Transactions' for 1880, p. 30), in a woman of thirty-two, was fatal after tracheotomy, but here the trachea and primary bronchi were affected, so that a continuous fibrinous cast was brought up; and clinically as well as pathologically the case was peculiar.

A remarkable circumstance, all the more striking because of the extreme rarity of the disease, is its occurrence in different members of the same family. Fuller met with it in two sisters; Watson relates the cases of two brothers, both of whom were affected within a twelvemonth.

Plastic bronchitis is supposed to be rarer in southern countries than in the north of Europe. Riegel says that, like acute pneumonia, it is most apt to occur towards the end of spring, when there are great daily variations of temperature. In one instance the recurrence of the attacks appeared to be connected with the catamenial periods. Eisenlohr met with a case in which fibrinous casts were expectorated during the second week of enteric fever.

There seems no reason to suppose that this remarkable affection is at all related to phthisis.

Treatment seems generally to be altogether ineffectual. Waldenburg, however, saw a case in a girl, aged eight and a half, who for more than four years had been coughing up fibrinous masses at intervals of a few days, and in whom a whey cure and the daily inhalation of lime-water succeeded in arresting the disease in six or seven weeks. Indeed, a spray of lime-water, or a solution of an alkaline carbonate, should always be employed in plastic bronchitis; the only doubt is whether they reach the lower air-passages in sufficient quantity. Emetics appear to be sometimes useful: probably it is best to use apomorphia hypodermically. Biermer recommends an active mercurial treatment; others have prescribed iodide of potassium with apparent advantage.

Dr Walshe believes that neither inhalation of iodine, nor exhibition of alkaline medicines, nor the best of general health, nor the most favoured climates, have the least beneficial effect in preventing or curing the attacks of this paradoxical disorder.

PNEUMONIA

AND INFLAMMATION OF THE LUNGS

"Spiritus gravis est ac fervidus. Facies rubet, et in ea præsertim malæ. Quod in oculis album est humidissimum et pingue apparet: acumen nasi simum fit; venæ in temporibus et cervice elatæ sunt: cibi fastidium. Pulsus in initio mali magnus, vacuus, creberrimus, quasi compressus. Calor in exterioribus partibus obscurus et humidior; in interioribus autem aridus atque ferventissimus; ex quo spiritus incalescit, sitis fit, et linguæ siccitas, et frigidi aeris desiderium, et animi molestia. Accedit etiam tussis, quæ plerumque sicca est, vel si quidquam excreatur pituita prodit, aut squamans, aut bile saturata, vel cruenta et coloris floridissimi."—ARETÆUS.

PNEUMONIA.*—*History and definition—Anatomy and histology: stages, locality, events—Complications—Signs—Symptoms—Ætiology—Pathology—Prognosis—Mode of death—Convalescence—Origin and nature—Treatment.*

Secondary pneumonia—Hypostatic pneumonia—Acute pulmonary congestion—Chronic lobar pneumonia.

BRONCHO-PNEUMONIA, *or Pulmonary Catarrh.—Causes in children and in adults—Anatomy—Symptoms and course—Prognosis and treatment—Vesicular, caseous, and pyæmic pneumonia—Inflammatory œdema of the lungs.*

CHRONIC INTERSTITIAL PNEUMONIA, *or Cirrhosis of the Lung.—Anatomy—Origin—Symptoms.*

SYPHILITIC INFLAMMATION OF THE LUNGS.—*Cases—Diagnosis—Anatomy—Acquired and hereditary forms.*

GANGRENOUS INFLAMMATION OF THE LUNGS.—*Anatomy—Origin—Fœtor and other symptoms—Treatment.*

BY *pneumonia* (the peripneumony of older writers), which in Greek medicine denoted "disease of the lungs," is now understood an inflammation of the texture of the lungs, and when the term is used without qualification, one that is acute and primary.

It corresponds to the anatomical condition known as hepatisation and due to exudation into the alveoli.

With qualifications the word is applied much more widely, to any form of pulmonary inflammation. In the sense now employed, it excludes (1) that form of lobular inflammation of the lungs which arises by extension from the bronchial tubes, and has been called catarrhal or lobular or *broncho-pneumonia*; (2) the suppurative or *pyæmic lobular pneumonia* which occurs as

* *Synonyms.*—Peripneumony—Fibrinous, Plastic, or Croupous pneumonia—Acute or Sthenic pneumonia—Lobar pneumonia—Acute inflammation of the lungs.

Περιπνευμονία (or in the Attic form περιπλευμονία), from πνεύμων or πλεύμων (whence *pulmo*), the lung, occurs in Hippocrates and Plato. It did not originally carry with it the notion of an inflammatory or febrile disorder. The prefix peri- was only dropped in the present century.

The Germans have adopted the designation of "croupous," because the exudation mainly consists of a fibrinous material like that of croup. In fact, "croupous" means "fibrinous" or "plastic." To English ears, however, this has an awkward sound, and the phrase is on other grounds objectionable. It suggests some connection with the malady known as croup, whereas there is none; and it confounds a clinical term with an anatomical condition.

the result of infective embolism; (3) *traumatic pneumonia* from wounds of the lung, injuries of the chest, or the penetration of foreign bodies into the air-passages. (4) It is a question whether the congestive or *hypostatic pneumonia* which so often forms the immediate cause of death in persons suffering from almost any chronic or acute disease, particularly enteric fever, ought not to be placed under a separate head. (5) There is no question that the *caseous pneumonia* which destroys the lung in phthisis must be separated from other kinds of pulmonary inflammation. (6) Lastly, chronic fibrous *interstitial pneumonia* is a distinct process both clinically and pathologically, whether it occurs as a conservative element in phthisis, or by extension from the peribronchial connective tissue and the pleura, and whether tubercular, traumatic, or syphilitic in origin.

Each of these so-called forms or varieties of pneumonia requires carefully chosen adjectives to discriminate it, and it would be well if each had a distinctive and substantive name. But when the term pneumonia is used without a qualifying epithet, it is generally understood (except perhaps in the case of children) to refer to the classical peripneumony.

For the sake of distinction, we may define the pneumonia now to be discussed as *idiopathic* in its origin, *acute* in its course, *lobar* in its extent, *basal* in its usual distribution, and *fibrinous* in the character of its exudation.

It is true that lobular and pyæmic inflammations of the lung are often acute, that idiopathic is at best a negative phrase, that the disease often exceeds and sometimes falls short of occupying a complete lobe, and that it may affect the apex of the lung; hence no adjective is quite satisfactory. Nevertheless we shall see that in its natural history and its clinical features, as well as in its anatomy, pneumonia in the restricted sense of the term is one of the most peculiar and distinctive of diseases.

Morbid anatomy.—The pneumonic process consists of a series of changes by which the spongy pulmonary tissue is rapidly converted into a solid mass. Systematic writers describe several distinct stages.

The first is the stage of "active congestion" or *engorgement*. The affected part of the lung is massive, heavy, and dark red in colour. It pits under the pressure of the finger, and a reddish frothy serum oozes from its cut surface; if the pressure is increased, its substance breaks down. Microscopically, the most obvious appearance is the dilated and tortuous state of the capillaries of the alveolar walls; minute punctiform hæmorrhages are also to be seen in the connective tissue between the lobules and beneath the pleura.

Next comes the stage of *red hepatisation*, so called because the texture of the lung is solid, like liver. It now sinks in water, it does not crepitate when pressed between the finger and the thumb, it is easily broken, and little or no fluid can be squeezed from it. Its cut surface has a dull, lustreless appearance, and is distinctly granular. The granules are composed of a solid inflammatory exudation, which completely fills up both alveoli and infundibula. Rindfleisch gives drawings of masses of it obtained by scraping the cut surface; they form complete casts of the interior of the spaces in which they were moulded. The red colour of the lung at this period is probably due partly to the large quantity of blood in its capillaries, partly to the fact that great numbers of red discs are extravasated and mingled with the coagulated fibrin and exuded leucocytes.

The third stage is that of *grey hepatisation*. This is characterised not only by a change in the colour of the affected parts of the lung-substance,

which now passes through reddish grey to grey or whitish yellow, but also by the diseased tissue becoming even softer than before, by its being less markedly granular on section, and by its beginning to exude on pressure a turbid fluid, more or less opaque, white, and puriform. The extreme forms of grey hepatisation are, in fact, described by some pathologists as constituting a fourth stage, which they term *purulent infiltration*. Histologically there is a wide difference between the characters of "red" and those of "grey hepatisation." In the grey stage no fibrinous coagula are visible; the substance which fills the alveoli now appears to consist merely of a mass of crowded leucocytes. The extravasated red discs are no longer to be seen. Rindfleisch speaks of them as becoming decolourised; perhaps we may suppose that they are absorbed by the rapidly multiplying leucocytes. To account for the change of colour it must also be assumed that the increased amount of exudation compresses the pulmonary capillaries and drives the blood out of them. But Rindfleisch remarks that it would be a great mistake to imagine that this occurs during life to the same extent as after death. He finds that in the dead body it is always easy to inject the vessels, and he therefore concludes that the heart, so long as it is beating, must be able to keep up a more or less active circulation through them. In other words, it is probable that the grey colour is, strictly speaking, a cadaveric change dependent on failure of the circulation after death.

The most important histological distinction between the two kinds of hepatisation is afforded by the state of the alveolar walls. In the "red" stage they are unaltered, except that their capillaries are distended; in the "grey" stage they are infiltrated with leucocytes, which fill up every interstice.

Distribution.—Pneumonia attacks parts of the lungs only, never the whole at once. It almost always begins at some one spot, from which it rapidly spreads.

All observers are agreed that the *right* lung is more often the seat of pneumonia than the left, the proportion being about as five to three. Sometimes both lungs are attacked together or, more often, in succession. On either side the *lower lobe* is affected far more frequently than any other part; Jürgensen says that it escapes altogether only in one case out of four, and probably this is above the mark. As a rule hepatisation begins at the extreme base, and extends gradually upwards from day to day; but sometimes it spreads downwards from the summit of the lower lobe, or upwards and downwards from its middle, or backwards from the anterior border. In the upper lobe it may either pass from the apex downwards, or from below upwards and forwards. Sometimes its distribution remains strictly limited by the lobar septa; sometimes its spreading edge forms a horizontal line, ignoring them altogether. The most common seat of pneumonia is the right base, next the left base, next both bases. Even at the apex left pneumonia is more rare than right. Henoch found right apex pneumonia in 21, and left apex pneumonia in only 4 cases out of 74 of lobar pneumonia in children. Dr Goodhart's numbers are 18 right and 17 left apex pneumonia out of 120 cases of lobar pneumonia in children.

Events.—The results of pneumonia are unlike those of ordinary inflammation. Either death occurs, or the whole affected tissue clears up and returns to its former condition. The formation of an *abscess* in the lung, as the result of true pneumonia, is admitted by all writers to be very rare, and

many, including the present writer, doubt whether it ever occurs. Cases have, indeed, been recorded; but the question is whether a more accurate pathology might not have led to a different interpretation. They may have been circumscribed empyema, or suppurating bronchial sacs or hydatids; or true pulmonary abscesses, but not of pneumonic origin—traumatic, pyæmic, or gangrenous. The termination of pneumonia in *gangrene*, though also very infrequent, undoubtedly occurs under special circumstances to be afterwards discussed (*infra*, p. 1014).

Pneumonia often ends by *resolution*, and the question then presents itself whether it is possible for the disease to pass through all its three stages above described and yet for the lung to recover. It is certain that many cases end favourably after there has been clear evidence from physical signs that the pulmonary tissue has become "consolidated," *i. e.* has reached the stage of red hepatisation; the only doubt is whether the supervention of the third stage of grey hepatisation is compatible with recovery. In their work on Pathological Anatomy, Wilks and Moxon express a decided opinion that in most cases the disease has not advanced far beyond the "red" stage when resolution begins.

There are obviously two ways in which the pulmonary alveoli may be emptied of the exudation that fills them: one is by its escaping into the air-passages and being expectorated; the other by its being reabsorbed into the blood. Rindfleisch maintains that the greater portion takes the former course; but most clinical physicians will agree with Jürgensen (in the article on pneumonia in 'Ziemssen's Handbuch') that he is mistaken, inasmuch as in many cases sputum is altogether wanting.

After the subsidence of pneumonia, if the patient should die at no long interval from some other disease, the lung is found to have nearly regained its healthy appearance, but to be slightly redder and tougher than natural. Two such cases at least have been observed at Guy's Hospital, but unfortunately no microscopical investigations were made as to the exact state of the pulmonary tissue. Rindfleisch speaks of a loss of elasticity as resulting from pneumonia, and as continuing a long time after recovery.

It is a very important question whether, instead of subsiding, true pneumonia ever leads to permanent changes in the lung, to the development of fibrous tissue, constituting what is termed cirrhosis or chronic pneumonia, or to a destructive process, ending in the formation of cavities more or less like those of phthisis. As regards cirrhosis, though some observers (including Wilks) are opposed to the belief that it ever arises in this way, cases will hereafter be cited which seem to point to its occurrence. It is, at all events, extremely rare. As for the supposed termination of pneumonia in phthisis, there is every reason to believe that the cases that have been so interpreted were phthisical from the beginning.

Concomitant post-mortem lesions.—Pleurisy of the dry kind is a constant part of the disease. Wherever the hepatisation reaches the surface of the lung, the corresponding part of the pleura always becomes covered with lymph. Accordingly some physicians speak of the disease as "pleuro-pneumonia," at least in those cases in which they discover physical evidence of pleurisy during life. But this seems to be an unnecessary refinement, and indeed to be rather misleading, since after death the dry exudation of pleurisy is found, even when there had been no signs of its presence during life. It is better to reserve the term pleuro-pneumonia for cases in which the pleurisy leads to fluid effusion, and so becomes of clinical importance.

Occasionally there is likewise *pericarditis*, which has probably arisen by extension from the pleura. Still more rarely the peritoneum also, especially the upper part of it, is found coated with lymph. Sometimes the mediastinal tissues are extensively infiltrated with a gelatinous exudation.

The bronchial lymph-glands are constantly found enlarged, pinkish grey, and soft. The subpleural lymphatics corresponding with the seat of the pneumonia may be not infrequently observed to be distended with inflammatory products, so as to give a marbled appearance to the surface.

As a very rare complication of pneumonia must be mentioned acute meningitis. Four cases have been already narrated (p. 649); two of these were cerebro-spinal meningitis; one was complicated by diphtheria, one by chronic Bright's disease, and one by the possibility of a traumatic or pyæmic origin.

Less infrequent is ulceration of the larynx; the ulcers are found over the arytænoid cartilages, just where they occur from many other causes.

Probably it is to the pyrexia which accompanies pneumonia that one should refer some other slight but almost constant morbid changes that are met with after death—a moderate degree of enlargement with softening of the spleen, slight catarrh of the intestine, and cloudy swelling of the kidneys.

Signs.—It is by physical examination alone that the seat and extent of pneumonia can from day to day be determined with accuracy, although the other symptoms commonly enable one to form a confident opinion of the nature of the disease.

The earliest signs are generally recognised by auscultation. According to Stokes, there is first audible a peculiar harshness of the vesicular murmur. But more often the first sign detected is an entirely new or adventitious sound which is called *pneumonic crepitation*, or sometimes "fine crepitation."* This is exactly like the sound produced by rubbing a lock of hair between the fingers close to one's ear, a comparison suggested by Dr C. J. B. Williams.† By Laennec, who discovered it, it was supposed to be pathognomonic of pneumonia. But sometimes it is not heard in undoubted cases of this disease, and it is now known to occur occasionally from œdema of the lungs. A sound very like it, if not absolutely the same, may often be heard with successive inspirations in the bases of the lungs of a person who has been lying on the back with some febrile disease, if he is made to sit up and breathe deeply, so as to fill those parts which have been for some time disused. In the last case there can be no doubt that the cause of the sound is the opening up of portions of the tissue that had become collapsed; and almost all observers are now agreed that in pneumonia it has a similar origin, being due to the inspiratory separation of the walls of alveoli and bronchioles which, being swollen, had come into contact in expiration. Crepitation is, indeed, heard only during inspiration, and sometimes only just at the end of deep inspiration, as after coughing. Its mechanism is therefore not that of other râles, whether crepitant or non-consonating; it

* *Fr.* Râle crépitant.—*Germ.* Das knisternde Rasseln.

† It is much "smaller" than any other râle, and would not be recognised as due to bursting of bubbles, like gurgling or mucous rattles. It is consonating, musical in character, and high-pitched in note. Laennec calls it "une espèce de crépitation ou de râle léger, dont le bruit peut être comparé à celui du sel que l'on fait décrépiter en le chauffant dans une bassine."

does not depend on the bursting of bubbles, and should always be distinguished from true "moist sounds."

One must search carefully for this important sign before concluding that it is not present. But, on the other hand, the fact must be admitted that in many cases of pneumonia this sign is not at any time discovered. Whether it is ever really altogether absent in such cases is perhaps doubtful, because it is in its nature transitory, and so may have passed off before an examination of the chest is made. Sometimes, however, it remains audible during almost the whole of the disease, not, indeed, at the same spot, for where there is complete consolidation it almost necessarily disappears; but in one spot after another, as they are successively attacked by the inflammatory process.

As the state of engorgement passes on to that of red hepatisation, there are developed other signs—dulness on percussion, bronchial breathing, and bronchophony with increased tactile vibration.

The degree of *dulness* varies much in different cases. It is not so absolute as in cases of fluid effusion into the pleura; nor is the sense of resistance to the finger so great. In exceptional cases the percussion-sound undergoes very curious modifications, the explanation of which is by no means obvious. Thus a cracked-pot sound is sometimes elicited; this is attributed by Dr Gee to the presence of "islets" of unsolidified lung embedded in the substance of the hepatised tissue. In other rare cases the sound has a more or less marked tympanitic quality. Sometimes the spot where this tympanitic sound is heard is not directly over the consolidated part of the lung, but in its neighbourhood, over pulmonary tissue which may be supposed to be relaxed as the result of the increased bulk of the consolidated part. Thus hepatisation of the upper lobe of the lung behind may cause a tympanitic percussion-note beneath the clavicle on the same side.

The *bronchial breathing* that accompanies the second stage of pneumonia may exhibit every variety of quality up to the amphoric. Usually it is a typical in-and-out, whiffing, tubular sound, equal with expiration and inspiration, and in quality not unlike a to-and-fro bellows murmur of the heart. Occasionally no such sounds are audible, and the explanation doubtless is that the tubes passing to the consolidated part happen to be filled with fibrinous plugs.

Bronchophony generally goes with bronchial breathing, and presents like differences of degree. Dr Gee remarks that in infants a bronchophonic cry is often the only auscultatory sign that can be obtained.

Increase of vocal fremitus, although sometimes well marked, is by no means constant. Probably its occurrence depends upon the state of the tubes leading to the consolidated part. If they are full of fluid secretion, the transmission downwards of the vocal vibrations is interrupted. It is only when the left side is the one on which the fremitus is greater that this sign can be regarded as of high clinical importance, because on the right side it is very often greater in healthy persons.

Lastly, when pneumonia affects only the central part of the lung, or reaches no part of the surface except that which is in contact with the diaphragm, physical signs are altogether absent. What is more common is for the disease to begin deeply in the interior, but to become superficial a few days later, so that all doubt as to the nature of the case is soon at an end.

During the stage of *resolution* the peculiar auscultatory signs of hepatisa-

tion disappear, and they are commonly replaced by râles, which may vary widely in character. Sometimes a crepitation is now heard, which may be scarcely less fine than that of the early period of the disease; this is called *crepitatio redux*. In other cases the sounds are so large, and at the same time so highly consonating in quality, that one might imagine the patient to be at an advanced period of phthisis, with the lung breaking up in all directions. Yet after a few days these sounds in their turn cease to be audible. A considerable time generally passes before the vesicular murmur becomes as loud and as distinct as it normally should be. The percussion-resonance also may long remain deficient, even when there has been no pleuritic effusion.

Clinical course.—An attack of pneumonia commonly sets in suddenly with a well-marked rigor. This occurred in 241 out of 280 cases, and again in 782 out of 975 cases ('Collective Investigation Record'). Sometimes, however, there is not more than a sensation of chilliness, which may almost escape notice. Sometimes, particularly in children, the disease is ushered in by a convulsive seizure, or by vomiting. As a rare exception when the pneumonia is idiopathic, but most frequently when it supervenes as a secondary complication of other diseases, its onset is insidious, and there are no symptoms to mark the exact time at which it begins.

Pyrexia develops very rapidly. Jürgensen cites a number of observations which show that the temperature may reach 104°, or even a higher point, within three or four hours from the shivering. At the same time the pulse is quickened, and becomes full and bounding in character. There are the usual complaints of malaise, headache, pains in the limbs, and anorexia, as in every other febrile disease. As the rigor passes off the cheeks acquire a crimson flush. A point on which Addison used to lay great stress is that, as tested by the hand, there is in pneumonia a pungent dry heat of skin hardly observed in any other affection. We saw how this has since been confirmed by the observations of Schülein (p. 37); and it agrees with the description of Aretæus: *calor aridus atque ferventissimus.*

So far there is nothing in the symptoms to show that the thoracic organs are the seat of the patient's illness. Before auscultation was practised it was not uncommon for a case of pneumonia to be called "typhus" or "continued fever," and for the autopsy to show for the first time the real nature of the case. Or if, as sometimes happens, there was violent delirium from the very commencement, and the patient became comatose and died in twenty-four or thirty-six hours, the disease was set down as "meningitis" or "encephalitis." But even then a careful observer seldom failed to notice indications that led him to form a correct judgment.

One point of great importance, on which Walshe especially insisted, is a change in the ratio of the pulse to the respiration. Healthy persons breathe once for every four or four and a half pulsations of the heart. In febrile diseases generally, both pulse and respiration are more frequent; but the relation between them is not much, if at all, disturbed. In pneumonia the increased frequency of the respiration is out of proportion to that of the pulse, the ratio being as one to three, or one to two, or higher still; for the patient may breathe sixty or eighty times a minute. Walshe has observed the number of respirations in the minute actually greater than that of the heart-beats. Jürgensen says that the cases in which this occurs are those of old people with slowly-acting hearts and atheromatous vessels. It is a curious fact, as Walshe remarks, that the amount of subjective

distress produced by such rapidity of breathing varies extremely in different cases; some patients, breathing thirty, forty, or even sixty times a minute, are wholly unconscious of any dyspnœa.

Another indication that should draw attention to the respiratory organs as the probable seat of the disease is, that the nostrils work during inspiration, and that the sterno-mastoidei and trapezii are tense and prominent. It may also be observed that the flushed cheeks and the lips have a slightly purplish tint. At present every educated practitioner, after noticing the aspect, and ascertaining the state of the pulse, respiration, and temperature of the patient, examines in every case, as a matter of routine, at least the apices of the lungs in front and their bases behind. Even with careful scrutiny, however, there is sometimes complete absence of the physical signs above described for the first twenty-four or forty-eight hours, a fact which is important to remember in forming a diagnosis. In marked cases the flushed face, erect posture, anxious look, bright eye, and rapid breathing, with the herpes of the lips, give a *facies* which is very characteristic.

Moreover, there are often symptoms which point clearly to the true nature of the illness, and show which side of the chest is affected.

One such symptom is *pain*. This is commonly situated near the nipple or towards the axilla, and may be the first symptom which the patient notices. This pain is identical with the *point de côté* of pleurisy; and in all probability, in cases of pneumonia, it is really due to the coincident pleurisy, as Addison long ago taught. Sometimes the pain does not appear until the patient has been ill some hours, or not until the second day, and it may be entirely absent.* It is exceedingly severe, and being increased by every movement, it leads the patient to endeavour to fix the ribs by pressure with his hand, and also causes him to curve his spine towards that side, so as to bring the ribs as close to one another as possible. Sometimes the act of drinking produces so much suffering that the patient will endure the great thirst produced by the disease rather than attempt to swallow.

The *cough*, which is another early symptom, is also modified by the pain, which interrupts and as far as possible cuts it short. The distress caused by the cough is greatly increased by the fact that the *expectoration* in pneumonia, though very scanty, is singularly tenacious and viscid, so that it is got rid of with extreme difficulty; even when it has reached the lips it often clings to them, and can only be removed by the handkerchief; and it adheres equally firmly to the spittoon, which can be inverted without any of it escaping. Pneumonic sputum has also a peculiar colour, due to the circumstance that the frothy mucus of which it consists is intimately mixed with blood that has perhaps undergone slight chemical changes since it left the vessels. This colour varies in shade in different cases. It is usually bright orange, tawny, or like the rust of iron, so that it is commonly called "rusty" expectoration. But sometimes the tint is a paler apple-yellow, and sometimes it is the bright scarlet tint of unaltered blood. Spitting of pure blood in such quantity as to deserve the name of hæmoptysis is not common; but it may occur in cases uncomplicated by tubercle, purpura, or Bright's disease, and the result may be as good and speedy a recovery as usual.

In some cases the sputum is thin, watery, and of a brownish-red colour, so that it is compared with prune-juice or liquorice-water. Such sputum is seen chiefly at advanced stages of the disease, and when it is taking an

* So Celsus: "*Id genus morbi* (*quod περιπνευμονικὸν Græci vocant*) *plus periculi quam doloris habet*" (iv, 7).

unfavourable course; but it may be present at an early period, and in cases that ultimately do well. Possibly, as Dr Wilson Fox suggested, it comes from a part of the lung affected with œdema.

Some patients, usually children, have no cough, and therefore no expectoration, throughout the whole course of pneumonia.

Remak showed, many years ago, that in pneumonic sputum there can sometimes be detected with the microscope branching fibrinous casts of the smallest bronchial tubes. Micrococci are often present (p. 997).

Having thus developed itself, pneumonia commonly runs on for some days with but little change. As more of the pulmonary tissue undergoes hepatisation, the physical signs become more marked; but there is often no corresponding increase in the severity of the symptoms.

The type of the *pyrexia* is, as a rule, continuous, with the usual diurnal oscillations. Wunderlich, however, states that it is not uncommon for an irregular and sudden elevation of temperature to occur, which is followed by a no less sudden fall through as many as 7° or even 9° F. to the normal point or below it, and that again in a few hours by a fresh elevation. We have observed more that one case at Guy's Hospital in which similar strange deviations in the regular course of the fever took place again and again, and, in fact, made up the greater part of the temperature chart, without any explanation being found.

The *pulse* may gain in frequency from day to day, but sometimes it remains for several days at 90 or 100, the only change being that it becomes gradually smaller and softer, or even dicrotic in character. In adults there is always ground for alarm if it rises to 120, but in children it may reach 130 to 140 without danger; in old people it is commonly much slower.

The *tongue* is thickly furred in pneumonia; as the disease advances it often becomes dry, brown, and covered with sordes. The bowels are generally constipated, but sometimes there is diarrhœa.

A symptom which usually makes its appearance between the second and the fifth day is an eruption of *herpes* upon the lips or face—a crop of clear painless vesicles occurring in a group on a red patch of skin. It is said to occur in from two fifths to one half of all cases of pneumonia, and to be far more common in cases that do well than in those that end fatally. This has long been a traditional opinion, and it seems to have been established by the observations of Geissler, who found ('Arch. d. Heilkunde,' 1861) that out of 431 cases the mortality among those in which there was no herpes was 29·3 per cent., whereas among those with herpes it was only 9·3 per cent.

After the first few days the *skin* in many cases becomes moist, and there is occasionally profuse sweating. An icteric tinge of the conjunctivæ is not uncommonly present, and sometimes there is well-marked *jaundice* of the skin. Formerly it was taught that this occurred only when the base of the right lung was the seat of the disease, and that it was due to the extension of "the disease" through the diaphragm to the liver-substance—an unscientific conception. The truth is that icterus may appear when pneumonia affects the left lung or the upper lobe of the right. The jaundice would seem to be due either to venous congestion of the liver or to catarrh of the common bile-duct accidentally present as a complication.

The *urine* in pneumonia is scanty, of high specific gravity, high-coloured, and strongly acid, depositing lithates in abundance. The amount of *chlorides* excreted by the kidneys undergoes a great diminution; they may even be altogether absent. At one time this was thought to be of great diagnostic

importance. But the same thing occurs more or less in other febrile diseases, the explanation being apparently that the salts in question are retained in the body, for, as Cohnheim argues, the diminished supply of them in the food is insufficient to account for so great a difference. During convalescence they reappear, probably in increased quantity. Not infrequently the urine contains *albumen*. This probably happens in from a fourth to a third of the cases. It is not of ill prognosis, and never leads to Bright's disease.

The *nervous system* is, as a rule, less affected in pneumonia than in other maladies with equally high temperatures. Many patients retain their consciousness throughout the whole of the illness, or merely wander a little at night during their broken sleep. But in other cases *delirium* appears as the fever increases, and it may be a furious maniacal type. In persons who have been intemperate, pneumonia is often attended with symptoms exactly like those of delirium tremens, or, to put it in another way, that affection may be present as a complication. An inherited neurotic tendency seems also to lead to the occurrence of severe cerebral symptoms in pneumonia. In the case of a man whose last hours were passed in a paroxysm of raging madness the author was told that hardly one of the brothers and sisters of the patient had escaped some form of neurosis. Pneumonia of the apex is particularly apt to cause delirium, or in children convulsions.

Modes of death.—The symptoms which precede death are generally the same. The breathing becomes more and more rapid, but shallow and ineffectual, the patient being no longer conscious of the necessity for filling his lungs. He sinks down in bed away from the pillows on which he was propped; the face is pale and livid in hue, leaden or slate-coloured; the skin is covered with a cold sweat; the pulse becomes weak, irregular, and finally imperceptible. Occasionally death is sudden, from the heart failing during some effort, as when the patient raises himself up in bed. Lastly, it now and then happens that the occurrence of the crisis (to be described in the next paragraph) is followed by prostration and collapse, from which the patient never rallies.

Defervescence.—In cases of pneumonia that end in recovery, the subsidence of the pyrexia and of the other symptoms is usually abrupt, *by crisis* (p. 30). In about 65 per cent. of cases, in which the exact duration of the disease can be reckoned from an initial rigor, or convulsion, or vomiting, the crisis is found to occur between the fifth and the eighth days: next often on the sixth, or the seventh. In some cases it is earlier; in others it is delayed until the second week. It is rare for fever to continue longer than this; and when defervescence fails to occur by the fourteenth day there is ground for suspicion either that the case is not one of true pneumonia, or else that some complication is present.

The crisis usually begins in the evening or night, scarcely ever in the forenoon. The fall of the temperature is as a rule rapid, being completed in about sixteen hours, and sometimes in five or six; but in some cases twenty-four hours pass before the thermometer reaches 98·5°. For the next day or two it often stands below normal.

Less frequently the defervescence in pneumonia takes place gradually, *by lysis*, and occupies two or three days.

In cases which end by crisis, it is surprising how much better the patient feels as soon as the pyrexia has subsided. His appetite quickly returns, he sleeps well, and his skin perspires comfortably. Both the pulse and the

respiration decline step by step with the temperature. Yet the physical signs may at first fail to indicate any improvement in the state of the affected lung, and several days may pass before the consolidation clears up.

There are no *sequelæ* of pneumonia. The patient may be allowed to eat as his appetite returns, and to go abroad when he feels able to. There is no danger of phthisis or other chronic affections following. In this respect it resembles typhus, and differs widely from enterica, as also from pleurisy and from broncho-pneumonia.

Recurrence.—A person who has once suffered from pneumonia is afterwards more likely to be attacked by the disease. Instances have been recorded in which it has recurred eight, ten, and even more times. Sometimes the same part of the lung is affected on successive occasions, sometimes not. Relapses are not frequent, if by this term we understand the repetition of the morbid process before the patient has completely recovered. But the spread of the disease to the other lung not infrequently comes on in the form of a relapse.

Ætiology.—Pneumonia is met with at all ages, though rare in children under three years old; and in both sexes, though commoner in men than in women. It is met with in all climates and in every race of the human family. Moreover, it is common among brutes. Pleuro-pneumonia is a frequent cause of death in monkeys, horses, and among cattle; it frequently assumes an epizoötic form, and decimates both oxen and sheep.

Different opinions have been expressed as to whether pneumonia is more apt to occur in persons who are strong and healthy, or in those who are weak and delicate. There is no difficulty in finding instances in support of either view, and one cannot help being profoundly impressed by the cases that one now and then sees of vigorous young men carried suddenly off by this disease. Yet these are, after all, exceptions. "Acute pneumonia occurring in a previously young and healthy subject" is more often described than seen, though if we except specific fevers it is probably more often seen as a primary invasion than any other acute inflammation.

If we take the cases of hepatisation of the lung as they occur in the deadhouse, the majority are found to be secondary to cardiac or renal disease, to enteric or other specific fevers, or to wasting maladies like cancer, diabetes, and paraplegia. On the other hand, it is not frequent for acute lobar pneumonia to be associated with phthisis, with bronchitis, or with primary pleurisy.

Even if these cases be excluded, and we examine the records of apparently primary idiopathic pneumonia, we find, as Dr Wilks has long taught, that in many cases the patient is old and feeble, or half starved, or habitually intemperate.

Occasionally pneumonia assumes an *epidemic* character among men, as among cattle. This is seldom seen now, but there is reason to believe that, like measles, syphilis, influenza, and perhaps the sweating sickness, it was once a more frequent type, and that its present sporadic occurrence has not been, and possibly may not be, always the rule.

In Dr Sturges' interesting monograph there are accounts of epidemic and very fatal pneumonia in Flanders during 1557, in the garrison of Philipsbourg in 1688, and in Iceland in 1863. He also refers to an epidemic in the 22nd Regiment, when stationed in New Brunswick, recorded by Dr Walshe in the 'Army Medical Reports' for 1867, and to one in the Mediterranean Fleet in 1860, described by Dr Bryson in the 'Lancet' (Jan.

9th, 1864). Jürgensen recorded the prevalence of epidemic pneumonia from 1873 to 1881 in the village of Lustnau, near Tübingen, and Dr Ballard reported to the Local Government Board an outbreak of apparently infectious pneumonia at Middlesbrough in 1888.*

Cases have been reported of *infectious* pneumonia. One striking instance of five adult brothers and sisters, living in the same house and successively carried off by the disease, was described by Dr Patchett in the 'Lancet' for 1882. It had occurred in 1876.

The principal exciting cause of pneumonia—as of pleurisy and bronchitis—is commonly supposed to be "catching cold." There is no physician who cannot recall to his memory instances in which patients were attacked immediately after getting wet through, or after lying on damp grass, or after some other very definite exposure to cold. But writers who have tabulated their cases, with the object of determining the frequency of the operation of cold as a cause of the disease, have failed to trace it in any but a comparatively small proportion. Among 205 cases collected by Grisolle, 45 were supposed to be due to cold; but among 186 cases of Ziemssen's only 10, and among 212 cases of Griesinger's only 4, could be clearly attributed to this cause.†

Again, in the relations of pneumonia and of bronchitis respectively to different climates and to different seasons of the year, there are differences which oppose the view that the two diseases have the same ætiology. Pneumonia does not, like bronchitis, increase in frequency with the latitude from the equator towards the poles. And with respect to the various seasons of the year, observations made at Vienna have shown that whereas in that city the prevalence of bronchitis reaches its maximum in March, and then gradually declines through the rest of the spring and summer, the prevalence of pneumonia increases steadily from February to May, after which it falls rapidly. This last part of the statement must not be taken as applying to other places besides Vienna, still less to Europe generally; for Jürgensen finds that there is a broad difference between continental and insular climates as regards the months in which pneumonia is most apt to occur; in the former it is between March and May, in the latter between December and February. But if the year be divided into two halves, the one from December to May, the other from June to November, then it is found that, throughout Europe, two thirds of the cases of pneumonia fall into the first half, one third into the second.

Another point which confirms the opinion that pneumonia is not directly due to cold is that sailors and others whose occupations expose them to bad weather do not seem especially prone to the disease.

Again, true pneumonia is not set up by local *injuries* to the chest, wounds of the lungs, foreign bodies in the bronchi, or any like causes. Traumatic inflammation of the pulmonary tissue is chronic and interstitial, or may be acute and caseous, but it is not hepatisation. It is impossible to induce the affection experimentally in animals.‡

In the pneumonic lung, as in so many other morbid structures, the

* See the bibliography by Dr Coupland in the Report on Acute Pneumonia ('Collective Investigation Record,' vol. ii, p. 10); also four instances, *ibid.*, vol. i, p. 104, and a full account of an epidemic of pneumonia in the Punjab by Surgeon-Major Maunsell, vol. ii, p. 77.

† These figures are taken from Jürgensen's article in 'Ziemssen's Handbuch.'

‡ Sommerbrodt stated that he saw it follow the injection of a solution of perchloride of iron into the air-passages; but Jürgensen repeated the experiment, and the result was negative.

discovery of microphytes has rewarded the diligence and skill of modern histologists. Friedländer first described in 1882 micrococci, usually occurring in pairs or chains, and surrounded by an envelope (*Diplococcus pneumoniæ*). They are frequently present in the hepatised lung, and have been found in the rusty sputum. Dr Giles found them in India ('Brit. Med. Journ.,' July 7th, 1883). But they may also occur in lobular and other kinds of inflammation of the lung. Moreover, other microphytes, spherical and rod-shaped, have been found in cases of pneumonia.

Pathology.—The traditional view is that pneumonia is an acute inflammation of the lung, and that the pyrexia and other symptoms are secondary to the local lesion. For many years, however, doubts have been expressed as to whether this is the true pathology of this remarkable disease; and it is possible to regard pneumonia as a general and specific disease to which the hepatised lung bears the same relation as the intestinal ulcers to enteric fever, or the angina to scarlatina. The following considerations bear upon this question, which is far from having only a speculative interest.

Are we right in assuming hepatisation to be an inflammatory process at all? We have seen that it cannot be caused by injury or irritants, and it is doubtful whether it is the direct result of exposure to cold. It does not lead to suppuration on the one hand or to fibroid induration on the other. The exudation is peculiar, and unlike that of undoubted inflammation which has extended from the bronchial tubes to the air-vesicles. The distribution is remarkable. The disease never or scarcely ever assumes a chronic course.

On the other hand, the inflammatory nature of the process seems witnessed to by the constantly concomitant pleurisy and occasional pericarditis; as also by the exudation of fibrin and leucocytes along with blood-discs. Moreover, we are familiar with other cases of non-traumatic inflammations which are more or less peculiar to the organs they affect, and which, as they do not originate from ordinary irritants, so fail to produce the ordinary results of inflammation. Such are the tubal nephritis of Bright's disease, the membranous laryngitis of diphtheria, and the synovitis of rheumatism.

If the characteristic lesion of pneumonia may be accepted as a special form of inflammation, it is not to the catarrhal inflammation of mucous, but to the plastic inflammation of serous membranes that it should be compared. The pulmonary alveoli are lined by endothelium with lymphatic stomata, not by columnar epithelium with a vascular mucosa; and this is in all likelihood developed from the mesoderm, like the pleuro-peritoneal endothelium, and not like that of the bronchial tubes, from an outgrowth of the hypoblast. The alveoli, not the "parenchyma of the lung," are the primary seat of the exudation of pneumonia, as Addison proved fifty years ago.

If the other symptoms of pneumonia are secondary to the pulmonary lesion, why is the temperature so much higher than in other inflammations, whether serous, mucous, or visceral—higher than in any other disease except specific fevers, tuberculosis, or pyæmia? Why is there albuminuria with cloudy swelling of the renal epithelium, which disappears with the pyrexia, and never leads to permanent local changes? Why are the chlorides so remarkably deficient in the urine? Why do the general symptoms sometimes precede evidence of the local lesion.*

* It has been asked why the general symptoms are so independent of the severity and extent of the local lesion. But its "severity" is probably the same, or nearly so, in every case, and its extent is certainly not without influence; double pneumonia is a more serious disease than single, and consolidation of three fourths of a lung than of half its lower lobe.

Again, the course is said to be too typical for that of a local inflammation. Why does crisis often occur at the end of a week, and why is convalescence so rapid and complete?

Pneumonia, though certainly not infectious in the ordinary sense of the term, sometimes occurs, as we have seen, in epidemics; yet it does not protect from future attacks.

Lastly, the presence of a microphyte, even if not yet ascertained to be constant and characteristic, suggests a specific origin and character.

In the first edition of the present work the author regarded as not disproved the common opinion that pneumonia is essentially a local inflammation, accompanied by symptomatic pyrexia. "The truth," he remarked, "seems to be that the question of the real pathology of pneumonia is involved in a much wider one, to which at present no positive answer can be given. We have seen, in the chapter on inflammation, that many modern observers believe that no form of spreading inflammation is due merely to the reaction of the organism against a local injury. If this be the case, some specific exciting cause, such as Jürgensen assumes for pneumonia, must be supposed to be present in a vast number of other inflammatory diseases. Probably many years will pass before the true relations of all of them are fully understood. In the meantime it would not be really advancing in the right direction to detach pneumonia from other thoracic affections, with some of which it may, after all, prove to be closely allied, or to force it into a close relation with the group of acute specific diseases as now understood, from which it certainly differs in many important particulars." It appears to the present writer that the evidence tends to show that pneumonia is neither a specific fever nor a mere local inflammation, but the special idiopathic disease of the lung, like acute nephritis and acute yellow atrophy of the liver. Much depends on the view taken of the pathology of inflammation generally, and on the validity of the distinction between acute pneumonia and other forms of inflammation of the lung.

Prognosis.—Uncomplicated pneumonia is far less dangerous than its severe symptoms seem to threaten. The average death-rate cannot be stated with accuracy; for, in the first place, it varies enormously, according to the number of primary cases in proportion to those which are secondary to some other disease; many of the latter are obviously hopeless from the first. Moreover, the danger of pneumonia is widely different at different ages—children as a rule recover from acute lobar pneumonia; and in young healthy adults, of temperate habits, the prognosis is almost as good; Jürgensen gives the death-rate in such cases as from 3 to 6 per cent. But to old people, *i. e.* to all above sixty—or to those who, though not old, are worn out by misery, dissipation, or drink*—the disease is exceedingly fatal. Nevertheless, from time to time one sees a patient recover even when the circumstances have appeared most adverse.

The most important considerations as to the prognosis in a case of pneumonia—apart from the age of the patient (which is almost as important as in typhus or in diabetes) and his habits—are the presence of renal disease and the presence of heart disease; either of these complications makes even a limited consolidation of a single lung in a young and temperate person of the gravest significance.

* Of 37 cases of pneumonia in persons reported as intemperate, 15 died, or about 2 in 5; of 228 reported "temperate," 42 died, or less than 1 in 5; and of 80 total abstainers, 9 died, or little over 1 in 10 ('Collective Investigation Record,' vol. i, p. 95). The third class would include a large proportion of children, and the first many elderly people.

Lastly, even in a young and healthy subject, danger is indicated if the local lesions are extensive, if the whole of one lung or if both lungs are affected. Experience has taught that it is never safe to speak confidently of the recovery of a patient with pneumonia, however favourable its course may be during the first few days. For what was single may soon become double pneumonia; a change for the worst is apt to occur suddenly; the pulse, of moderate frequency hitherto, runs up quickly, respiration becomes more frequent and shallow, the strength fails with terrible rapidity, and in a few hours the end may come. In all probability the cause of the fatal issue in such cases is the supervention of inflammatory œdema or "acute congestion" in parts of the lungs previously unaffected. Other cases go steadily downwards from the very commencement.

In children above infancy, primary lobar pneumonia is of good prognosis. But the less apparently severe cases which appear in rachitic children, or after measles and whooping-cough, and perhaps are really catarrhal pneumonia affecting a number of lobules close together, are more dangerous, particularly in children under three years old. Of 120 cases of lobar pneumonia in children Dr Goodhart lost 25.

Treatment.—Pneumonia has been the battle-field of therapeutics as well as of pathology, since it was described clinically by Laennec and anatomically by Rokitansky.

From 1820 to 1840 antiphlogistic treatment was vigorously applied to this, the typically sthenic inflammation in a young and healthy subject. In France especially *la saignée coup sur coup* was practised in the hope and belief of "jugulating" the disease. In England twenty ounces of blood and more were often abstracted from the arm, time after time, or the patient was bled in the recumbent posture, so as to obtain a larger flow before syncope occurred. Any failure was ascribed to want of early and bold venesection.*

Mercury, usually in the form of calomel, and combined with opium, was also given as a necessary adjunct to venesection, particularly in England. Antimony was often conjoined with mercurials, and in Italy it was given in large and nauseating doses as a specific remedy for pneumonia.

A great revulsion of practice occurred in this country in the fifth and sixth decades of the century against "antiphlogistic" and in favour of "corroborant" treatment of inflammations, and of acute diseases generally. Wine and brandy, often in "heroic" doses, were prescribed in pneumonia under the influence of the late Dr Todd.

Meantime, some physicians, among whom the late Dr Hughes Bennett, of Edinburgh, deserves the foremost place, had been observing the natural history of pneumonia, and had found that, at least in young and temperate patients, its danger had been much over-estimated, that it tended to recovery after a week's duration, and that neither bleeding, nor mercury, nor antimony nor alcohol was necessary in favourable cases, or could be depended on to save life in unfavourable ones. For many years past the treatment of pneumonia in England has been practically the same as that of typhus, enteric fever,

* In Germany Skoda, then at the head of the Viennese school, had learned to distrust large and systematic depletion, although he still bled in the earlier stages of pneumonia, and believed in the power of drugs to defibrinate the blood and cut short the disease. There was a remarkable contrast between the treatment of pneumonia in his wards, as witnessed by the writer in the year 1865, and in those of Bouillaud during the preceding winter, where repeated bleeding was still the rule. At the same time cases of pneumonia were being "cured" at the Homœopathic Hospital in Vienna by infinitesimal doses of useless drugs.

or scarlatina—expectant, not in the sense of doing nothing for the patient, but of putting him in the best circumstances for recovery when the malady has run its course, and watching meantime for any unfavourable symptom, so as to meet it when it appears.

Of late, however, it has been asserted by some German physicians that better results can be obtained by vigorous antipyretic treatment, like that which has been described in the chapter on enteric fever. At Basle cold baths have been used whenever the temperature rose a little above 102° Fahr. Jürgensen advises that 104° should be the point at which baths should be systematically employed; while for patients whose temperature ranges at from 101° to 103° he merely orders a tepid bath in the morning, so as to increase the normal remission during the early part of the day. He also administers quinine in doses of thirty grains at intervals of forty-eight hours. The theoretical basis of this practice is that the great danger of pneumonia is failure of the heart, as the result of the combined action of pyrexia and of disturbance of the pulmonary circulation. In proof of its efficacy Jürgensen adduces a tabulated statement of the fatal cases that have occurred in his practice, showing that scarcely any of his patients died except such as had some dangerous complication. He makes a point of giving wine as a stimulant to the heart before each cold bath; and when signs of cardiac failure appear, he gives alcohol freely, as well as camphor and musk.

Few English physicians, however, believe that pneumonia can be jugulated by cold baths any more than by bleeding, or that moderate pyrexia is so injurious that it must be persistently attacked.

Even if cold baths are harmless, there is reason to believe that antipyretic drugs are as liable to do mischief as the antiphlogistic treatment which they now supersede. A careful trial of antipyrrhin in a series of cases of acute pneumonia led the late Dr Botkin, of St Petersburg, to the candid avowal that it was doubtful if it ever did good, and certain that it often did harm.

The present writer has more than once employed venesection in what seemed a suitable case without preventing death, has used cold baths to check pyrexia without doing any harm, but with doubtful effect on the course of the disease, has given aconite from the beginning without in the least altering the rise of temperature or averting serious complications, and has seen quinine, antimony, wine, and brandy, all in turn prove useless to check the progress of the disease.

The non-specific and rationally expectant treatment of pneumonia, as generally carried on in hospitals and in private practice in London, is somewhat as follows.

On the first rigor and rise in temperature the patient is put to bed, kept undisturbed, and his strength husbanded. The worst cases are those in which the patient has kept up during the first day of illness, or when some unhappy street outcast has walked about with the disease upon him until brought exhausted to a hospital.

The room must be kept comfortably warm. The patient must not talk, and must not sit up in bed. A single calomel purge does no harm, and has the advantage of preventing the exhibition of physic afterwards, when the patient is less able to bear its effects.

The diet should consist of beef-tea, milk, and arrowroot or gruel, with tea, fruit, jelly or ice, if desired, and a free supply of water, effervescing

drinks, or any harmless form of diluent. A cup of tea is harmless and refreshing, and strong coffee is sometimes a useful stimulant. Nitre, citrate of potash, or acetate of ammonia are usually prescribed, and probably are of real service as diuretics, and by promoting the solution and excretion of inflammatory products towards the close of the disease; but they need not be insisted on if (even when suitably disguised) they are refused by a child.

When pleuritic pain is present it may often be relieved by poultices, a mustard plaster, or, according to some authorities, by a blister; but in really severe pain nothing gives so rapid and complete relief as a few leeches. In aged or feeble patients we must trust to hot applications and morphia injections.

When cough and expectoration are difficult, ipecacuanha with paregoric and squills is indicated. If there is continued want of sleep, ten grains of Dover's powder is probably the most efficient remedy, and mere symptomatic albuminuria does not counter-indicate it. If, however, there is any doubt as to the efficiency of the kidneys, it is safer to give chloral hydrate or hyoscyamine.

If the temperature rises over 104° in the case of an adult, cold sponging should be employed, and if this is ineffectual and delirium is present, Leiter's coils, or the wet pack or ice-bags under the armpits are probably safer than the cold bath. Nevertheless, if the heart is sound and the patient young, a bath may be used. Dr Lees has lately published some good results of treatment with ice-bags ('Lancet,' ii, 1889), and Dr Goodhart has recorded his own experience of the treatment of pneumonia by cold applications in the 'Guy's Hospital Reports' for the same year.

If cyanosis appears, with signs of dilatation of the right side of the heart —a small, frequent, and feeble pulse, epigastric pulsation, distended and pulsating jugular veins—the abstraction of six, eight, or even ten ounces of blood from the arm will relieve the pressing symptoms and perhaps do permanent good. Wet or dry cupping between the shoulders is generally less effectual.

In the case of children, writes Dr Eustace Smith, where there is great dyspnœa and threatened cardiac failure from over-distension of the right side of the heart, life may often be saved by taking one, two, or more ounces of blood from the arm, according to the age of the patient. "I can look back," he continues, "to some fatal cases which I now believe might have been saved had I had the courage to relieve the labouring heart by judicious removal of blood."

The greatest danger in pneumonia is not from the fever nor from the pulmonary obstruction—it is from failure of the heart. To meet this, digitalis may be given with advantage, but it is less effectual than in cases of valvular disease. Ammonia, with or without senega, ether, and Hoffmann's anodyne (Sp. Ætheris co.) are all valuable drugs. But the most important means of meeting either an obstructed pulmonary circulation or a directly failing left ventricle is to use brandy frequently and in measured doses, but increased if necessary up to twelve, fifteen, or twenty ounces in the day. Children and young adults seldom need it, but there are few patients above forty, and probably none above fifty, who do not need stimulants in larger or smaller quantities from very early in the disease. Sometimes port wine or champagne suits better than brandy; the kind of stimulant may be decided by the patient's feelings, but the quantity and

frequency must depend on the state of the pulse and the first sound of the heart.

Secondary forms of pneumonia.—We have seen how the course of pneumonia differs according to the age and habits of the patient, and according as the heart and the kidneys are healthy or the reverse. Another important practical distinction is between primary pneumonia, with or without complications, and pneumonia secondary to other diseases.

In *rheumatic fever* pneumonia is not common, but when it occurs it is a very grave complication.

In the course of *Bright's disease*, particularly in the parenchymatous and lardaceous forms, pneumonia is almost always fatal.

In *disease of the heart* it commonly attends pulmonary hæmorrhage (or "apoplexy," as it has been called), a condition in which the air-vesicles are filled with blood, and a patch of lung becomes solid, dark red on section, and like damson-cheese in appearance. Hepatisation often takes place around the wedge-shaped mass of solid lung, but it does not spread far, and the symptoms are not so severe as those of primary pneumonia.

In typhus, enterica, and other *fevers* ordinary lobar pneumonia may occur (pp. 112, 129, 148); but far more frequently a condition of the lungs is recognised during life, and verified after death, which has been called "typhoid pneumonia," or, more properly, "*hypostatic pneumonia with congestion.*" It is found occupying not the bases, but the backs of the lungs—the lowest part as the patient lies in bed; it almost always affects both lungs more or less; it is not continuous, but consists of patches of airless tissue, including several lobules, and surrounded by congested and œdematous but crepitant lung (cf. pp. 111, 144). It is not accompanied by the high temperature of primary pneumonia nor by its other striking characters, and yields imperfect resonance rather than dulness, toneless or subcrepitant râles rather than true pneumonic crepitation. When recognised, it shows prostration in general, and weakness of the cardiac muscle in particular, and calls for ammonia and for brandy.*

Alcoholic pneumonia, or that which is found associated with delirium tremens, has been already mentioned (p. 994).

Inflammatory œdema and acute pulmonary congestion.—Dropsy of the lung is not infrequent in the course of Bright's disease; as with œdema of the glottis, it is probably always in some degree inflammatory. But apart from these cases there is an œdematous inflammation of the lungs which comes on acutely and without previous disease. It has been described as acute pulmonary congestion, and may or may not be identical with the first stage of hepatisation (p. 986). A case is narrated by Hertz in 'Ziemssen's Handbuch,' and another by Dr Leuf in the 'American Journal of Medical Science,' January, 1885. Such marked and severe cases are rare, but many intelligent observers believe that "active congestion" of the lungs, distinct from pneumonia and from phthisis, is a real and not infrequent malady.†

Chronic lobar pneumonia.—We have seen that one peculiarity of acute pneumonia is that, when the patient recovers, the hepatised lung clears up completely, and the tissue retains no trace of injury from the severe per-

* It is in this sense of hypostatic congestion that Arbuthnot's dictum applies: "A peripneumony is the last fatal symptom of every disease, for nobody dies without a stagnation of the blood in the lungs" ('Of Diet,' chap. iii).

† See on this subject Hodgkin's remarks ('Lectures on Morbid Anatomy,' vol. ii, p. 129).

version of structure it has passed through. But to this, as to every pathological rule, there are occasional exceptions. Abscess following pneumonia is so rare that its very existence is doubtful (p. 987); gangrene only ensues under special conditions, which will be discussed further on; and phthisis does not originate in acute lobar pneumonia.

But Addison described certain rare and exceptional cases in which, after an illness of a few weeks, with all the evidences of consolidation present, the patient dies, and the lung is found uniformly "albuminised." The section is not soft, lacerable, and granular, as in acute grey hepatisation, but smooth, solid, and tough. The alveoli are full of fibrillated lymph, and the exudation cells may have begun to be granular and fatty. It is a condition of the lung very liable to break down, and thus lead to rapid phthisis; on the other hand, it is a condition from which recovery may take place, and the lung be completely restored.

Wilks and Moxon, after quoting this account, continue, "That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must therefore believe that there is a true chronic pneumonia, whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term 'uniform albuminous induration.'"*

They add, however, that such chronic pneumonia may be of the lobular kind, and Addison himself said that this condition may be limited to one or a few lobules only; so that it may perhaps be doubted whether this "least frequent of the permanent pneumonic indurations of the lung" is not, after all, an unusually extensive catarrhal pneumonia.

BRONCHO-PNEUMONIA.†—In children, acute bronchitis affecting the smaller bronchial tubes is often accompanied by inflammation of the pulmonary alveoli, usually of both lungs. And even in adults the same thing is observed, although very rarely. Among children it is especially at an early age that this is apt to occur. Ziemssen, out of ninety-eight cases, found that sixty-seven—and Steffen, out of seventy-two-cases, found that fifty-two—occurred in patients under three years old.

Ætiology.—In many instances the affection is secondary to some infective disease. Measles and whooping-cough furnish by far the largest number of cases of broncho-pneumonia. It is also sometimes seen after diphtheria, rubeola, smallpox, or scarlet fever. Jürgensen insists, as other writers had before, on the greatly increased liability to broncho-pneumonia, as a complication of bronchitis (whether after measles or independently of any infective disease), in those who are obliged to breathe impure air in the close, narrow rooms of the poor. He is even disposed to attribute the greater frequency of the affection in winter to defect of ventilation rather than directly to inclemency of season. It is probable that rickets renders a child more apt to become affected with broncho-pneumonia if it is attacked with bronchitis. Dr Wilks, in the 'Guy's Hospital Reports' for 1860, showed that broncho-pneumonia is a common cause of death in children suffering from severe burns.

* 'Pathological Anatomy,' p. 338. Addison's original description appeared in the 'Guy's Hospital Reports' for 1843, and will be found in the volume of his collected papers published by the New Sydenham Society, p. 27.

† *Syn.*—Catarrhal pneumonia, Lobular pneumonia, Pulmonary or Alveolar catarrh.

As to the special causes of the exceptional cases when broncho-pneumonia like that of infants occurs in adults, it is at present difficult to say anything definite. The following are a few cases which occurred at Guy's Hospital. A woman, aged thirty, having miscarried eight days before, was attacked with shivering and headache, and died after an illness that lasted ten days. A man, aged twenty-five, of dissipated habits, who was said to have been under a course of mercurial treatment for syphilis, received a blow on the nose while he was drunken; this was followed by epistaxis, which continued until the nares were plugged; the mucous membrane then suppurated profusely, the discharge being very fœtid; and he sank and died at the end of a week. A third case was that of a man aged twenty-eight, a singer at a music-hall. He had a fall from a cart, and this led to an illness which proved fatal in three weeks. At first he tried to go on with his singing, but he was soon obliged to give it up. When admitted, shortly before his death, he was already comatose. In this instance some of the patches showed central sloughs, but in the reports of the two other cases it is expressly stated that the appearances were identical with those that are commonly seen in children. It seems probable that in the second case the inflammation was set up by the inhalation of fœtid pus from the nasal cavities into the air-passages. Indeed, the report of another case, in which a similar affection of the lungs was met with as a complication of tubercular meningitis in a woman aged thirty-four, attributes broncho-pneumonia to the entrance of food into the bronchial tubes while she was struggling in her delirium.

Breathing irritant gases, especially chlorine in large quantity, sometimes sets up intense broncho-pneumonia. It is not improbable that the immediate cause of the ordinary broncho-pneumonia of children is often, if not always, the entrance into the alveoli of inflammatory products formed in the smaller tubes, as the result of violent inspiratory efforts.

It is also said that broncho-pneumonia is not infrequent in extreme old age, and that it often ends the life of those who have long laboured under senile bronchial catarrh.

Anatomy.—The most obvious indication that pneumonic consolidation in a given case has arisen by extension from the bronchial tubes is its occurring in scattered patches throughout the lungs. These patches are more or less rounded in form; they are usually of about the size of peas, but sometimes may be larger. They are reddish brown in colour, or more or less grey, or greyish yellow, according to the stage to which the inflammation has advanced. As seen upon the cut surface of the lung, they appear slightly raised above the rest of the parenchyma. They have a dull, lustreless appearance, and are granular on section, but the granulations are less obvious than in the red hepatisation of genuine acute pneumonia. The substance of the patches is soft and friable, and when squeezed they emit a more or less opaque fluid. They are sometimes so closely collected together in part of a lung that a whole lobe may seem to be consolidated. Even then, however, the lobular markings are usually distinct, and the cut surface has not that even and uniform appearance which characterises true lobar hepatisation. Many lobules are found collapsed and others dilated.

More or less pleurisy is very commonly associated with broncho-pneumonia, there being a thin layer of lymph upon the pleural surface, especially over any patches that happen to lie just beneath the serous membrane. Indeed, both at the bedside and in the deadhouse it is a remarkable fact

that, altogether apart from the presence of any obvious pneumonia, pleurisy is a far more frequent complication of bronchitis than one would have anticipated: in adults affected with bronchial inflammation a pleuritic friction-sound may often be detected if it is listened for.

Histologically, the morbid process in broncho-pneumonia is a *catarrhal* inflammation; that is to say, the cells which fill the affected alveoli are many of them epithelial in character, large, of irregular shapes, with bold nuclei. With them are found leucocytes and mucus, but neither blood-discs nor fibrin. Probably the seat of the disease is the intra-lobular air-passages, and the contents of the vesicles are derived from the former source.

In some cases the inflammation appears to spread to the connective tissue by which the pulmonary lobules are united together. Jürgensen speaks of "thick whitish-grey bands, which are seen crossing one another upon the cut surface of the organ." In 1878 a well-marked instance of this occurred in a child, aged three, who died after an illness of five weeks' duration, which perhaps began in whooping-cough. The left lung contained many patches of broncho-pneumonia. The right lung was almost entirely consolidated. Its tissue, however, felt hard; and the fibrous tissue in it had obviously undergone a great increase. Such cases seem to suggest an explanation of an appearance which one not infrequently sees in making autopsies of persons at all periods of life, namely, marbling of a part or the whole of a lung by fibrous bands which intersect one another and divide it up into irregular areas. Such an affection is commonly regarded as indicating early cirrhosis of the organ. But it does not look like a progressive lesion, and is far more likely to be a residue of a former attack of inflammation. Acute lobar pneumonia appears never to cause this appearance.

Clinical course.—The recognition of broncho-pneumonia, in addition to the acute bronchitis which precedes and gives origin to it, is usually more or less uncertain. Physical examination of the chest often helps but little. If several lobules side by side beneath the pleura are consolidated, there may be impairment of resonance (or even dulness) on percussion, bronchial breathing and bronchophony, the latter being especially obvious when the patient, if a child, is crying. Moreover, the bronchitic râles often become clear and "consonating" in quality. Jürgensen lays stress on the occurrence of a crepitating râle like that heard in acute pneumonia, but less fine, and audible during expiration as well as inspiration.

Nor, again, is broncho-pneumonia attended by very marked symptoms. Ziemssen has rightly laid stress on the importance of a sudden rise of temperature, as an indication of its supervention in the course of acute bronchitis, if the pyrexia should previously have been moderate. But, as already stated, the thermometer may indicate 104° or even higher in a child affected with bronchitis, independently of any complication (p. 957). Jürgensen says that broncho-pneumonia—as, for example, after measles—may be accompanied with a temperature of 105·8° for days together, with scarcely any remissions. In fatal cases the temperature sometimes rises before death, reaching perhaps 107° or 108°; sometimes it falls below normal. The pulse is often extremely rapid, so that it cannot be counted at the wrist. In children a pulse rate of 150 to 200 is by no means incompatible with subsequent recovery; so that it must not lead one to give an absolutely fatal prognosis. It is surprising, too, how hurried the breathing may be in cases which yet do perfectly well. What is really alarming is for the pulse to be feeble and "running" from emptiness of the arteries. When the

disease ends in recovery, the pyrexia and the other symptoms gradually subside (*lysis*); there is never a sudden fall of temperature (*crisis*).

Herpes on the lips seldom accompanies this affection. Albuminuria and diarrhœa are not infrequent. As regards what may be termed chest-symptoms, it often happens that with the supervention of broncho-pneumonia the cough from which the patient had been suffering ceases, or becomes less loud and shorter in character. Usually no expectoration makes its appearance. There is generally extreme restlessness, the child tossing about in bed, and asking to be taken up by its nurse and shifted in position every few minutes. The breathing is often shallow. The face and the lips are apt to become pale, with perhaps a livid blush upon the cheeks. There is on the whole more dyspnœa and distress than in cases of lobar pneumonia.

Diagnosis.—One point, of great clinical importance, is that broncho-pneumonia sometimes gives rise to cerebral symptoms of so prominent a character that one may easily suppose the case to be one of *tubercular meningitis*. Delirium, coma, retraction of the occiput, vomiting, strabismus, convulsions, may all appear in turn. Jürgensen makes the formal statement that, in a child affected with broncho-pneumonia, it may be absolutely impossible to say with certainty whether these symptoms are due to tubercles present in the brain. Of course the question is then whether the pulmonary affection is not really *acute tuberculosis*; and, apart from nervous symptoms, this is often difficult to decide.

Broncho-pneumonia in children may be mistaken for *enteric fever*. Rose spots are far from constant in children; diarrhœa is frequent in broncho-pneumonia, and often absent in enterica; but a full abdomen and a swollen spleen point to the latter, and the curve of temperature, with its relation to the respiration, will generally decide the question.

Prognosis.—The ordinary duration of broncho-pneumonia is about a fortnight; but sometimes it runs on for three or four weeks or even longer. It may destroy life very rapidly—within a few days, or even in twenty-four hours. Sometimes the cause of death seems to be marasmus, all acute symptoms having passed off. Jürgensen gives some figures from which it appears that the disease is fatal in from half to two thirds of the cases. When consecutive to measles the risk is said to be smaller than this. Convulsions are of bad omen. Very young infants are far more likely to die than older children; and the prognosis is also more serious in those who are weakly, rachitic, or very fat. In children it is far more dangerous a disease than lobar pneumonia. Of forty-five cases of acute lobular broncho-pneumonia in children, recorded by Dr Goodhart, twenty were fatal. He notes the frequency with which, when one lung is inflamed in a child, the other shows signs of the same process here and there in a partial and often transient form.

Treatment.—Emetics do good service in the first few days; afterwards they are too depressing. Expectorants are useful, but opium must be avoided. The ammonia and senega mixture is almost always valuable; and sweetened with glycerine or treacle or syrup of tolu it will be taken by children as well as other medicines. Counter-irritation is often useful, and mustard poultices are well borne by the skin of even young children. Dr Eustace Smith recommends dry cupping of the back in bad cases. Diarrhœa should be checked by chalk powder. Stimulants are almost always necessary—sweetened brandy and water, rum and milk, or the egg-and-brandy mixture.

During convalescence, which is often long and tedious—contrasting with

that of lobar pneumonia—quinine and iron are indicated, with cod-liver oil; and it is desirable to remove the child to the sea-side as soon as practicable.

Vesicular pneumonia is a name used by Stokes, and applies to the first stage of broncho-pneumonia. Usually, in all probability, it soon becomes lobular, but occasionally it may be seen after death as a series of minute dots over the cut surface of a lung, too small for lobules, though too large for single vesicles, and these are found to contain catarrhal products. The same appearance to the naked eye may be produced by cross-section of small bronchial tubes, each surrounded with peribronchial inflammation.

When catarrhal or lobular broncho-pneumonia becomes chronic, the products are apt to undergo *caseous degeneration*. Instead of being removed by expectoration or by disintegration and lymphatic absorption, they remain and set up a spreading alveolar catarrh, which goes on to, but unfortunately rarely ends in, a local tubercular process. This is the ordinary beginning of phthisis, and will be discussed in a future chapter.

The term "lobular pneumonia" is also applied to the suppurative process which attacks the lungs in *pyæmia*. It is lobular in distribution, but its exudation is neither catarrhal, caseous, nor fibrinous, but purulent; and it never occurs except as part of a general process of embolism and septicæmia. The modern doctrine of pyæmia was to a large extent worked out by Virchow and Cohnheim in the case of the pulmonary circulation. When the embolus is non-infective, it produces hæmorrhage by the same mechanism as in the case of the brain; but there is no reason to suppose that this is the only cause of pulmonary apoplexy. Under extreme congestion from obstruction on the left side of the heart the capillaries of the alveoli may give way; or extravasation may occur from changes in the vessels, or in the blood itself, as in cases of purpura hæmorrhagica. When the embolus is infective it produces intense congestion, and soon after suppurative inflammation. The abscesses (or earlier only congested patches) are multiple, and are most numerous in the back part of the lung, and generally near the surface.

The symptoms of this form of lobular pneumonia are merged in those of the fatal disease of which it is part (p. 67), but its presence may be suspected when a case of pyæmia is complicated by pain and other signs of pleurisy, which is caused by an abscess reaching the surface of the lung.

CIRRHOSIS OF THE LUNG.*—This term was first used in 1838 by Sir Dominic Corrigan for an affection of the pulmonary tissue, consisting in a replacement of its alveolar structure by a fibroid material, histologically analogous to that which in the liver causes the disease known as cirrhosis of that organ. Unfortunately, however, the mere presence of such a fibroid material in the lung is far from being characteristic of any one morbid process. The condition which has been termed "fibroid phthisis" is probably, in all but an insignificant minority of cases, nothing but a regressive stage, or a very chronic form, of a really tubercular process. There is reason to believe that the same is also true of the pulmonary diseases which are set up by the inhalation of dust. The question will again come before us when we deal with phthisis and pneumo-coniosis.

There remain cases which are not tubercular, and never have been. One

* *Synonyms.*—Chronic interstitial Pneumonia—Iron-grey Induration of Addison—Induration ardoisée of Cruveilhier—Induration grise et mélanique of Andral—Fibroid lung.

peculiarity that characterises them is that whereas the whole of one lung may be shrunken into a hard grey mass, showing no trace of its proper structure, the other one is perfectly free from any similar morbid process; whereas in phthisis, long before one lung is completely destroyed, the other always becomes involved in the disease. Again, in these cases, when one part of a lung is affected before the rest, it is usually the lower lobe. In each of five cases of double cirrhosis observed at Guy's Hospital the bases of both lungs were found to have undergone cirrhosis; in not a single instance was there any indication of the presence of tubercle, nor were any caseous nodules or masses present. These cases are usually complicated by pleuritic adhesions on the affected side, and often by bronchiectasis.

Anatomy.—The process begins, according to some pathologists, in peribronchitis, which spreads from the smaller tubes to the elastic tissue surrounding the alveoli; according to others, in a chronic inflammation of the subpleural and interlobular connective tissue. Both origins, however, are denied by others, and Wilks and Moxon state decidedly that the process commences in the alveoli and their walls. When fibro-nucleated material occupies the pulmonary structures, and cells and nuclei are found within the alveoli, they refer these cases to double chronic pneumonia. Of cirrhosis affecting one lung only, with thickened pleura and dilated tubes, the same authors say that sections of the lung show not only fibrous tissue surrounding the bronchial tubes and blood-vessels, but also thickening of the alveolar walls. This is well shown in a drawing given by Dr Coats (fig. 231), in which the alveoli are represented as lined with large nucleated epithelioid cells, quite unlike those of the normal lung, a condition also represented in a drawing by Heschl, reproduced in the late Dr Wilson Fox's article in 'Reynolds' System,' p. 765. When the fibroid degeneration has reached its full development, the lung on section exhibits large tracts of white, slate-coloured, black, or marbled iron-grey tissue, which is completely airless, firm, and sometimes of cartilaginous hardness. The microscope shows only dense, white, fibrous tissue, with no trace remaining of the pulmonary structures which it has displaced.

Origin.—This chronic fibroid induration is an important conservative process in phthisis, few cases of which are entirely without some cicatricial induration; but as a process independent of tubercle its pathology is still a matter of uncertainty; possibly it has more than one mode of origin.

(1) Now and again it seems to have begun in an attack of acute lobar *pneumonia.* This question was already touched upon (pp. 988, 1003) when the opinion of Dr Wilks was cited, that acute lobar inflammation of the lung, if it does not prove fatal, always undergoes resolution, and never leads to a chronic induration of the pulmonary tissue. But, after all, the evidence on which this opinion was based must have been of a negative kind, consisting in the observation of a number of cases, in none of which such a result was noted. In hospital practice it can very rarely happen that a patient comes under the eye of the same physician during an acute illness, and again, months or years afterwards, when a chronic affection of the lung proves fatal. Moreover, medical literature contains several carefully recorded cases in which the origin of cirrhosis in pneumonia appears to have been clearly established. The two following are taken from Dr Bastian's collection of thirty cases, tabulated in the second volume of 'Reynolds' System.' The first occurs in Charcot's 'Thèse de Paris.' The patient, a man aged sixty-one, was admitted on March 30th, 1850. He had

been attacked five days before with rigors and pain in the side, and had rusty sputa. There were all the signs of pneumonia affecting the whole of the right lung. These continued with but little alteration until his death on July 19th. At the autopsy the right lung was of a greyish-blue colour on section, as hard as cartilage, shrunken to two thirds its natural size, and enveloped in an immensely thickened fibrous mass. The other case was recorded by Dr Mayne ('Dublin Hosp. Gaz.,' May, 1857). It is that of a man aged fifty-four, who in July, 1855, after a hard day's work, was seized with rigors and all the symptoms of pleuro-pneumonia. The acute disease subsided, but he never afterwards regained his health and strength. In October, 1856, he was attacked with fresh febrile symptoms, and he died at the end of the year. The lung, on *post-mortem* examination, was found affected with well-marked cirrhosis.

Dürr has recorded, in a volume published by Jürgensen, two cases which occurred in very young children. In each of these the primary attack appeared to be one of lobar, not catarrhal, pneumonia. Probably, however, the "pneumonia" which leads to cirrhosis is as a rule lobular and catarrhal, and in children it is often secondary to measles or whooping-cough.

To this origin in alveolar catarrh Dr Fagge was disposed to refer one variety of the affection, in which, instead of the tissue of any part of the lung being all converted into a fibrous substance, it is marbled or intersected by bands crossing one another, so as to split it up into areas of various sizes and shapes. The clinical significance of this appearance, which is not uncommonly seen in the deadhouse, is not known. It is generally supposed to represent an early stage of cirrhosis, such as might, if the patient had lived, have involved the whole lung. But may it not be rather a residue or relic of a former acute process?

Many pathologists who find difficulty in admitting the origin of pulmonary cirrhosis in true lobar pneumonia would readily allow the probability of so much more chronic, more irregular, and, so to speak, more irritative a process as lobular or alveolar catarrh, being the antecedent of iron-grey cirrhotic induration.

(2) A frequent concomitant of cirrhosis is *pleurisy*.

In most cases the pleura over the affected part of the lung is adherent to the parietal layer, and the two together form a dense white mass, of cartilaginous hardness and from a quarter of an inch to an inch in thickness. The only way to remove the lung from the chest at the autopsy is to cut it out with the knife. There is also a similar thickening of the pleural layers separating the different lobes. This state of the serous membrane has led to the supposition that the affection began in an attack of pleurisy, and that the lung-substance was invaded from the surface by extension along the interlobular septa. A case showing how easily erroneous conclusions may be drawn, even from *post-mortem* observations, occurred at Guy's Hospital in 1877. A man aged twenty-seven died of disease of the left side of the chest, with a history of having had inflammation of the lungs at the age of fifteen, and some thoracic affection even before that, in early childhood. There was a localised empyema in front, and the pleura elsewhere was thickened in places to the extent of an inch. The lung was very small, but its tissue was generally healthy, except that it was intersected by fibrous bands. It would have been natural to infer that the organ had been invaded from without. But the apex of the other lung was affected in a precisely similar way, although the pleura covering it was in a normal state.

(3) *Dilatation of the bronchial tubes* in the affected lung or part of a lung is present in most cases of cirrhosis.

So intimate, indeed, has been supposed to be the connection between these two morbid conditions, that Grainger Stewart and Jürgensen discuss them together. But various forms of bronchiectasis occur without there being any change in the pulmonary tissue, unless it be emphysema. And in six of Dr Bastian's thirty collected cases of cirrhosis it is expressly stated that the tubes were of their natural size. As to the relation between the bronchial dilatation and the affection of the lung, when they coexist, there are differences of opinion. Some think that a chronic inflammatory process may start from the smaller tubes, and lead to a gradual fibrous thickening of the alveolar walls, with obliteration of their cavities. But the very definite localisation of the morbid process, the completeness of the destruction of the pulmonary texture, and the fact that the pleura becomes so greatly thickened, are arguments against this view. By Corrigan it was suggested that the occurrence of bronchiectasis was secondary to the cirrhosis; he imagined that the contraction of the adventitious fibrous material in the lung dragged upon the walls of the tubes, so as to widen their channels. Dr Wilson Fox, however, seems more likely to be right in thinking that if the dilatation of the tubes really follows the lung affection, it is caused by the expiratory force of a cough, exactly as it is in other circumstances; at an early stage of the disease it is reasonable to suppose that all the affected structures may be soft and yielding.

On the whole, it appears premature to refer the origin of cirrhosis to a starting-point other than the pulmonary alveoli. Many of those cases which cannot be traced to an antecedent pulmonary attack of inflammation, may have arisen from an illness that occurred in childhood and had been forgotten. In others the morbid process may be chronic from its commencement, a true primary chronic inflammation of the lung.

Incidence.—Cirrhosis is most apt to occur in adult men. In Dr Bastian's list there were 24 male and 6 female patients. Two were only seven, 3 were between sixteen and twenty, 21 between twenty-four and fifty-seven, and 4 between sixty-one and seventy-one. In 1887 we had a typical example in Mary Ward, in a boy seven years old.

Symptoms.—As a clinical disease cirrhosis varies according to its extent.

If it affects only a portion of one lung (or even of both), the patient becomes a chronic invalid, with cough, dyspnœa, and most of the other symptoms of phthisis. The expectoration is purulent, sometimes stained with blood, sometimes dirty-grey in colour and offensive; the fingers become clubbed, and there is more or less marked emaciation. The physical signs are those of partial consolidation of the lungs, with the addition of more or less abundant râles. Such cases are not uncommon in hospital practice. After staying a certain number of weeks in the ward, the patient is discharged much as he came in, or at best with some relief to his symptoms. In the five cases of cirrhosis of the bases of both lungs, above referred to (p. 1008), the cause of death was either quite unconnected with the pulmonary affection, or it was an attack of acute pneumonia, or lardaceous disease of the kidneys, the result of the protracted suppuration.

Very different is the course of cirrhosis when it involves the whole of one lung. In that case the affected side of the chest falls in, so that it measures in each direction much less than the other side.

There is often considerable difficulty in distinguishing the disease from chronic pleurisy or emphysema with retraction, after paracentesis. According to Dr Walshe, there is not in cirrhosis the same degree of twisting of the ribs on their axes as in pleurisy, nor is the shoulder lowered so much, nor is the inferior angle of the scapula tilted so far outwards. Great assistance in the diagnosis may be afforded by the discovery of râles on auscultation, and by the presence of abundant purulent, and perhaps offensive, sputum.

From a malignant tumour cirrhosis is commonly distinguished by the state of the mediastinum. This is pushed away by the growth of a tumour, and is dragged over to the farthest possible extent by cirrhosis. If the right lung is cirrhosed, the heart is seen beating at the right nipple; if the left, its visible pulsation may extend upward nearly to the left clavicle. In either case the opposite lung undergoes an extreme degree of enlargement, so that the whole sternal region, and even the space beneath the costal cartilages on the affected side, becomes resonant on percussion, and transmits to the ear a loud vesicular murmur. After a time, however, this over-distended and perhaps hypertrophied lung fails to carry on the respiratory function efficiently. The right side of the heart becomes dilated, a tricuspid regurgitant murmur develops itself at the ensiform cartilage, the patient suffers from permanent orthopnœa, the liver becomes nutmegged, and ascites and œdema of the lower limbs set in. The case, in fact, assumes all the characters of chronic valvular disease of the heart with dropsy, and terminates fatally in the same way.

The *prognosis* of pulmonary cirrhosis is like that of the more chronic cases of phthisis.

Of the *treatment*, all that need be said is that for the relief of the different symptoms such remedies must be used as are recommended for the like symptoms in other diseases, such as phthisis, bronchitis and bronchiectasis, which are more commonly met with in practice.

SYPHILITIC DISEASE OF THE LUNGS.—Only within the last thirty years has it been recognised that other viscera than the testis, and other regions than those accessible to the surgeon's touch, may be seats of syphilitic disease. It is now certain that (apart from the question whether phthisis is ever of syphilitic origin) there is a form of chronic interstitial pneumonia, or cirrhosis of the lungs, characterised by deep scarring and contraction of the tissue, by peribronchitis and tracheitis, often with ulceration and deformity, and by the presence of typical gummata. The process affects the roots or bases rather than the apices; it often starts from previous ulceration of the air-passages; and it is apt to lead to gangrene.

The disease is a rare one, and would be little but a pathological curiosity were it not for the importance of recognising the true pathology of these cases in order to treat them successfully.

Lancereaux described cases of this kind in France ('Traité de la Syphilis,' 1866); Dittrich and Virchow in Germany ('Kr. Geschw.,' ii, 463); and Wilks in this country ('Path. Trans.,' vol. ix, 1858, with plate, and 'Guy's Hosp. Reports' for 1863, 3rd series, vol. ix, p. 33, two cases).

In a series of twenty-two cases of visceral syphilis brought before the Pathological Society in 1877, Dr Greenfield described three of presumably syphilitic affection of the lungs (vol. xxviii, p. 258). In the same volume is the description of a specimen shown by Dr Sutton of chronic syphilitic

pneumonia, from a patient under Dr Gull and Mr Durham in Guy's Hospital (p. 304); three of "fibroid phthisis" in syphilitic patients, by Dr Goodhart, with a histological drawing in which peribronchitis is clearly shown (p. 313, and abstracts of nineteen cases from Guy's Hospital, p. 322); one apparently of gummata coalesced into a large mass in one lung, by Dr Gowers, with a histological drawing (p. 330); one by Dr T. H. Green of a similar large mass in one lung of a man with "undoubtedly syphilitic lesions in his liver;" two by the late Dr Mahomed—both in women with undoubted syphilitic disease, but the pulmonary lesion less certainly of the same origin and perhaps in an early stage; lastly, three of gummata in the lung from the museum at Netley by Dr Aitken.

The following cases have come under the writer's notice:—(1) A woman of about forty at the Hôpital Beaujon in Paris. She was wasted, with cough, purulent expectoration, and hæmoptysis; and from other proofs of lues was treated with perchloride of mercury to her great benefit. (2) A groom of about thirty who, together with signs of chronic pneumonia and hæmoptysis, had tibial nodes and amygdaloid lymph-glands. He improved greatly under iodide of potassium and mercury, with which, however, it is right to add, cod-liver oil was given; and when last seen he had gained weight, was free from pain, and able to resume work.* (3) A strong and muscular seafaring man about forty, who had symptoms and physical signs resembling phthisis, but was well nourished and had a good appetite. A gumma was discovered near the hip-joint; he was put on iodide of potassium, and under this treatment not only did the node disappear, but his cough and other pulmonary symptoms ceased, and he was to all appearance cured. (4) A patient seen with Dr Miller, of Norwood, who had certainly suffered from syphilis, and in whom there were physical signs of very local consolidation in one lung, together with laryngeal ulceration and hæmoptysis. He was treated with mercury, but died from stenosis of the air-passages before much good could be effected. An autopsy was obtained, and beside deformity and contraction of the trachea and bronchi, there were several gummata in the right lung, with scarring and fibrous induration. (5) A patient in Guy's Hospital, aged forty-two, with signs of chronic disease of the lungs, which had been called consumption, and with no history or signs of past syphilitic lesions. The nature of the case was not recognised until after the man's death from eclampsia. Previous to this severe caries of one ulna had led to such extensive suppuration that the arm was amputated. At the autopsy were found caries of the frontal bone with pachymeningitis, fibroid testes, and two small fibroid patches in the left ventricle. The two primary bronchi were contracted and deformed. The right lung contained a single fibroid nodule in the lower lobe; the left was solid in patches, firm and grey, with dilated tubes. There was nothing that could be called tubercular in either lung, the larynx and ileum were healthy, and there were no miliary tubercles anywhere to be found ('Path. Trans.,' 1877). Microscopical examination of the indurated tissue in the lung showed it to consist of a fibro-nuclear growth with numerous vessels. (6) A sailor aged forty-four, in Guy's Hospital, February, 1876. There were signs of chronic phthisis with hæmoptysis; a history of chancre, but none of secondary lesions, and intense dyspnœa, evidently from obstruction to entrance of air. The larynx was perfectly normal, and there were no signs of aneurysm or thoracic tumour pressing on trachea. Tracheotomy was therefore not performed, and the

* This case closely resembled one recorded by Dr Walshe on p. 513 of his 4th edition.

patient died six days after admission. There was found after death ulceration with stenosis of the trachea, "fibroid phthisis" with one old vomica and much puckering and cicatrisation. There were scars on the surface of liver, but no actual gumma. The liver and kidneys were lardaceous.

Between *syphilis pulmonum* and chronic interstitial pneumonia (so-called fibroid phthisis, or cirrhosis of the lungs) there is no anatomical distinction but the presence of gummata. But we have two unfailing criteria, one in the associated changes in other viscera, the other in reaction to treatment.

Syphilis is no protection from true phthisis, and what has been called syphilitic phthisis is in most cases nothing more than true tubercular phthisis in a syphilitic subject, which runs its course uninfluenced by the latter disease. But there is also a form of chronic pneumonia, with fibroid induration and bronchiectasis, with irregular local distribution, with no tubercle and little or no caseation, which starts either from gummata or from thickened patches of pleura, or from a chronic contracting peribronchitis. This peribronchitis is associated with an ulcerative inflammation of the trachea or bronchi, or both, which is closely related to the ordinary syphilitic inflammation of the larynx. The symptoms during life are indistinguishable from those of ordinary phthisis, though the physical signs point to a more chronic and fibroid, less acute and caseous, form of disease. If the physical signs are confined to one lung and absent from the apices, one may suspect the true nature of the case; but it is only by concomitant lesions of other organs and by the effect of treatment that we can establish the diagnosis during life. Hæmoptysis is often a striking feature. Dr W. H. Porter, of New York, mentions tenderness of the tibiæ and sternum on pressure as a symptom of value. The absence of the bacillus tuberculosis is a most important diagnostic sign during life.*

Hereditary syphilis of the lungs.—It is quite possible that some of the cases of gummata with cicatrices and chronic induration just described may be due not to acquired, but to congenital syphilis.

But there is another form of pulmonary disease which appears to occur only in children who show signs of hereditary lues. It is uniform, without gummata or cicatrices, and with no primary lesion of the trachea and bronchi, or of the pleura. Lungs in this condition have been described by Wagner and Virchow in Germany, by Robin in France,† and by Wilks and Moxon in this country, as a diffused form of hepatisation, firm, dense, or even tough in texture, white in colour, and airless. They are sometimes both affected, more often one, either throughout a lobe or in smaller circumscribed masses. The more universal cases are, as might be supposed, found in stillborn children, or in those who only survive birth a few days. The condition seems to be essentially chronic thickening of the alveolar walls and of the interlobular septa, whereby the alveoli are compressed, and the part affected rendered heavy, bulky, and more or less completely solid. On certain points there is conflicting evidence. Some writers describe the diseased patches or lobes as quite exsanguine with obliterated capillaries; others speak of free production of new vessels, so that the new growth is very vascular. Dr Greenfield described the lung of a presumably syphilitic child, twelve months old, in the 'Pathological Transactions' for 1876 (xxvii,

* Beside the papers quoted above, the following may be mentioned:—Dr Porter ('New York Med. Journ.,' July, 1885), with plate; Dr Moxon ('Guy's Hosp. Rep.,' 3rd series, vol. xiii, p. 374); Dr Goodhart (*ibid.*, vol. xxv, p. 31); the late Dr Wilson Fox's article in 'Reynolds' System;' and Dr Bäumler's in 'Ziemssen's Handbuch.'

† Lorain and Robin call it "epithelioma" of the lungs.

p. 43). It was tough, yellowish white in colour, with a smooth (not granular) section, and exuded very scanty fluid. Histological drawings are given which show bands of fibrous tissue obliterating the alveoli, of which the walls are remarkably thickened, but the endothelium is unaltered. There was unfortunately no positive proof of syphilis in this case; but Dr Goodhart mentions a specimen ('Diseases of Children,' chap. xxii) in which similar *post-mortem* appearances were found in a child three months old, together with undoubted syphilis of the liver. Microscopically, it showed all the features exactly as described by Dr Greenfield —excessive fibro-nucleated growth, extreme vascularity, and collapse of the air-vesicles.

The symptoms observed during life appear to be inconspicuous.

GANGRENE OF THE LUNG.—This affection bears out the statement made with regard to gangrene in general (p. 54), namely, that the death of any part of the human body is always either the result of an intense inflammation, or else of any injury which, if a little less severe, would have set up inflammation, but which by its violence kills the tissues outright before there is time for inflammation to occur.

Anatomy.—Of pulmonary gangrene without antecedent pneumonia it would be difficult to find unequivocal examples. But in the *post-mortem* room cases are sometimes seen in which there has been rapid and extensive sloughing of a portion of a lung, and in which no zone of hepatised tissue separates the gangrenous part from that which is healthy or merely œdematous. In such cases the fact of there having been inflammation is unproven. Generally, however, the sloughing mass lies within a more or less broad area of consolidated lung-substance, of which it had evidently at one time formed a part. Should the disease have proved fatal at an early stage, one condition may gradually merge into the other; should it have reached a more advanced stage, there may be a well-marked line of demarcation, or the dead tissue may have been cast off with the formation of a cavity. Experience in the deadhouse does not support the distinction between two separate forms of pulmonary gangrene, the one "circumscribed," the other "diffuse," though the distinction has been taught since the time of Laennec, who was the first to recognise gangrene of the lungs as a special affection.

The diseased part is of a dirty greenish brown or black colour, and so soft as to be sometimes almost diffluent. It is often horribly fœtid, but occasionally the odour has been little marked, as Cruveilhier long ago noticed. Microscopically, the pulmonary structure is hardly to be recognised, the alveolar walls having broken down into granular detritus.

Origin.—Pneumonia (*i. e.* the acute disease which causes lobar hepatisation) rarely leads to gangrene, except in very old and feeble persons, in drunkards, and in those who are exhausted by some other malady—for example, by diabetes. But one or two instances have occurred in Guy's Hospital, of what had appeared to be an ordinary attack of pneumonia in a healthy subject which, when the acute stage subsided, was followed by symptoms which seemed to indicate that at least some of the hepatised tissues had undergone sloughing. Dr Walshe records just such a case—that of a man who was slowly recovering from an attack of pneumonia of the right lower lobe, when hæmoptysis set in, and was followed by the copious expectoration of a frothy, intensely fœtid sputum, while at the same time physical signs like those which indicate the formation of a cavity

made their appearance. Ultimately this patient left the hospital in fair general health, and free from all physical signs except those commonly denoting consolidation.

Another pulmonary disease, in the course of which gangrene may occur, is phthisis. Dr Walshe speaks of having seen some six cases in which the special fœtor appeared incidentally in connection with *tuberculous vomicæ* already formed. Pulmonary gangrene may also complicate cirrhosis and bronchiectasis.

But in the majority of cases gangrene of the lung arises out of an inflammatory process of *septic* character. There are various ways in which such a process may be set up. Sometimes it is by direct extension from neighbouring parts, as when an ulcerating cancerous growth in the œsophagus eats its way into the lung, or when perforation occurs from a suppurating hydatid or from simple abscess of the liver, or from suppuration spreading from an ulcer of the stomach, or from a putrescent empyema. Sometimes the infection is brought by the blood-vessels. Thus septic emboli may become lodged in branches of the pulmonary artery, either derived from a cerebral sinus, which was inflamed after otorrhœa, or from a systemic vein in the neighbourhood of unhealthy inflammation, or from the right side of the heart in a case of ulcerative endocarditis. In yet other cases the disease starts from the bronchial tubes. A foreign body, as a piece of bone impacted in one of the bronchi, is very apt to set up a sloughing pneumonia; or it may be caused by the entrance into the air-passages of pulpy or liquid food, as in patients who have chronic laryngeal disease, or in those who are comatose from apoplexy, or in lunatics who have to be fed by force. A like result may be produced by matters from the stomach drawn into the lungs during the act of vomiting, especially in persons rendered insensible by anæsthetics; we have had instances of death brought about in this way in cases of hernia or of intestinal obstruction, in which there had been a copious discharge upwards of the contents of the small intestine. Again, putrid materials that pass into the air-passages may be originally derived from the mouth, as in cases of gangrene of the cheek or of the tonsils, of diphtheria of the fauces, or of sloughing cancer of the tongue; Volkmann has suggested that sometimes a severe disease of the ear may lead directly to pulmonary gangrene, from morbid secretions dropping down through the Eustachian tubes into the pharynx, and not (as is more usual) through the occurrence of thrombosis and embolism. Lastly, there are cases in which a sloughing pneumonia is due, not to the entrance into the bronchi of matters from without, but to the decomposition of retained secretion.

Sputum.—In fœtid bronchitis, as we have seen (p. 975), the patient's breath and his expectoration may have either the true odour of gangrene, dependent upon the decomposition of dead tissues, or a peculiar nauseous acrid odour, which is sometimes not unlike that of fæcal matter, and which appears to be due to the presence of free fatty acids.

In the latter case the sputum presents the further peculiarity of separating into three layers, in the lower of which are found certain masses or "plugs," consisting of exudation that has accumulated in the dilated tubes, and undergone chemical and other changes there (p. 974). But the same description does not apply to other forms of sloughing of the lung; for unless there is an antecedent bronchiectasis, the peculiar "plugs" cannot be formed, nor is there any reason why fatty acids should be set free. The odour in all other cases is simply that of gangrene—an indescribable

fœtor, but one which is always of the same character, though it varies greatly in intensity, being sometimes only just perceptible, sometimes so strong as to poison the whole air of a room. As a rule, the patient's breath has the same smell as the expectoration, especially after coughing; and it now and then happens that the breath is characteristically offensive for some days, while the sputum remains odourless. Moreover, there are cases in which during life no fœtor is discoverable, either in the breath or in the expectoration, so that the presence of gangrene may not be suspected until it is seen at the autopsy. Hertz (in 'Ziemssen's Handbuch') accounts for the fact by assuming that the tubes passing from the sloughing parts are obstructed by secretion. But, judging from experience in the deadhouse, it would seem that fœtor is most often absent before death in cases of acute gangrene, which would be commonly described as belonging to the "diffuse" variety of the affection, and in which, therefore, it is most unlikely that obstruction of tubes would be present.

The fœtid sputum of gangrene of the lung is commonly of a dirty grey or greenish colour; sometimes it is brown or almost black, from the presence of altered blood. The microscope does not often lead to the detection of recognisable fragments of pulmonary tissue, although such fragments are so often to be found in cases of phthisis. Obvious hæmoptysis is said to occur seldom in adults, but frequently in children. Fatal hæmorrhage, due to the erosion of a large vessel, is an event of great rarity. When the surface of the lung is affected the pulmonary pleura usually gives way, leading to the formation of pneumothorax, which is quickly followed by septic pleurisy. If there should happen to be local closure of the serous cavity by adhesions, it is said that a subcutaneous emphysema may develop itself, or that an abscess may form, which may open externally after burrowing to a greater or less distance. Another occasional effect of the presence of a patch of gangrene in the lung is said to be the dropping of putrid matters into tubes belonging to other parts of the organ, so as to set up sloughing in them also. In this way, according to Hertz, the diffuse form of gangrene may arise out of the circumscribed.

The only *physical signs* that can be said to belong to gangrene of the lung are such as serve to indicate the formation of a cavity in the organ at a spot where the tissue previously was either healthy or simply consolidated. But it can be only in very rare cases that such signs are to be definitely made out. They would include amphoric breathing, consonating or metallic râles, and loud bronchophony. When there is a possibility of the presence of phthisis, the mere detection of a cavity proves nothing as to the exact seat of the sloughing process, unless it is known that no vomica existed at the same spot before the fœtid expectoration began.

The general *symptoms* that accompany gangrene of the lung are often very severe, but it does not seem that they point to the presence of this affection so definitely as might be supposed from the statements made by most writers on the subject. It is said, for example, that the pulse is small, feeble, and very frequent, and that the pyrexia quickly passes into an adynamic form, with great prostration of the vital powers. That absorption of putrid matters into the blood from the lung should produce such results is, indeed, to be expected; but when the characteristic fœtor is absent it surely is not possible for anyone, from the intensity of the general symptoms alone, to suspect that sloughing of the lung is taking place. In ordinary pneumonia the patient often falls into a similar condition before

death; and the same may be said of many other diseases that sometimes lead to pulmonary gangrene. Nor does it appear that when the sloughing part is very limited in extent the nature of the morbid process is commonly to be inferred from the fact that the constitutional symptoms are disproportionately severe. The truth rather is that in such cases the patient's general condition often remains for several weeks much better than might have been anticipated. Hertz, indeed, speaks of anorexia and gastric disorder, and even diarrhœa, as being caused by the swallowing of some of the offensive material expectorated from the lung, but this would probably be difficult of proof.

Prognosis.—It is only when the gangrene is limited to a small part of the lung that recovery is possible. How minute a slough may cause fœtor is well shown by one of the cases of phthisis complicated with gangrene, which are recorded by Dr Walshe; in that instance the expectoration of a pea-like mass brought the fœtor to an end. Unfortunately he does not say for how long a time it had been present. When a case of gangrene is about to end favourably, the separation of the dead tissue is doubtless followed by the formation of a lining membrane to the cavity left by it, and perhaps this may ultimately shrink and become converted into a fibrous cicatrix.

Treatment.—It is an important point to diminish the fœtor as far as possible, and this applies also to cases of putrid bronchitis (p. 976). The most effectual means of attaining the object aimed at is by inhalations of oil of turpentine, carbolic acid, *oleum cadinum*, or eucalyptol. Turpentine inhalations were used by Skoda about thirty years ago. His plan was to pour a teaspoonful or two of oil of turpentine upon the surface of some boiling water, and to let the patient draw the vapour into the lungs. A better method is to use a Siegel's spray apparatus, so as to atomise a liquid containing from five to two parts of carbolic acid in 100 parts of water, or of a solution of common salt. The inhalations may be repeated two or three times a day. Care must be taken that there is not enough absorbed to set up headache or giddiness, or to give the urine a dark colour. The *oleum cadinum*, or the eucalyptol, may be directly inspired from a sponge placed in an "ori-nasal respirator." The effect of such inhalations is sometimes very striking in cases of fœtid bronchitis.

It is needless to say that the strength of the patient must be maintained by food, and that the administration of stimulants in large doses is sometimes necessary. Ammonia, camphor, ether, quinine, and the tincture of perchloride of iron may each in turn do good service. Oil of turpentine may also be administered by the mouth in doses of twenty or thirty drops, either beaten up with the yolk of an egg, or made into an emulsion with tragacanth or tincture of quillaia.

DISEASES OF THE PLEURA

"Side-stitches that shall pen thy breath up."
The Tempest.

PLEURISY—*Its anatomy—Its physical signs—Pleuritic effusion—Compressed and carnified lung—Dulness—Ægophony—Symptoms—Course and events—Empyema—Diagnosis—of dry pleurisy—of serous effusion—of empyema—Hydrothorax—Hæmothorax—Pleuritic effusion in children—Complications of pleurisy—Its ætiology and relation to tubercle—Prognosis—Treatment of pleurisy and of empyema—Paracentesis and its results.*

PNEUMOTHORAX—*Origin and pathology—Post-mortem characters—Physical signs—Diagnosis and symptoms—Prognosis—Treatment.*

PLEURISY or pleuritis* was mentioned by Hippocrates, and was described by other ancient Greek writers, who undoubtedly were referring to cases of the same disease to which we now apply that name. Nevertheless, it is only during the present century that its real nature has been known to be an inflammation of the two surfaces of the pleura. For, until percussion and auscultation were discovered, there was no possibility of drawing valid distinctions between pleurisy and pneumonia in clinical practice.

Anatomically, pleurisy closely resembles the inflammations of other serous membranes. The earliest morbid appearance is generally said to be injection of the smaller blood-vessels and perhaps the formation of ecchymoses. But, as a matter of fact, one often finds patches of recent lymph upon the surface of a lung without any reddening, when an inflammation of moderate intensity has set in shortly before death, from some other disease; and ecchymoses by themselves indicate, not an early stage of pleurisy, but rather pyæmic infection.

In many instances the effusion of lymph upon the opposed surfaces of the serous membrane occurs only at the very commencement of an attack of pleurisy, of which it forms a "plastic stage." If the morbid process stops here, the case is said to be one of *dry pleurisy*. When the inflammation afterwards subsides, the morbid material sometimes undergoes complete absorption, leaving the pleura in its natural condition, or slightly dull and opaque. But very often the two surfaces have in the meantime adhered together, and remain henceforth connected by separate bands or by a uniform layer formed of connective or fibrous tissue, which may have a free supply of blood-vessels. If the plastic stage, as is more often the case, is succeeded by exudation of serum the case becomes one of pleurisy with effusion.

Signs of dry pleurisy.—There is an auscultatory sign which, when it can be heard, is of itself almost conclusive as to the presence of lymph. This

* Πλευρῖτις sc. *νόσος*, *i. e.* the "side-complaint," the stitch in the ribs. The adjectival termination, *ιτις*, has been taken from this word and from *φρενῖτις* to denote inflammation, and has thus been used to form Peritonitis, Nephritis, Orchitis, and so on.

is the "friction-sound" or "pleuritic rub," due to the movement upon one another of the two roughened serous surfaces. Hippocrates described the pleura as "creaking like leather," and the comparison is often exactly applicable to the sound which is conveyed to the ear from the chest of a patient with pleurisy. Yet Laennec, though he must have often heard this sound, failed to understand its meaning, and left to Reynaud the credit of its right interpretation. Laennec supposed that it indicated emphysema, especially what he described as interlobular emphysema.*

It is difficult to describe in words the characters of this *pleuritic rub*; one must hear it to appreciate them. In its most typical form it consists of an irregular succession of short, harsh sounds, which give one exactly the impression of something catching or dragging against an obstruction and then slipping, but only to catch or drag once more. The patient himself is often conscious of a rough grating sensation each time he breathes; and sometimes one can *feel* the rub by placing one's hand over the affected part of the chest, *i. e.* the vibrations which affect the ear also affect the hand, just as a sonorous rhonchus or a loud cardiac bruit may be appreciated as a tactile vibration or thrill. Sometimes a rub accompanies both inspiration and expiration. Sometimes it is to be detected only at the end of a deep inspiration, when the lungs are just becoming expanded to the greatest possible degree. It may be heard within twelve hours of the commencement of the disease, and in cases of dry pleurisy it may persist for days or even weeks with but little alteration. But it much more often disappears after a short time, because fluid effusion is formed which keeps the two surfaces apart. If the inflammation is spreading, it may, after it has ceased to be audible at one spot, be discovered at another. A rub is not often present over a large area at once. The part of the chest at which one is most likely to hear it is in the axilla, outside the nipple, or in the back outside the angle of the scapula. The reason is not only that pleurisy more frequently affects the surface of the lower lobe than that of the upper, but also that the descent of the diaphragm causes an actual movement of the pulmonary upon the costal pleura, which is wanting elsewhere. Sometimes, however, a rub can be heard over the front of the chest as high as the clavicle.

Another variety of pleuritic rub is equally diagnostic. Instead of resembling the tearing of thick paper, it is, as Hippocrates heard it, just like the creaking of thick leather. The rhythm and its locality are the same. The "grating" rub is more like a râle; the creaking rub more like a rhonchus. Both when heard are very characteristic. But in some cases, a sound which is really due to pleural friction is so ill defined that one cannot distinguish it from moist or dry sounds in the bronchial tubes. This has long been taught at Guy's Hospital. In his well-known paper on the "Difficulties and Fallacies attending Physical Diagnosis," Addison cited a case of Dr Barlow's, in which lymph upon the adjacent surfaces of the liver and of the parietal peritoneum caused a "crepitus, which closely

* There are still some who think that this (or a precisely similar sound) may be due to emphysema (p. 971), or to miliary tuberculosis of the pleura. Again, Dr Walshe has expressed the opinion that a friction-sound may be heard when there is no lymph whatever, if the serous membrane is rough from "simple vascularity." But this conclusion appears to be hardly warranted by the case from which he drew it. Death occurred sixteen days after a rub had been heard, whereupon fluid was found in the pleural space, but no plastic exudation. May not lymph have existed previously, and have undergone disintegration, or absorption by leucocytes?

resembled a mucous rattle" ('Collected Works,' p. 87). It is hardly possible to obtain positive proof of the fact so far as regards the pleura itself, because, even if an autopsy shows lymph upon the serous membrane, one cannot be quite certain that an adjacent tube may not during life have contained fluid secretion.

The locality and the fact that coughing does not alter the sound, as well as the rhythm, usually help us to a correct diagnosis; a rub is very rarely heard near the apex, it is undisturbed by expectoration, and it is most often heard at the end of inspiration, instantly checked by the pain which is felt, never during expiration alone.

The only other physical signs of the plastic stage of pleurisy are a certain degree of impairment of movement of the affected side of the chest and a corresponding enfeeblement of the respiratory murmur.

Pleuritic effusion.—In most cases of pleurisy liquid is effused into the serous cavity, often in large quantity and with great rapidity. Two or three quarts are not uncommonly found, and Watson cites a case in which Crampton, of Dublin, drew off from the left pleura as much as fourteen imperial pints. The liquid is commonly translucent and of a yellowish colour, perhaps containing shreds and floating masses of fibrin. It has an alkaline reaction; according to Fräntzel, who writes on pleurisy in 'Ziemssen's Handbuch,' its specific gravity may vary within wide limits, from 1005 to 1030. In other cases it is more or less opaque, and on standing throws down a layer of greenish-yellow pus. Or it may be altogether purulent; in which case its reaction to test-paper is acid. This constitutes what is termed an *empyema.** As a rule, no doubt, the formation of pus in the pleural cavity is a gradual process, the liquid being at first serous or sero-purulent, and becoming more and more opaque as the leucocytes in it increase in numbers; in such cases both the parietal and the pulmonary surfaces may still remain coated with more or less thick layers of fibrin. But when the inflammation is from the first exceptionally violent—as, for instance, when it is set up by the entrance of putrid matter from without—primary suppuration may occur, and the pleura may remain as smooth and shining as the peritoneum under similar circumstances.

Purulent, as compared with serous pleuritic effusion, is far more common in children than in adults. It is the rule after admission of air to the pleura and in septic or traumatic cases, and also when secondary to variola, but is rare when secondary to rheumatic fever, to Bright's disease, or to cancer, and not common when of tubercular origin.

Sometimes a pleuritic effusion is of a deep brown or purple colour, from admixture of blood. This may be due to the fact that the patient has scurvy or purpura; or it may depend upon the presence of tubercles, or of cancer. According to Fräntzel, "a hæmorrhagic tubercular pleurisy" is less rare in persons advanced in years than in those who are younger; but the only example of it that has been lately met at Guy's Hospital was in a man aged thirty-five. The same writer hints at cases, comparable with hæmorrhagic pachymeningitis, in which after connective tissue has already been

* In England the meaning attached to the word *empyema* is, that of a collection of pus in the pleural cavity. But on the Continent, in spite of etymology, collections of serous fluid, and even of blood, are included under the same name (see Littré and Robin's 'Dictionary'). It has also been used as a synonym for thoracocentesis, so that when the pus escapes through an intercostal space, and has to be let out with the knife, an *empyema necessitatis* is said to arise.

formed as the result of a pleurisy, fresh inflammation is lighted up attended with extravasation of blood; but he does not say that he has actually met with such an instance. The author once removed five ounces of liquid of a dark brown colour from a patient who had pleurisy as a sequela of scarlet fever. He rapidly recovered.

Liquid pleuritic effusion usually gravitates into the most dependent part of the serous cavity, whatever may have been its starting-point. Thus, at the commencement of the disease, lymph may have covered the front and side of the lung, but when serum or sero-purulent fluid is poured out, it falls into the back and lower part of the chest if the patient is in bed, or it accumulates above the diaphragm if he is not recumbent. But this rule is liable to exceptions, when portions of the lung have previously become fixed to the chest-wall as the result of a former attack of pleurisy, or when adhesions rapidly develop early in the illness. Thus a considerable quantity of liquid may accumulate somewhere towards the upper part of the pleural sac, or between the lung and the pericardium, or between two lobes of the lung itself, without there being any in the usual position at the base. And even when the whole of the serous membrane is affected the seat of a serous or purulent effusion may be more or less irregularly circumscribed. Between 1873 and 1876 four instances of this occurred in our *post-mortem* room. In one case there was a broad adhesion to the lateral region of the chest and another to the diaphragm, so that the liquid filled the upper part of the pleural cavity while crepitant lung-tissue still existed in the lower part. In another case there was liquid at the base behind, and also above the root of the lung, with an intervening zone where the lung was firmly adherent. But the limitation of pleuritic effusion by adhesions is seldom so complete as to lead to the formation of two or more collections of fluid entirely cut off from one another; generally speaking, they communicate freely, as can be shown at an autopsy by passing a bent probe behind the bands of adhesion from space to space.

A necessary consequence of the presence of liquid in the pleural sac is that the lung becomes *compressed*, reduced in size, and at last emptied of its blood as well as of its air by effusion; but this is a very inadequate way of stating the case. Every physiologist will admit that the elasticity of the pulmonary tissue must lead to its receding as soon as the pressure in the pleural cavity exceeds that of the atmosphere, until it has become collapsed to at least the same extent as when air is admitted into the serous cavity after death. But, further, Lichtheim has proved by certain experiments, quoted above in the chapter on bronchitis (p. 959), that the elasticity of the lung does not become exhausted until the alveoli are completely emptied of all their gaseous contents. The reason why a lung is not rendered altogether airless when the pleural sac is laid open in the dead body seems to be mainly that the walls of the tubes presently fall together and offer a resistance to the further escape of air, which the elasticity of the pulmonary tissue is unable to overcome. And during life an additional force is in operation to empty the alveoli of air, namely, absorption by the blood-current circulating in their walls. A lung undergoes compression by pleural fluid until, when the cavity is full, it is absolutely free from air; and beyond this point it undoubtedly may become compressed until the blood is also driven out of its substance. It appears advisable to mark this distinction by separate names, and a lung which is bloodless as well as airless may be said to be *carnified*, whereas a lung which is merely airless may be

spoken of as *collapsed*. Both terms have long been in use, but not with the precise shades of meaning here assigned to them. A carnified lung has a very peculiar appearance; it has a slaty-grey tint, and is described as being mouse-coloured. Its cut surface is smooth, very firm, and dry, showing the flattened orifices of air-tubes and vessels closely packed together. Sometimes, if bronchitis has existed as a complication, the tubes contain pus; and if pneumonia or œdema should happen to be present, its characters are necessarily modified. The position occupied by a lung entirely compressed by liquid effusion is, as a rule, determined by its root; it becomes flattened against the mediastinum and backwards towards the spine, and if covered by a mass of false membranes its very presence may be altogether overlooked. Probably the notion of patients living on after having "entirely lost one lung," which is now applied to those who have phthisis, had its origin in autopsies made in cases of pleurisy of long standing. But when the organ has previously been firmly fixed by adhesions it cannot thus be driven inwards, and it may lie in the summit of the pleural space or be irregularly pushed to one side or even downwards. The most common deviation from the rule is doubtless when the upper part of the lung is affected with tubercular disease, and there is consequently a solid mass occupying a corresponding extent of the pleural cavity.

When pleural effusion is insufficient in quantity to empty the whole of the lung of air, and when therefore there is no *compression* of the entire organ, the effect on the pulmonary tissue is remarkable. One would anticipate that the elasticity of the organ would lead to a gradual and uniform shrinking of its substance, so that all parts would contain less air than before, without any part becoming completely airless. But the contrary is the fact. Dr Moxon long ago pointed out, when he was Demonstrator of Pathology at Guy's Hospital, that the presence of even a few ounces of liquid in any part of the pleural cavity causes a total collapse of the pulmonary tissue which ought to occupy that space. The writer has since repeatedly verified his observation; and Cohnheim, at p. 190 of vol. ii of his 'Vorlesungen,' makes a precisely similar statement. One often sees a small triangular area of completely collapsed lung at the posterior inferior corner of the organ, or a thin strip of it running up along its posterior margin. Nay, a mere enlargement of the heart, without there being any pleural effusion, may give rise to complete airlessness of the inner surface of the left lung; and distension of the abdomen, thrusting up the diaphragm, may cause a similar affection of the lower surface either on one side or on both. The explanation of these remarkable facts can hardly be understood except in connection with the mode of origin of collapse of the pulmonary tissue, which has been discussed in the chapter on bronchitis (*supra*, p. 959). It depends upon the general principle, that, whenever a part of the lung fails to be acted upon by the inspiratory forces, it becomes airless, notwithstanding that the tubes which serve it remain patent.

The production of local collapse of the lung-substance as the result of the effusion of moderate quantities of liquid into the pleural cavity has important clinical bearings. It accounts for a circumstance which has long attracted the attention of clinical physicians, namely, that temporary changes of posture on the part of the patient often fail to alter the position of the liquid within the chest, so far as one can tell by percussion. The statement

was once made by a great teacher of medicine, that, whereas in a chronic pleurisy the effusion could be made to gravitate to a different part of the pleural cavity, this could not be done in acute pleurisy, because it was held in the meshes of fibrinous exudation. But surely it is quite the exception to be able to elicit evidence of gravitation, even in chronic cases, although paracentesis is followed by a free flow of liquid through the trocar. Even if one can alter the level of dulness by making the patient sit up, the alteration does not amount to more than a finger's breadth or two. The contrast is very great between these results and the free gravitation in cases of hydro-pneumothorax. If a certain part of the lung is rendered altogether airless by pleuritic effusion, the fluid may (as it were) be *held up* in a fixed position in opposition to the force of gravity.

When the liquid is purulent, even when its quantity is not very large, one can often in thin patients make out that the intercostal spaces are less depressed and offer more resistance to the finger than on the sound side; and when the pleural cavity is full of effusion the ribs may be obviously wider apart, and the spaces between them may bulge or (occasionally) yield fluctuation. This, however, is but seldom observed in the case of serous effusion. On measurement one generally finds, if there is much liquid, that the affected side is enlarged, and sometimes the difference between the two halves of the chest is considerable. In determining this Dr Gee's cyrtometer is very useful. The play of the ribs in respiration is greatly impaired, much more so than during the plastic stage of pleurisy. Moreover, in consequence of the extent to which the sternum is carried forwards, the movement even of the unaffected side during breathing may be much diminished.

Displacement of organs.—Long before the lung has undergone complete compression, other adjacent structures feel the pressure of the effused fluid. The mediastinum is pushed over to the opposite side, the elasticity of the unaffected lung no doubt assisting to displace it. Thus if the pleurisy be on the right side, the apex-beat of the heart is felt and seen during life to be situated further to the left than usual; it may lie some distance outside the left nipple. This dislocation of the heart is, however, much more readily produced if the disease be on the left side. The apex-beat may then be in the epigastrium, between the sternum and the right nipple, or even to the right of the nipple, while in its normal position no trace of pulsation can be detected. Some observers have supposed that in such cases the heart swings over like a pendulum, and that its long axis is now directed downwards and to the right, so that the part which beats against the chest-wall is still the apex of the left ventricle. But nothing seen in the deadhouse supports this opinion. Probably the displacement of the heart is attended with little change in the inclination of its axis, and the impulse is given by some part of the right ventricle.

As above noted, displacement of the heart is much more obvious and extensive when the effusion is in the left pleura than when in the right.

The cardiac sounds have occasionally been found altered under these circumstances. Dr Hope heard a systolic murmur over the aorta, which disappeared when the pleuritic fluid underwent absorption. Dr Walshe met with a case in which each sound of the heart was more or less masked by a blowing murmur for several successive days, while effusion into the left pleural cavity was at its height. He thinks that the diastolic murmur

must certainly have depended upon displacement of the heart, producing tension of the aorta.

It is of some importance to know what amount of liquid is required in order to produce a perceptible cardiac displacement. Fräntzel says that effusion scarcely ever reaches up to the third rib without affecting the position of the apex-beat to a greater or less extent, and that even smaller amounts of liquid often suffice. He also observes that in children the heart is more easily thrown out of its place than in older persons. Another point mentioned by him is that when there have been previous adhesions between the pericardium and the left lung, pleurisy on the left side may cause the heart to be carried backwards away from the chest-wall, so that no impulse whatever can be felt or seen. But when the amount of effusion is moderate, the normal apex-beat may be absent without there being any discoverable impulse elsewhere, the reason probably being that the sternum conceals it. In all cases of this kind the stethoscope must be used with the object of determining the spot at which the cardiac sounds are heard loudest.

The diaphragm is pushed downwards whenever the amount of pleuritic effusion is at all considerable. The displacement of the liver or of the spleen may be recognised by percussion, or one or the other may be felt projecting below the ribs.*

Physical signs of effusion.—In the clinical recognition of pleuritic effusion we depend on physical examination of the patient. Several signs already mentioned must be carefully sought for; these are enlargement of the affected side, impairment of its movements, an altered state of the intercostal spaces, and displacement of the thoracic and abdominal organs. There remain the results of percussion and of auscultation; and of these two methods the former is by far the more valuable, as was long ago maintained by Piorry, in opposition to Laennec.

Dulness on *percussion* is, in fact, the main sign of pleuritic effusion. The percussion-sound becomes altered long before there is any evidence of pressure upon adjacent viscera. A circumscribed collection of serum and pus may of course cause dulness of any part of the chest, but when fluid lies free in the pleural space, the dulness is to be made out first at the base behind. One must not, however, suppose that small quantities of fluid ought always to be discovered by this means. Wintrich long ago declared that eight or ten ounces could scarcely be detected with certainty, and a considerably larger amount may sometimes escape recognition.

Much depends upon the habitual posture of the patient. If he is sitting upright in bed, the diaphragmatic, rather than the posterior, surface of the lung becomes compressed, and the percussion-sound may at first be scarcely altered. If he is lying down, the fluid is more or less spread out over the back. When the percussion-sound down to the very bottom of the lung is perfectly resonant, one may feel sure that effusion, if present at all, is in such small quantity as to be clinically unimportant.

The dulness caused by fluid in the pleura differs from that produced by consolidation of the lung in being more absolute, and in the greater sense of resistance which it conveys to the finger in percussion.

* According to Fräntzel, when the distension of the pleura is extreme, it is sometimes possible to detect an elongated, tense, fluctuating swelling, which protrudes below the costal cartilages, and which is nothing else than the front part of the diaphragm.

If the effusion is large, the whole of the back and side of the chest are devoid of resonance. It is remarkable in such cases that over the front of the chest—below the clavicle, and downwards nearly to the nipple—the percussion-note is subtympanitic. Skoda first pointed out this fact, which remains associated with his name. Its explanation is still doubtful. German writers are content to ascribe it to "relaxation of the pulmonary tissue;" Dr Walshe thinks that it depends upon the presence of air in the minute tubes of the carnified lung, so that a condition essential to its production is that these tubes should not have undergone compression as well as the lung-substance. The explanation suggested by Dr Bristowe is the diminution of the vibrating area formed by the thoracic walls (p. 929).

Auscultation is of less assistance than percussion in the detection of pleuritic effusion. In many cases one finds that the breath-sounds on the affected side of the chest are enfeebled, indistinct, or altogether wanting. But, not infrequently, tubular breathing is audible over part of the compressed lung, and in some exceptional cases this can be heard so extensively that one might suppose the air to be entering freely everywhere.

The same curious uncertainty applies to *auscultation of the voice*. As a rule, it is conveyed to the ear more feebly than on the healthy side of the chest; but sometimes there is bronchophony, and this may occasionally be extremely loud. There is, however, one modification of vocal resonance which, when present, is very characteristic of pleural effusion. It was discovered by Laennec, who called it *ægophony*, from its resemblance to the bleating of a goat. He also compared it to the artificial voice of "Punch" in the street (p. 940, *note*). Both these comparisons are excellent, and the sign is readily caught even by an untrained ear. It may be described as high-pitched, tremulous bronchophony, with a nasal twang. With regard to the frequency of this sign, widely opposed statements have been made by writers in consequence of their differing as to the definition of the term. Almost all the best observers are agreed that what may be called *pure* ægophony is rare, and seldom lasts more than a few days. But between it and bronchophony there are all degrees of transition; and if one is to speak of the voice as ægophonic in every case in which it reaches the surface of the chest with more or less of a twang, there are very few instances of pleuritic effusion in which this change in its character is altogether absent. In one particular region ægophony is observed far more frequently than elsewhere, namely, about the inferior angle of the scapula, and round towards the axilla. But sometimes it is heard in front, near the nipple, or even close to the clavicle.

Its production is believed to depend upon the presence of a rather thin layer of liquid between the lung and the parietes (cf. p. 940). Consequently it generally disappears as the effusion increases, unless there are adhesions, which keep the lung fixed at a certain distance from the surface of the chest. But in some rare cases, not explained by the existence of adhesions, ægophony may persist in spite of abundant accumulation of fluid; and, according to some experienced auscultators, it is sometimes present when there is no fluid effusion at all. Dr Walshe points out that one source of fallacy lies in the possibility of overlooking the fact that the ordinary voice of the patient is shrill and tremulous, as it so often is in women of advanced age. Again, bronchophony may acquire a nasal twang if the nostrils are closed, just as the ordinary voice does.

With pleural effusion the *tactile fremitus* felt when the patient speaks is

diminished or absent, a sign which is often of value in distinguishing this condition from solidification of the lung. Nevertheless, in exceptional cases vocal fremitus is palpable.

General symptoms.—The symptoms of pleurisy vary widely in severity. They are sometimes so marked as to suggest the nature of the case at once; they are sometimes almost, if not quite, absent.

Foremost among them is *pain* in the side, the *point de côté* of French writers. This is often very violent, and of a sharp tearing or cutting or stabbing character. It is increased both by movement and by pressure, but more by a deep breath, by a laugh, a cough, or a hiccup. The patient, therefore, breathes in a shallow, jerky manner. His cough, if he coughs at all, is short and half suppressed, and he abstains as much as possible from laughing or sneezing. He lies during the early part of his illness on his back and on the unaffected side, and he shrinks from percussion. The severe pain contrasts strikingly with its absence in bronchitis and pneumonia. It is comparable to that of the inflamed peritoneum, and, like it, is excited or greatly increased by pressure. Hence the pain on breathing and the relief obtained by restraining the movements of the chest by bandages. Cruveilhier rightly accounted for the fact that its seat is so often limited to the lower part of the chest—about the nipple or between the fifth and eighth ribs—since here there is so much more movement of the visceral upon the costal pleura than higher up. But the pain is sometimes felt in the shoulder, in the armpit, or beneath the clavicle. In exceptional cases it is referred to the terminal branches of the intercostal nerves; to the hypochondrium, so as to lead to a mistaken diagnosis of hepatitis; to the loins, so that the case has been called one of lumbago; or to the neighbourhood of the umbilicus, so that peritonitis has been suspected.*

In some cases pain is altogether absent, and thus a large quantity of effusion may accumulate in the pleural cavity without its presence being thought of. Fräntzel says that this is apt to occur in children, in very old people, and in lunatics. It often happens that pain subsides or disappears towards the end of the first week, or even after two or three days.†

Next to pain, *dyspnœa* is the most striking symptom of pleurisy. The breathing is short and jerking, but it is also increased in frequency, especially when the patient exerts himself, as in lifting anything or in going upstairs. Sometimes the scaleni and the other muscles of forced inspiration are brought into action; and the nostrils dilate each time air is drawn into the chest. As effusion accumulates, the patient begins to find that he can lie over towards the affected side more comfortably than in any other position, because the weight of the fluid is then removed from the mediastinum. He often has orthopnœa, for the diaphragm works more freely while the upright posture is maintained. Sometimes there is lividity of the cheeks and lips.

The dyspnœa of pleurisy is generally more marked in robust plethoric patients than in those who are anæmic and wasted. As Andral long ago pointed out, it sometimes happens that pleurisy scarcely interferes with the

* I have myself had a patient whose sole complaint was of pain in the crista ilii; I feel sure that if it had not happened that a short while before my attention had been specially directed to this question, I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub.—C. H. F.

† Laennec, and afterwards Gerhardt, declared that the pain of pleurisy was sometimes seated upon the opposite side of the chest to that which was inflamed, but this would require more evidence than that of authority for us to accept it.

patient's comfort. He had a patient who went on with his work as a carter, in spite of an enormous effusion into his pleura; and Watson speaks of a butcher, who in the same condition was convinced that he was well, and fit to leave the hospital.

Cough is seldom entirely absent in pleurisy; and there appears to be no doubt that it may occur independently of any affection of the lung or of the bronchial tubes. Sometimes it can be excited by percussion or pressure over the painful intercostal spaces, or by changes of posture. During the operation of *paracentesis thoracis* it may be produced by movements of the trocar. In his experiments on animals Kohts found that it was caused by irritation of the parietal, but not of the pulmonary, pleura. The cough of uncomplicated pleurisy is dry, *i. e.* unattended with expectoration.

The *pyrexia* of pleurisy is generally moderate. The disease, when uncomplicated, seldom sets in with a violent rigor. But slight chills returning day after day are observed in many cases, especially when the patient remains out of bed during the day. The temperature commonly ranges at about 101° or 102°, but in the most severe forms of the disease it may reach 104°, or even higher in children. In persons suffering from cancer, or from chronic renal disease, there may be no pyrexia at all. The pulse is accelerated. Fräntzel insists upon the importance of watching it with care; for as effusion goes on, although the temperature may fall, the pulse not only becomes more rapid, but its volume and tension diminish, in consequence of the obstruction to the flow of blood through the pulmonary vessels. This affords a valuable indication of the degree of danger to the patient's life; and during paracentesis the physician may be able to feel the pulse becoming fuller, and at the same time slower, under his finger, showing the immediate relief given by the removal of the fluid.

Side affected.—It has been thought that pleuritic effusion is much more common on the left side than on the right.* Dr Eddison, of Leeds, in 40 purulent cases found 20 right to 20 left; Dr Richards, of Birmingham, 3 right to 7 left; Dr Griffith, of Leeds, 19 right to 26 left; Mr Godlee, 22 right to 28 left. Of 44 cases of empyema under the writer's care, 16 were right and 28 left. Adding these numbers together, we find that of 189 cases of empyema, 80 were right and 109 left. Cases of double empyema have been recorded in children. Double serous or plastic pleurisy is not uncommon.

Course and event.—The course of pleurisy varies widely in different cases. In the most severe form, which is very rare, but of which Fräntzel says that he has seen three examples, the patient quickly falls into a typhoid state, with delirium, stupor, and a dry fissured tongue: the dyspnœa and lividity increase, so as to threaten his life by the end of the first week; and though paracentesis be performed and repeated, the effusion collects again so rapidly that the fatal issue is scarcely retarded.

Even when the disease seems to be attended with no alarming symptoms one must never regard it as free from danger, if the amount of effusion is large. Death sometimes occurs quite suddenly and unexpectedly. In 1874 this happened to a patient in Guy's Hospital with double pleurisy, who a short time before had been seen quietly asleep. For some days previously this patient had had much dyspnœa, and it seems not unlikely that the im-

* "That pleurisies are *only* on the left side is a popular tenet not less absurd than dangerous" (Sir Thos. Browne, 'Pseudodoxia epidemica,' bk. iii, chap. 3).

mediate cause of death was exhaustion of the respiratory centre. But in another case, which ended fatally after an hour's extreme distress of breathing on the evening after admission, it was observed that the pulse ceased before the respiration. Here the effusion was on the right side; but there was œdema of the left lung, which no doubt helped to kill the patient.

For the occurrence of fatal syncope, when the left pleura is the one affected, a special explanation has been suggested by Bartels, of Kiel, in the 'Deutsches Archiv' for 1868. It depends upon the anatomical fact (which has been verified after death on several occasions by him and by Frântzel) that when the heart is pushed far over to the right the mouth of the inferior vena cava becomes bent almost at a right angle, just above the quadrilateral aperture in the diaphragm, the wall of the auricle forming a fold which covers a large part of the aperture. This is supposed to interfere with the flow of blood to the heart, especially if the diaphragm is suddenly curved upwards in a fit of coughing, or if a sudden muscular effort is made.*

But pleurisy is not often attended with such risks. The inflammation need not go beyond the exudation of lymph, and after a time it may subside, leaving adhesions which fix the lung to the chest-wall for the rest of life. Whether dry pleurisy necessarily ends thus in adhesion of the affected part, or whether it may not sometimes pass off without permanent traces, is a difficult question. What is well known to every pathologist is the fact that an adherent pleura is often found when there has been no history of any affection of the chest. Dr Gee gives a tracing, made with the cyrtometer, which shows that in a child the chest may be markedly contracted on one side, as the result of a universal closure of the pleural space, without there having been any symptoms to suggest the presence of such a morbid condition. There is no clear evidence that the adhesions left by a dry pleurisy at all affect the health.

The *duration* of an attack of dry pleurisy is sometimes exceedingly brief, often not more than two or three days.†

When pleurisy gives rise to effusion and the amount of liquid remains small, one can express a confident opinion that absorption will take place in time, and that the compressed pulmonary tissue will expand and resume its functions. And even if the quantity should be very large, there is always a possibility that the patient's ultimate recovery may be complete.

Percussion usually affords the earliest indication of the subsidence of pleural effusion. The dulness becomes less extensive and less absolute, not only in front, but also over the upper part of the lung behind. A little later, the displaced organs return to their proper situations, and the side may fall back to its previous dimensions. A feeble vesicular murmur may then be heard where none had been discoverable before. But with regard to this, there is a source of fallacy which must be borne in mind. At a certain stage of pleurisy, even while the affected lung still remains completely flattened and airless (as subsequently appears from an autopsy), the inspiratory sound from the opposite lung is very apt to be carried across

* Trousseau had previously attributed the occurrence of sudden death in cases of pleuritic effusion to "twisting of the aorta and large vessels," as a result of displacement of the heart; but he does not seem to have had the inferior vena cava in view.

† I was once asked to visit a student who had been seized the same day with violent pain in the side, and who told me that he knew he had pleurisy, because he had had the disease before. I heard a very well-marked rub on auscultation, and told him that I should come to see him on the following day. But when I came he assured me that he was well again; and on listening I could detect nothing abnormal.—C. H. F.

the spine in such a way as to suggest that air enters both sides of the chest. It seems that the pleura when overfilled may transmit sounds almost as well as a solid lung (cf. p. 1025, of bronchophony).

Another physical sign which commonly attends the absorption period of pleural effusion is what is termed a "redux rub." This exactly resembles the friction-sound of the earliest stage of the disease, differing (if at all) only in being still louder and in being heard over a more extensive area. It often remains audible for several days or even weeks together, and may be accompanied by a return of pain in the side, without fresh inflammation.

Even when the attack has so far subsided that the patient feels well, and perhaps resumes his occupation, it often happens that the side still remains more or less dull on percussion, and that the breath-sounds over the affected part are much feebler than natural.

Empyema.—If the pleuritic effusion is purulent, it very rarely undergoes absorption, but perforates the pleura, and thus makes its way out of the body. This never happens with a serous effusion.

Sometimes the empyema escapes *through the bronchial tubes.* If this should occur while the patient is asleep, or if he should be so exceedingly feeble as not to be able to expectorate, he may be instantly suffocated. But surprisingly large quantities of pus are sometimes ejected, with much less distress than might have been anticipated. If the opening leads directly into a large tube, air passes into the pleural cavity to take the place of the liquid, and a "pyo-pneumothorax" is established, which will be discussed further on (p. 1043). But in many cases of empyema discharging through the lung no pneumothorax follows. This appeared so remarkable to some of the older physicians, that they supposed that pus was capable of undergoing absorption from the pleura, and of being afterwards excreted from the bronchial mucous membrane. The true explanation was given by Traube in 1872 ('Ges. Abhandlungen,' vol. iii, p. 44). If the pleura alone is eaten through, the alveolar texture of the compressed lung may allow pus to be forced through it by violent coughing, while it yet fails to afford a passage to air in the opposite direction, especially as there is little or no movement of that side of the chest during inspiration.

Such cases often end in recovery, as Hippocrates knew, when he wrote: "Those in whom a pleurisy ends in suppuration, may be cured if they bring up the matter within forty days from the rupture into the pleura" ('Aph.,' v, 15).

In other cases, but far less often, an empyema makes its way outwards *through the parietal pleura.* An intercostal space is then usually perforated; and a soft elastic swelling, of greater or less size, forms beneath the skin, which ultimately becomes reddened, ulcerates through, and allows an enormous quantity of pus to escape. At the present day one seldom has an opportunity of observing this result of pleurisy, because scarcely any practitioner now fails to diagnose a large pleuritic effusion, and to treat it surgically. The point at which perforation is most apt to occur has (in the 'Lancet' for 1882) been defined by Mr Marshall to be in the fifth space, below the nipple; here there is a weak spot in the chest-wall, covered only by the internal intercostal muscle and a thin layer belonging to the great pectoral and the external oblique muscles. But the orifice may be elsewhere, and is sometimes as high as the second space. Or, again, the diaphragm may be perforated by an empyema. In 1865 a man died in Guy's Hospital of an empyema, which had been tapped eleven days previously. A hole,

which had a diameter of a quarter of an inch, was found in the fleshy substance of the diaphragm, and below it was a large circumscribed abscess, which had not opened yet into the peritoneum.

Again, the pus may make its way backwards and point *in the loin*. In 1858 a boy nine years old was in the hospital for pleurisy, and was discharged convalescent. Afterwards he came back with a pulsating swelling in the left lumbar region, which proved to be an abscess and was opened. Two months later the boy died of tubercular meningitis, and an autopsy showed that the left lung was still contracted, and that a sinuous channel, six inches long, extended down from the pleural space behind the diaphragm to the external opening. In one recorded case the pus from an empyema burrowed until it actually reached the popliteal space.

Except when the pus escapes through the pulmonary tissue, the spontaneous discharge of an empyema is almost always followed by a protracted illness, and often ends in the death of the patient. Entrance of air into the serous space renders the contained fluids putrid; and this leads to irritative fever, and to more or less rapid emaciation and exhaustion. If not, the pleural fistula may go on discharging for five, ten, fifteen years, or even longer, until lardaceous changes develop themselves in the abdominal viscera, and cause death by renal dropsy. In one patient under the writer's care, empyema had lasted fourteen years, and when she died at about seventy-two there was no albuminuria nor sign of enlargement of the liver or spleen.

The only chance of recovery in cases of this kind seems to be that the whole cavity should be gradually obliterated by the abundant formation of granulation tissue, and by the contraction of the dense fibrous material which becomes developed from it. This indurated substance sometimes reaches the thickness of an inch. At the same time all the structures which surround the pleura become dragged inwards, so as to reduce it within the smallest possible limits. The ribs fall in and may almost come into contact with one another; they remain motionless during inspiration, or (as was once observed by Dr Gee) their anterior parts may actually recede and move backwards each time that the healthy half of the chest expands and draws the sternum forwards. The dorsal spine becomes curved, so as to present a concavity towards the affected side. The shoulder sinks, the diaphragm is dragged upwards with the abdominal viscera; the mediastinal structures are pulled over, and the heart is often brought so widely into contact with the ribs that its impulse can be seen and felt over a far more extensive area than under normal circumstances.

Diagnosis.—As a rule, the recognition of pleurisy and pleural effusion is easy, being based directly upon the characteristic physical signs. But many practitioners are too ready to set down to this disease cases in which there is no symptom except a *pain in the side*. Hospital practice teaches us to distrust the statements of patients when they tell us that they have been under treatment for pleurisy. It may have been neuralgia or intercostal rheumatism, *i. e.* myalgia; sometimes an eruption of *shingles* has escaped notice, from not having been looked for. Costal *periostitis* and abscess of the chest-wall are other affections the possible presence of which must not be forgotten. The only case in which it is allowable to diagnose pleurisy without positive evidence from percussion or auscultation is when violent pain in the lower part of the chest is increased by each breath and accom-

panied by pyrexia. We may then reasonably suppose that there is inflammation of the diaphragmatic and corresponding pulmonary pleura.

Even when we think we hear a *pleuritic rub*, there is need of caution. Dr Gairdner has recorded an instance in which a sound which he describes as having a shuffling character, attended with a tactile sensation as of a jerking movement, produced by something rubbing up and down against the walls of the chest, proved to be due to emphysema of the lung. And Guttmann cites a case of Jurgensen's, in which a similar effect was produced by tubercles of the pulmonary pleura. It is said that the crepitus of a broken rib has been mistaken for a pleuritic rub.

Frequently what has been called a soft pleuritic rub turns out to be a mucous râle. Another mistake to beware of is that of attributing to pericarditis a friction-sound which is really pleuritic.

Pleuritic effusion has sometimes been diagnosed when the disease (if on the right side) has been a *hydatid* in the liver, or a hepatic *abscess*, or hypophrenic abscess, or when (if on the left side) it has been an abscess between the liver and the diaphragm connected with the spleen. Again, it is remarkable that all the examples of very large chronic *pericardial effusion* which have occurred at Guy's Hospital have been set down to pleurisy. This occurred to the writer in 1889, there being moderate effusion in the left pleura combined with the presence of forty-two ounces of serum in the pericardium in a case of Bright's disease. The accumulation was very gradual and the symptoms remarkably slight. One ought to be on one's guard, and by carefully mapping out the area of percussion-dulness and marking exactly how far it extends in front, at the side, and behind, the figure produced would probably decide the point. No doubt the disease might still be a circumscribed empyema; but at least one would be saved from imagining that the fluid lay free in the pleural cavity, and by paracentesis no harm would probably be done.

When there is a very large effusion of pus into the left side of the chest, pulsation synchronous with the heart can sometimes be felt in the intercostal spaces near the nipple or above it and towards the clavicle, so that the presence of an *aneurysm* may be suspected. A case of this kind was recorded two centuries and a half ago by Baillon; its real nature was cleared up by the bursting of the swelling with discharge of pus. In our own time Dr Walshe and Dr Macdonnell, of Montreal ('Dublin Journ. Med. Sci.,' 1844), have studied "pulsating empyema;" Comby in France ('Arch. Gén.,' 1883), Kepler in Germany ('Arch. f. kl. Med.,' 1887), and Osler in America ('Trans. Assoc. Amer. Physicians,' 1888, p. 330) have collected about forty cases. Its pathology is not clear, but seems to depend on extreme tension, with weakening of the intercostal muscles. Pulsating empyema occurs almost always on the left side.

Again, the physical signs may point to the presence of a large pleuritic effusion occupying the lower and back part of the chest on one side, and yet they may turn out to be due to solid lung. Traube has related a case in which he imagined that there was, besides hepatisation of the left lung, a large pleuritic effusion, but the autopsy showed that the serous cavity had been closed by former adhesions; the great diminution of tactile vibration in this instance was attributed to plugging of the smaller bronchial tubes by lymph. Most English observers follow Laennec in believing that the detection of ægophony is conclusive evidence that there is at least some fluid effusion into the pleura; but Fräntzel declares, as the result of careful

observation directed to this question for some years, that this rule is not invariable, as had, indeed, long ago been asserted by Skoda.

In chronic cases a more serious error may be committed; that of mistaking for pleuritic effusion a mass of *malignant growth*. Every physician of experience must either have seen this mistake made by others or have made it himself. One should therefore never give an opinion without having thought of such a possibility; the points to be especially noticed are whether the area of dulness corresponds in shape with that caused by a distended pleura, and whether tactile vibration is or is not still to be felt in certain positions. It must be borne in mind that a new growth situated in the mediastinum or in the lung is often accompanied by effusion into the pleura, so that a strictly differential diagnosis may after all be less accurate than a more doubtful opinion.

Fräntzel relates a converse case, in which a large hæmorrhagic effusion was for a time supposed to be a solid tumour.

Even when the presence of liquid in the pleural cavity is positively and correctly diagnosed, it is not always due to pleurisy.

Passive dropsy of the serous cavity, or *hydrothorax*, may give rise to precisely similar physical signs, except that, being probably never altogether unilateral, it is not likely to displace the heart. This condition is most common in cardiac disease or in extreme anæmia, but often complicates inflammatory effusion in cases of Bright's disease.

Again, pure blood may fill the pleura, constituting *hæmatothorax*. Apart from surgical injuries, the chief cause of such an affection is the rupture of an aneurysm of the aorta. Watson mentions a case in which caries of a rib led to ulceration of the intercostal artery and to distension of that side of the chest with blood, a large part of which was clotted in concentric layers. The admixture of a certain amount of blood with serum in a pleural effusion is almost certain evidence that the primary disease is either tubercular or malignant.

Diagnosis of empyema is often difficult. One of the chief indications of the formation of pus is the continuance of high evening temperatures after the first two or three weeks; the pyrexia in many instances assumes a regularly hectic type. Œdema of the subcutaneous tissue of the affected side of the chest has long been mentioned as a sign of empyema, but it is often absent where suppuration is going on; and Fräntzel cites a case observed by Traube, in which it was present, but in which a fibrino-serous liquid was withdrawn by paracentesis. The best way of detecting the œdema is to pinch up a fold of skin, and to compare its thickness with that of a similar fold on the opposite half of the body. Fräntzel and Peter each found in certain cases of pleurisy that the temperature of the skin was persistently higher by about a degree Fahr. on the diseased than upon the healthy side of the chest, and in almost every instance the effusion proved to be purulent. As practical rules, we may bear in mind that empyema is far more common in a child than in an adult, that rheumatic pleurisy never is purulent and renal pleurisy rarely, but that tubercular is often so, while traumatic commonly, and pyæmic pleurisy always, causes an empyema. Among adults, empyema is more common in men than in women.

In children, the difficulties of diagnosis of pleural effusion are greater than in adults, especially between it and broncho-pneumonia with extensive collapse; and the temperature affords little help in distinguishing a purulent from a serous effusion, for it is so readily raised in a child. Dr Thomas

Barlow and Mr Parker (in a paper read at the British Medical Association in Manchester, 1877) quote a case of serous pleurisy with a temperature of 103·4°, and of empyema with the highest evening temperature 101·5°. They look upon anæmia and clubbing of the fingers as the best signs of the purulent character of an effusion, but depend on the practical decision of the hypodermic syringe, which has now replaced the grooved needle.

The frequency of empyema in children is illustrated by the following figures quoted in the above paper: out of 44 consecutive cases of pleurisy in the Great Ormond Street Hospital, 27 were purulent, and in another series of 16 cases 14 were purulent.

Complications.—These are rare; pleurisy does not lead to pneumonia, nor directly to phthisis, though it is often secondary to each. It often accompanies bronchitis as the result of a chill, with or without evidence of broncho-pneumonia.

Empyema is generally attended with more or less exudation into the subserous connective tissue. In a case which occurred at Guy's Hospital, in 1872, the surface of the lung was covered with reticulated lines, due to the presence of pus in the lymph-channels beneath the visceral layer of the pleura. In another case, in 1869, pus was found outside the parietal layer of the pleura, infiltrating the intercostal muscles; there was also an abscess as large as a walnut in the mediastinum. In a third case, in 1873, the mediastinal tissues were three quarters of an inch thick from infiltration with puriform lymph. All three were examples of double pleurisy with pericarditis.

The secondary pleurisy of phthisis seems never to extend to the pericardium. But the more intense and violent forms of the disease are exceedingly apt to do so. Moreover, there are cases in which both pleuræ, the pericardium, and the peritoneum become simultaneously inflamed; at least it cannot be determined that one of them was affected first (cf. p. 60). As a rule such cases present very acute symptoms and run a rapid course, but this is not invariable.*

Whenever the two pleuræ are attacked with inflammation simultaneously or in succession, one ought to look for some underlying condition, such as Bright's disease or tuberculosis.

Ætiology.—(*a*) *Of "idiopathic" pleurisy.*—Foremost among the causes of pleurisy is *cold*. This has been so commonly set down as giving rise to all forms of internal inflammation, and often with so little reason, that one cannot be surprised that many physicians are reluctant to recognise its ope-

* In 1876 a woman, aged twenty-three, was admitted into Guy's Hospital with what was supposed to be enteric fever. After a fortnight fluid effusion was detected in the left pleura, and forty-two ounces were drawn off by the aspirator. Her febrile symptoms continued; she became emaciated and died. Towards the last it was naturally thought that she was suffering from tubercular disease. However, on making the autopsy, we could discover no tubercles nor any primary visceral lesion. Beside some pleuritic effusion on the left side there was lymph over the whole of the right pleura; the pericardium was adherent by a recent plastic exudation; the liver and the spleen were fixed to the diaphragm; and the lower part of the abdominal cavity contained some purulent fluid. It may be that the disease was of rheumatic origin, for the patient had a painful affection of her joints about two months before; but there was no evidence of endocarditis, present or past. The case is of interest, not only pathologically, but also on account of its obscurity during life. For the thoracic serous cavities and the upper part of the peritoneal space have been repeatedly found all closed by old adhesions, when there has been no history of any chest affection; and it would now seem that such a result may arise from an illness which clinically might be taken for fever.—C. H. F.

ration (see a lecture by Dr Ransom, of Nottingham). But the clinical evidence of the direct dependence of pleurisy upon cold cannot be explained away. Fräntzel cites, for example, cases of persons who have been attacked after having exposed one side of the body to a draught in changing their clothes while heated, or after having got one side wet through in a driving rain. Other cases have immediately resulted from sitting near an open window or a badly fitting door, especially during convalescence from some acute illness.

Injuries to the chest may give rise to pleurisy. Not only does this occur when the ribs have been broken, but sometimes when there is no evidence of damage to the walls of the chest.

(*b*) *Of secondary pleurisy.*—Certain general diseases are very apt to be attended by pleurisy as a complication. This is the case with acute *rheumatism;* but rheumatic inflammation of the pleura occurs only during the fever, not independently of it, as with pericarditis or endocarditis. It is very difficult to prove the rheumatic origin of the serous inflammation in any other cases, and it is much better not to assume it.* Scarlet fever must also be mentioned. Fräntzel lays down the rule that enteric fever never becomes complicated with pleurisy during its early stage, while the morning temperature is below 102°; and it is not frequent later.

Among chronic affections none is so commonly accompanied by pleurisy as *Bright's disease;* so that the urine should always be carefully and repeatedly tested for albumen.

Secondary pleurisy is often due to the direct extension of inflammation from some adjacent structure. To the pathologist there is probably no condition which is so familiar as this; but the affection is in many cases found to be quite recent and early at the time of death, so that it has no clinical significance. Among the less obvious starting-points for a severe or even fatal pleurisy may be mentioned abscesses in the armpit, operations upon the breast, suppuration of the cervical connective tissue after tracheotomy, caries of the ribs, mediastinal abscess, cancer of the œsophagus, and caries of the dorsal vertebræ. Or its origin may be below the diaphragm, the connecting lymph-channels discovered by Recklinghausen probably conveying the inflammatory process from one serous cavity to another. Thus pleurisy which was the immediate cause of death has been found to be due to extension from a peritonitis of puerperal origin, or following ovariotomy, or arising from disease of the rectum. Again, a very acute inflammation of the pleura, with fœtor of the pus, has started from the upper end of a psoas abscess, or from a localised abscess behind the stomach due to a perforating gastric ulcer. Mediastinal growths without inflammation frequently produce pleuritic effusion; so also do malignant tumours of the walls of the chest or of the mamma.

Dry pleurisy occurs in every case of acute *pneumonia*, and we must remember that what seems to be an uncomplicated and simple attack of pleurisy may really be dependent upon extensive pneumonia, of which there is sometimes little or no clinical evidence. When pleural effusion has once taken place it may be impossible to discover by physical signs the presence of hepatisation of the corresponding part of the lung. It often happens that the characters of the sputa afford the only clue to the real nature of the case, or a correct diagnosis may depend entirely upon one's having seen the patient at an earlier period, before the fluid was poured out into the serous cavity. Again, it is not improbable that a very limited patch of pneumonia,

* By "rheumatic pleurisy" many writers only mean pleurisy due to catching cold—a misleading use of the term.

involving the surface of the lung, may sometimes be the starting-point of a diffused and severe pleurisy.

In *pyæmia* (especially when resulting from thrombosis of the cerebral sinuses, itself consequent upon disease of the temporal bone) pleurisy is sometimes the most conspicuous feature of the case, and may be mistaken for the primary disease, and the cause of all the patient's symptoms.

The relation of pleurisy to *phthisis* is of the greatest importance, on account of its bearing on prognosis. One is frequently seeing patients who, having favourably passed through an attack of pleurisy, are shortly afterwards seized with hæmoptysis, or develop signs of tubercular disease of the lungs. Sometimes, no doubt, the presence of such disease can be detected, even while the serous inflammation is in progress, if one is careful to examine the upper lobes thoroughly. But in many cases all the clinical evidence points to the conclusion that the pulmonary affection has been of later development. Those who adopt Buhl's infective theory of tubercle might maintain that it is really secondary, and due to the absorption of caseous matter into the blood. But the primary pleuritic inflammation is almost always of a serous, not a purulent character, and it is far more probable that it is due to a few pulmonary tubercles on the surface of the lung setting up irritation and effusion.*

It is a curious question whether pleuritic effusion, while it continues to compress one of the lungs, favours a fresh development or a further growth of tubercles in that organ, or whether it may not rather be adverse to such an occurrence, even though it may increase the susceptibility of the other lung, which has to perform extra work. The following six cases bear on this question. In one there were no tubercles except in the opposite lung; in another the tubercles were much less numerous on the side of the pleurisy; in two others it was observed that on that side they were all of old date and inactive. On the other hand, there was one case in which they were more abundant in the compressed lung than in the opposite one; and once a lung entirely airless was full of tubercles (some grey and others caseous) in its lower lobe, where their presence, the apex being healthy, is an occurrence so exceptional that one could hardly doubt that the pleurisy had determined their formation.

On the whole, it is important to remember that, excluding traumatic cases and direct exposure to cold, the majority of cases of pleuritic effusion in young adults, if not occurring in the course of Rheumatic Fever, are tubercular, and that the majority of cases in elderly persons are renal or secondary to malignant disease.

Prognosis.—This depends chiefly upon the answer to the question as to the origin of the pleurisy set forth in the preceding paragraphs.

Acute idiopathic pleurisy from exposure to cold in a healthy subject is scarcely ever fatal. With moderate antiphlogistic treatment it is speedily relieved, and cured without leaving sequelæ behind. Even when neglected, and when one side of the chest has been allowed to fill with serum, it is remarkable how well such cases do after aspiration.

An effusion which half fills the pleura without pain or fever, and is only discovered by physical examination in search of a cause for the patient's dyspnœa,—this should make one suspect tubercle or Bright's disease. But when these can both be negatived, the prognosis still is good.

* See on this subject a paper by Dr Barrs, of Leeds ('Brit. Med. Journ.,' 1890, i, 1058).

To leave a patient, however, with one side of the chest full of fluid is never safe. He may die suddenly from syncope, or œdema of the other lung may suffocate him before help can be brought.

The pleurisy which forms part of acute pneumonia adds to the pain, but not to the danger of the primary disease; and the same is true when pleurisy is secondary to lymphoma, or other mediastinal growths. In the latter case it is usually latent, and often as much dropsical as inflammatory.

Tubercular pleurisy does not usually add to the dangers of phthisis, and often appears to retard its progress by limiting the amount of blood as well as of air in the diseased lung, for caseous inflammation does not flourish in anæmic and airless pulmonary tissues. As we have stated above, the effect of carnification on miliary tuberculosis is uncertain. On the other hand, pleurisy secondary to Bright's disease, whether dry or combined with hydrothorax, is a very serious complication, only less so than the pericarditis which often supervenes under the same circumstances.

When pleuritic effusion is purulent, the prognosis is more grave. Formerly the event was often fatal, by pyæmia, by hectic fever, by secondary tubercular phthisis, or by lardaceous disease; and in the best cases much deformity was the usual result, as described above (p. 1030).

But the bolder treatment on which modern surgery safely ventures, has wonderfully improved the prognosis of empyema. In the case of children a single free opening made and a drainage-tube inserted has repeatedly been followed by re-expansion of the lung and complete recovery, without the least sign of the disease remaining except the scar of the operation. With adults the result is not so often perfect, but here also complete cures are not infrequent, and complete failure is the exception.

Treatment.—It is evident from the facts above stated that the treatment of pleurisy is of great importance. There are cases which, left to themselves, run a course as favourable as could be desired. There are others which, even if they do not end fatally, leave the patient crippled and deformed, worn out and emaciated by the drain of a purulent discharge, or the victim of an incurable affection of the kidneys or intestine.

Except in the mild adhesive form of the disease, a person affected with pleurisy should be kept strictly in bed during the early stage. For the relief of pain, the application of a blister is perhaps more generally serviceable than anything else, and it may be used with advantage at any period. But many physicians hold that for prompt and grateful relief of the acute pain, as well as of the dyspnœa, no remedy is so efficacious as the application of half a dozen leeches to the side. Among milder measures, such as poulticing and the application of belladonna, none are so effectual as careful bandaging of the affected side, so as to restrain its movement in respiration.

Of drugs, those most commonly prescribed when effusion has taken place are purgatives, diuretics (such as digitalis, acetate, iodide, citrate, and nitrate of potass), and mercurial liniment externally. Marked results have followed the application of mercurial ointment, a rub which had been heard day after day for weeks ceasing almost at once, or fluid effusion clearing away after a somewhat longer period. The diet should be light and spare. The plan of allowing the patient very little to drink was often practised at Guy's Hospital by Sir William Gull and by Dr Moxon, and is advocated by Niemeyer. Fräntzel remarks that a very rapid absorption of fluids from the chest has been noticed when the patient has been attacked by cholera;

and the writer has seen the same result from an attack of summer diarrhœa. As a rule, it is better to act on the kidneys than on the skin or the bowels.

Paracentesis thoracis.—Whenever the physical signs indicate that there is considerable liquid effusion into the pleural cavity, the question of removing it by tapping must be carefully considered. This procedure, it is interesting to know, dates back to Hippocrates. But although Trousseau advocated it as far back as 1843, and Hughes as strongly in 1844, few physicians, until twenty years later, understood its importance or safety.*

There is still difference of opinion as to how soon the operation should be undertaken. Trousseau used to tap early and frequently, but if as soon as the signs of fluid in the pleura were recognised, paracentesis were performed, "dry tapping" would be much more common and the results less satisfactory. The best practice seems to be first to try to procure absorption of the effused serum by diuretics and laxatives.

If percussion shows that the quantity of fluid is moderate, it is as a rule advisable to wait. The presence of pyrexia is, at an early stage of pleurisy, a reason for delay. The patient must be watched very closely, to see whether the effusion increases or diminishes as time goes on. Unless it begins to diminish in the course of a fortnight, no further postponement of tapping is desirable. For, even though the quantity of liquid should remain unaltered, the probability that the lung will quickly expand and regain its functions after paracentesis will become less and less, in proportion to the length of time during which it has been compressed; for the layers of lymph that bind it down have been allowed to become fibrous and to contract. Moreover, the withdrawal of a part of the fluid by operation seems often to facilitate the absorption of the rest; it is supposed that the subpleural lymph-channels are mechanically pressed upon by the liquid, and that the flow through them is interfered with; but the same good effect sometimes follows a dry tapping.

We may safely delay with a patient in a hospital ward watched day and night, and with immediate assistance at hand, when it would be imprudent to leave fluid in the chest of one living at a distance from medical aid. In cases of Bright's disease when one lung is half carnified by fluid, paracentesis should be performed at once, or sudden œdema of the other may prove fatal before help can come.

In any case, if percussion shows that one side of the chest is full of fluid, the operation cannot safely be delayed (p. 1028) even until the following day; and this in spite of the absence of obvious distress of breathing, or the presence of pyrexia.

Whenever we have reason to believe empyema to be present by the signs and probabilities mentioned above (p. 1032), we should aspirate at once. As Kussmaul stated in the 'Deutsches Archiv' for 1868, if there be pus in the pleural cavity, its removal is often followed by the cessation of pyrexia.†

* A paper by the late Dr Hughes and Mr Cock recording twenty cases of pleuritic effusion treated by paracentesis appeared in the 'Guy's Reports' for 1844.

† Of this I saw a most striking instance in 1881 in a man who had pleurisy as a sequel of enteric fever. His temperature rose every afternoon to between 102° and 103°, falling in the night and morning. I had made one unsuccessful attempt to draw off the effused fluid, the reason of my failure being that I used a very fine aspirator needle, because the area of percussion dulness was not in the usual position behind, but at the side of the chest, just outside the situation of the pericardium, so that I felt some hesitation in acting upon my diagnosis. However, six days later, when I visited him, I found him in a most critical condition, with great anxiety of face and with extreme distress of breathing. As

When it is decided to tap the chest, some physicians recommend that a preliminary puncture should be made by means of an empty hypodermic syringe, into the chamber of which some of the pleuritic liquid may be drawn. But it often happens that this procedure leads to no result, even though the diagnosis is correct. It has been recommended to fill the syringe with water first and inject a few drops first to ensure a result; but this plan does not always succeed, and may lead to septic contamination. On the whole, the use of the instrument seems to be undesirable, at least when the condition of the patient is so serious as to make the removal of the effusion a matter of importance. For either liquid enters the syringe or it does not. In the former case a larger trocar is at once employed, and the patient might as well have been saved the slight pain of the preliminary puncture. In the latter case one is very much hampered in taking any further step, which is yet perhaps essential to his safety.

The operation has been much facilitated by the introduction of Dieulafoy's aspirator. Its use is greatly to be preferred to an ordinary trocar for paracentesis of the chest, for through a trocar fluid will only escape from the pleural cavity when the pressure there is greater than the atmospheric pressure. Several pints may be present, and yet sometimes there may be only a momentary flow of it during the act of expiration, or when the patient happens to cough. Moreover, there is great danger of air being sucked back into the pleural space by a deep inspiration.*

Even when the aspirator is used, there is often difficulty in getting out any considerable quantity of the liquid. Pieces of lymph are drawn against the inner orifice of the tube, or its channel may be occluded by viscid or curdy pus. Sometimes one can restore the flow by moving the cannula in various directions. If this fails, it may be necessary to withdraw it and to make a second puncture at a different spot. It should be a rule without exception never to employ for tapping the chest the hollow needles which are commonly sold with the aspiratory apparatus. One expects the lung to be expanded as the fluid is withdrawn, and there must be a very great risk of its being wounded if there be a sharp point in the way. A perfectly safe instrument consists of a trocar, and a cannula with a lateral opening, to which the tube of the aspirator is fitted; there is also a stopcock which can be turned at the moment when the trocar, having

the only chance of saving his life I had a somewhat larger trocar plunged into the chest at exactly the same spot as before. Aspiration was then performed, but at first no fluid appeared. However, I took the instrument and pushed it inwards, feeling, as I did this, that the end of it encountered and seemed to pass through a resisting membrane. A quantity of rather viscid blood-stained liquid at once escaped, and of this four and a half ounces were withdrawn. The patient was instantly relieved, and from that time went on to recovery without a bad symptom. His temperature, which was rising at the time of the operation and had reached 100·8°, fell from that very moment; three hours later it was normal, and during the next three days it only once reached 99·4°.—C. H. F.

* Some of those who advocate the operation maintain that the introduction of air is a matter of but little importance, on the ground (which cannot be denied) that it has often occurred without ill effects. But few surgeons would deny that it must involve the risk of giving a septic character to the inflammatory process. However, one can prevent it by surrounding the mouth of the trocar with a piece of moistened gold-beater's skin, which acts as a valve; or, as has been usual in England, one can fit on an elastic tube and make it dip beneath the surface of water containing carbolic acid, in a basin on the floor.

In tapping the chest for simple pleuritic effusion Mr Davies-Colley thinks the best plan is, after introducing the cannula, to attach a long rubber tube to it, and to evacuate the cavity by means of the siphon-action exerted by the fluid in the pendent tube. If a stoppage occurs in the flow, the aspirator may be used to remove it, but otherwise it is better to avoid the strong suction of this instrument, as it may give rise to hæmorrhage.

punctured the chest, is being withdrawn. Another advantage of this instrument is, that should the cannula become obstructed, a blunt probe can be passed in to clear it, without having to detach the aspirator-tube.

The best spot for puncturing the chest is generally said to be about two inches outside the edge of the pectoralis major, and just above the edge of the sixth rib on the left side or of the fifth rib on the right side. At Guy's Hospital it has been usual to select a lower intercostal space, and a point much nearer to the posterior fold of the axilla—the seventh space just outside the angle of the scapula. Bowditch recommended that the instrument should be introduced between the ninth and the eleventh ribs. Whichever space is chosen, we should keep close to the upper edge of a rib in order not to wound the intercostal artery. In 1855 this accident happened during an operation performed at Guy's Hospital; the patient (who had phthisis) became faint at the time, and died the same evening; a pound of clotted blood was found in the base of the chest. Fräntzel speaks of the use of a "capillary" trocar as obviating all risk of such an occurrence; but instances of it are very rare, and from the difficulty of extracting the fluid in many cases, it is injudicious to employ too small an instrument.

It is not desirable, in performing paracentesis, to attempt to empty the pleural cavity. Not more than from one to three pints should be withdrawn at once. In many cases, during the operation or immediately afterwards, paroxysms of cough occur, which are best relieved by a hypodermic injection of morphia.* It is easy to see that the increased activity of circulation in the pulmonary vessels, which must be one result of the operation, may lead to the giving way of any weak spot in their walls; and hæmoptysis is not a very rare effect of paracentesis.

A more frequent occurrence is œdema of the pulmonary tissue on the affected side. This seems to be the cause of a phenomenon which attracted much attention in Paris several years ago—the expectoration after thoracocentesis of large quantities of a frothy liquid containing much albumen. The patient to whom this accident happens may have experienced the usual relief from the operation; but after an interval of from ten minutes to an hour his breathing becomes distressed, he begins to cough, and he may turn livid, and die in a quarter of an hour. Twenty-one instances of it were collected by Terrillon in a monograph published in 1873; most of them, however, ended in recovery, and some were comparatively slight. The close resemblance between the fluid discharged from the air-passages in such cases and that withdrawn by the trocar so short a time before seems to have led some distinguished French observers to suppose that the expectorated albumen was dependent upon the presence of a communication between the cavity of the pleura and the interior of the lung, their notion being either that the lung was wounded during the thoracocentesis or that a perforation existed previously, which became opened out

* As Fräntzel remarks, cough is probably set up by the re-entry of air into the tubes of the lung which had been compressed, or it may be from irritation of the surface of the lung by the cannula. That it is not always due to the latter cause is shown by its being sometimes produced when the quantity of fluid is still so great that such contact cannot have taken place. Moreover, he has repeatedly felt the orifice of the instrument rest against the pulmonary pleura without any cough resulting. Since he has used the aspirator, and drawn off pleuritic effusion very slowly, he has very seldom observed these severe fits of coughing. That they are not always due to expansion of the lung seems probable by their sometimes occurring in cases of empyema with old adhesions.

as the pressure was removed from the surface of the organ. But, as Terrillon had no difficulty in showing, such opinions are quite untenable, and the only reasonable explanation is that there is suddenly produced an active hyperæmia, which leads to œdema of the pulmonary tissue.

Among the objections that have been urged against the performance of thoracocentesis, except in cases of absolute necessity, one, which had the support of Dr Stokes, of Dublin, and of Sir Thomas Watson, is that it may lead to the conversion into pus of an effusion originally sero-fibrinous. And although this suggestion is rejected by Trousseau, there is probably truth in it. Nothing is more likely than that the vessels in an inflamed pleura are often weakened and dilated to such an extent that a sudden and great removal of pressure from them may cause increased exudation. And in some cases in which a clear fluid is withdrawn at a first operation pus appears on the second or third occasion, even when there is no reason to believe that air has entered, or that the instrument was not scrupulously clean. Thus, although it accords with the natural tendency of the disease, that as it advances the proportion of leucocytes in the exudation should become greater, one cannot but admit that this process may sometimes be hastened by the performance of an operation.*

In certain cases the fluid quickly accumulates again after paracentesis, so that the patient's condition becomes as bad as before, and the operation has to be repeated. When this happens two or three times in succession, perhaps at intervals of only a few days, Fräntzel advises that one should desist from further interference, which will only precipitate the downward course of the case. But this is not the writer's experience in the case of a girl, a patient of Mr Lacey, of Woolwich, who was tapped fourteen times for hydrothorax in the course of mitral disease, sometimes at intervals of only a very few days, and yet recovered.

Paracentesis in empyema.—If the fluid obtained by paracentesis is purulent, the further treatment requires to be modified. One plan is to withdraw as much of the pus as possible, and then to close the puncture with lint and plaster, for sometimes what remains of the effusion undergoes absorption. Cheesy masses, and even thick, hard deposits of calcareous matter, are now and then found after death lying between adherent pleural surfaces; and there can be little doubt that such residues have usually, if not always, had their origin in an empyema. In other cases a collection of liquid pus, enclosed in a dense capsule, has been discovered in the pleural cavity, when death was due to another cause. Dr Moxon cites such an instance, in which there was a history of pleurisy three years before, and in which physical signs of fluid in the chest had been observed during the intervening period. But in most cases the effusion quickly reaccumulates, a second operation is required, and after this a third. Each time adhesions may possibly form between the opposed surfaces of the pleura, so as to narrow the cavity; but a third operation should be an incision.

The other plan is to make a free incision as soon as the pus is seen flowing

* Fräntzel speaks of a burning pain in the seat of puncture and of an indefinite sense of pressure on the affected side as being generally present, and as lasting in some cases for a day or two. He also says that thoracocentesis is usually followed by a slight elevation of temperature, and that until the second, third, or fourth day a more or less considerable increase of effusion may be observed, after which a somewhat rapid process of absorption sets in, attended with diuresis. How such statements agree with the fact that an existing pyrexia is often suddenly cut short by the operation is not apparent. However this may be, there is no doubt that patients experience marked relief from the operation.

out, to put in a drainage-tube, and to allow the pus to escape into an antiseptic dressing, treating the empyema, in fact, as a large abscess: and this the present writer believes to be, as a rule, the safer as well as the more effectual practice.

In children we have sometimes attained excellent results by making an aperture just large enough to admit one end of a long elastic tube, of which the other end is carried beneath the surface of carbolised liquid in a jar placed beneath the bed. The elastic skin of a child grasps the tube firmly, and does not ulcerate. The negative pressure of the column of liquid, acting hydrostatically, seems gradually to raise the compressed lung. Within a short time the flow of pus may cease, and a permanent cure may be obtained. With older patients similar success is rare.

Injections of iodine, or of permanganate of potass, are often used to wash out the cavity at intervals of twenty-four or forty-eight hours. The incision should be made along the upper border of the rib, a short distance in front of the angle. A large tube should be introduced, and care should be taken to fix it in such a way that only a small portion of the tube enters the pleural cavity. In children it may be necessary to excise part of a rib to allow of the introduction of a sufficiently large tube.*

The operation of washing out the chest is not altogether free from danger. In 1876 Dr Cayley read before the Clinical Society a case which had occurred to him, and in which, while a solution of iodine was being injected, the patient suddenly became pale, unconscious, and convulsed; the temperature rose to 107°, and death followed in sixteen hours. He cites three cases recorded by French observers, in each of which like symptoms appeared, though one of them ended favourably. In 1874, at Guy's Hospital, a girl, aged sixteen, died in precisely the same way. She had had a drainage-tube inserted into the right chest for an empyema five weeks before, and was going on well. One day she was sitting up, and her chest was being washed out with carbolic acid, when she suddenly ceased to breathe, and, although artificial respiration was set in action, remained unconscious, with muscular twitchings, until death. Nothing has been found, on *post-mortem* examination, to account for such accidents. A suggestion that thrombi in the pulmonary veins may have been dislodged

* A somewhat different method is recommended by Fräntzel. Having, at the time of the operation, got rid of as much as possible of the pus, he pushes a long catheter downwards towards the spine, and slowly injects through it distilled water, at a temperature of 100° Fahr., until the space is full. He then draws off the water by another catheter with an exhausting syringe, and he repeats this procedure three or four times until what returns is quite clear. Masses of fibrin, sometimes as large as the palm of the hand, generally appear in the wound while this is being done, and are carefully removed. This is one of the advantages of Fräntzel's plan, for sometimes much trouble is caused at a later period by such masses, which may decompose. Finally, he fixes in the aperture a flat silver cannula, with a broad plate fitting upon the surface of the chest, of such a size that two catheters can be passed through it side by side; outside this are placed antiseptic dressings, and over them a bag of ice. Each day afterwards the pleural cavity is twice washed out by means of the catheters, of which one is introduced with great care to the farthest possible point, so as to prevent any accumulation of pus from taking place. He deems it important for the patient to lie in such a position that the wound is at a higher level than any part of the pleural space, so that the water may gravitate into every part. After two days a ½ per cent. solution of common salt is substituted; and later still a very dilute solution of iodine or of carbolic acid. Of eleven patients treated in this manner five were completely cured; five died, but most of them from causes which had little or nothing to do with the operation; one was under observation when Fräntzel wrote, and was doing well.

and have formed emboli in the heart or in the cerebral arteries seems to have been shown to be without foundation. It is noteworthy that in every one of the cases the chest had been washed out many times before without any ill effects occurring; the only difference being that in two instances a somewhat larger quantity of fluid was being injected than usual.

The process by which the sac of a discharging empyema becomes gradually obliterated seems to consist in the formation of granulation tissue, and in the union of the two opposed surfaces; in most cases this union probably begins at the root of the lung, and spreads from one point to another until it reaches the external orifice. Even before this has obliterated the cavity, absorption of any air in the pleura takes place by solution in the lymph of the serous membrane, and, the wound being closed, the negative pressure in the pleura soon allows of the lung expanding if it is not bound down by adhesions. Fräntzel suggests, with much probability, that the compressed lung is re-inflated with air from the opposite lung during the act of coughing, or as the result of simple expiratory efforts with closed glottis.

The results of treatment of empyema by free incision and drainage, with modern methods and antiseptic precautions, are most encouraging. Even in adults one may again and again see complete recovery of the lung with no resulting deformity. In children the practice is still more successful. Thus Dr Goodhart reports that of 50 cases under Dr Frederick Taylor's care or his own 42 completely recovered, a sinus remained in 3, and only 5 died—1 from suppurative pericarditis, 1 from septic pneumonia, and 1 from measles and broncho-pneumonia.

In a certain number of cases the cure of an empyema remains incomplete. The cavity may have shrunk to very narrow dimensions, the chest may have regained a fair amount of resonance over a large part of its surface, air may enter the lungs pretty freely, but there is a fistulous opening from which small quantities of pus continually drain away. In such cases, and generally when other treatment has proved unsuccessful, it is now the practice to excise portions of one or more ribs, so as to allow the side of the chest to fall in and meet the lung. This operation seems to have been first performed by Dr Peitavy, and is now common.*

PNEUMOTHORAX.—Morgagni mentions the fact that air sometimes accumulates in the cavity of the pleura; but the word pneumothorax was first used in 1803 by Itard, and it was left for Laennec to give a full description of this affection.

Ætiology.—The older writers supposed that in exceptional cases gases form in the pleural cavity as the result of chemical decomposition or of liquid effusion, by direct secretion from the lining membrane. But this belief is unsupported by pathology or by experience; air is never found in the pleural space except when admitted from without.

* In the 'Birmingham Medical Review' for 1880 Dr William Thomas recorded several cases which were so treated, and in nearly all of them the result was highly satisfactory, the wound healing in a few weeks, and the lung re-expanding completely. The rib, too, was restored by a new growth of bone. It is to be observed, however, that the patients were all children under eight years old. In 1877 Mr Howse excised portions of three ribs from a child, aged six, a patient of Dr Taylor, in the Evelina Hospital, with results which are detailed in vol. xiii of the Clinical Society's 'Transactions.' Some improvement followed, but the cavity of the empyema did not close, and ultimately the child died with lardaceous organs. We have had numerous cases since, many successful; and sometimes the operation is absolutely necessary to give room for the drainage-tube. But it is better avoided when possible.

In the great majority of cases pneumothorax is a consequence of perforation of the *visceral layer* of the pleura, whereby air escapes from the lung. Often this arises from direct violence: broken ribs are exceedingly apt to wound the lung, and in persons who are run over or severely crushed the lung may be torn without there being any fracture of bone or laceration of the costal pleura. Exceptional instances of pneumothorax occur in medical practice in which a lung, previously healthy, is ruptured during a straining effort, or in a paroxysm of whooping-cough.* The fact that the bullæ of emphysema have often the thinnest conceivable walls might lead one to suppose that pneumothorax would occasionally arise from their rupture. But this (if it ever happens) must be exceedingly rare.

Occasionally the entrance of air into the pleural space is the result of local ulceration or sloughing of the pulmonary pleura. Sometimes, though very seldom, the disease is acute pneumonia running on to gangrene. Much more often it is a slough, dependent upon infective embolism in the case of pyæmia. Sometimes it is sacculated dilatations of the bronchial tubes which open into the pleura. But such cases are extremely rare.

The only common cause of pneumothorax is the rupture of a superficial vomica in phthisis. Walshe estimates that nine out of ten cases arise in this way, and Fräntzel puts it at fourteen to one. It would occur much oftener than it does but for the adhesive pleurisy, which accompanies chronic phthisis and generally seals up the pleural cavity. Even when there is no evidence of phthisis either before or afterwards, it is a question whether the spontaneous development of pneumothorax, without any previous violent muscular effort, is not due to the rupture of a small vomica, by which neither physical signs nor symptoms have been produced.

The following case was related by Prof. Vogel, of Dorpat, in the second volume of the 'Deutsches Archiv.' A woman, aged twenty-nine, became suddenly the subject of pneumothorax one morning at nine o'clock. Some months previously she had a slight loose cough, and more recently a little pricking pain in the region of the liver; when the attack began she was engaged in turning over her child's bed, and just before she had been lifting its bath. Vogel himself was inclined to think that she had latent tubercular disease, but in four weeks she recovered completely.

Another way in which pneumothorax may arise is from perforation of the visceral pleura by an empyema discharging itself through the air-passages. In medical practice this cause comes next to phthisis in frequency. Possibly pleurisy may give rise to pneumothorax at an earlier stage, if the inflammation is sufficiently intense to lead to sloughing of the visceral layer of the membrane. Thus in 1869 a man, aged forty-two, was brought into Guy's Hospital very ill, and died half an hour after. He was found to have acute pericarditis, mediastinal inflammation, and early pleurisy on the left side. But the principal seat of disease was the right pleural cavity. This contained fœtid gas, and four and a half pints of dirty purulent fluid. In the upper lobe of the lung there were two openings, and through these air had doubtless entered. But the pulmonary pleura was gangrenous over an area of two square inches, and the substance of the lung beneath it to a

* Fräntzel relates in 'Ziemssen's Handbuch' how a youth of nineteen, who was exerting all his strength to push a heavy cask, felt something give way in his chest, and became suddenly short of breath and powerless. As he recovered in six weeks, without any pulmonary disease being discovered, it may probably be assumed that none had existed before.

depth of half an inch. That the pleurisy was of exceptional severity was evident from the fact that there was suppuration outside its parietal layer, among the intercostal muscles.

There still remain cases in which the air is not derived from the lung at all, but either from the outside of the chest, or from some part of the alimentary canal. As a consequence of perforation of the thoracic walls (apart from the effect of penetrating wounds of the chest), pneumothorax may be seen by physicians when an empyema has broken through spontaneously, or has been let out by operation. When the pus points of its own accord, the channel by which it reaches the surface is commonly oblique and indirect, so that air fails to find its way along it. Dr Moxon drew attention to the possible occurrence of double pneumothorax as a cause of death after tracheotomy, subcutaneous emphysema extending down from the wound so as to fill the mediastinal connective tissue with air, which then bursts into both pleural cavities. One such case occurred in a woman, aged thirty-three, who died in less than twenty-four hours after the operation. Emphysema had spread over the neck, chest, and arms as far as the fingers. Both lungs were found collapsed and almost airless.

The part of the alimentary canal which is most often the starting-point of pneumothorax is the œsophagus; a malignant growth may eat its way into the pleural cavity, or the ulceration due to a foreign body may have a like result. But sometimes a gastric ulcer, after setting up a circumscribed hypophrenic abscess, has led to perforation of the diaphragm; and a hydatid cyst of the liver has been known to open communications in two opposite directions, with the bowel below, and with the pleural space above.

Physical causes of pneumothorax.—Few hospital physicians can have failed to meet with cases difficult to explain, in which communication of the pleura with the outer air, through the parietes of the chest or through the lung, has not been followed by pneumothorax. In most cases pleural adhesions, at or in the near neighbourhood of the perforation, afford a probable account of the difficulty, and their presence has been confirmed after the patient's death. But certain cases seem incapable of this explanation. In a lecture reported in the 'British Medical Journal' for June 4th, 1887, Dr Goodhart —after referring to the fact that pleuritic effusion may be "held up," so to speak, over the lung instead of all gravitating to the bottom of the chest (cf. p. 1022)—proceeds to comment on the rarity of pneumothorax after fracture of the ribs, particularly in young patients, and on the free re-expansion of the lung, even after a large opening has been made into the chest by paracentesis, and seems to think that there must be some forces at work to allow of the lung becoming inflated again by inspiration different from those known to physics. Mr Godlee replied in a subsequent number of the same journal from the surgical point of view, arguing the expediency of excision of the ribs in the treatment of empyema.

The subject was dealt with by Dr Samuel West in his interesting Bradshaw Lecture before the College of Physicians (*ibid.*, August, 1887). He showed experimentally that there is considerable power of cohesion between opposed surfaces of serous membrane, and attributed to this cause the fact that pneumothorax does not occur so readily as one might have supposed.

There can be no doubt in the mind of any physiologist who has seen the unfailing and immediate collapse of the lungs which follows a free incision into the pleura in the case of an animal, that when the pressure on the inside and outside of the lung is equal, it at once shrinks to the bulk

which the elasticity of its tissue permits. This is proved experimentally by Donder's "schema," and by a similar arrangement of the dead thorax with manometers to gauge the pressure. The terrible result of tapping a healthy pleura when the other lung is incapable of expansion confirms the conclusion derived from more frequent and harmless experiments.

Apart from any mechanical obstacle to separation of the two layers of pleura, either from adhesions or from their natural cohesion, it is obvious that when a pleuritic effusion is tapped and runs out, the intra-thoracic pressure must be greater than the barometric pressure of the air at the time. When it becomes greater with expiration, the pus or serum runs with a jerk; when it lessens with inspiration, the flow lessens also; when the pressure on the chest becomes negative, the flow ceases; or, if there is not enough fluid to close the trocar, air is sucked in. But when the orifice is closed, after a little air has been admitted no more will enter, and that already in the pleura will be quickly dissolved by the remaining fluid, or will be absorbed by the lymphatics of the pleura—first the oxygen, then the nitrogen and carbonic dioxide. As the air is thus absorbed, the pressure in the pleura diminishes, and the lung again expands. Even if a large opening is made the lung is not emptied of air; it is only reduced to the condition observed after the thorax is opened, after death from some disease which has not affected the thorax. There is no carnified (*i. e.* completely airless) lung to be re-expanded, unless pleuritic effusion has compressed it for a long time. As soon as the pressure on its surface becomes a little less than that in the trachea the compressed lung will begin to expand again, if not mechanically prevented by thick adhesions.

Anatomy.—The recognition of pneumothorax is not always a perfectly simple matter, even in the dead body. In making an autopsy, at the moment when the knife is first plunged into the thorax, the air can sometimes be heard to rush out; or, if a puncture is made with a trocar, it may escape in a jet, so as to blow out a lighted match. But this occurs only when its pressure is greater than that of the atmosphere, which is by no means generally the case. In all probability the existence of air in the pleural space is very often overlooked in ordinary *post-mortem* examinations, especially in the bodies of phthisical patients, in whom, from the presence of extensive adhesions, the collapse of the lung has been only partial. The best way of making sure whether there is pneumothorax or not is to puncture the chest under water, which may be done either by dissecting off the tissues from the ribs, so as to form a pouch that can be filled with water, or by pouring water into the abdomen, and then perforating the diaphragm with a trocar. If pleuritic effusion is present, it is sufficient to shake the body before opening the chest; then, if there is any air, the liquid will be frothy.

When the pneumothorax arises from perforation of the visceral pleura, the aperture by which the air entered is sometimes plainly visible; it may be as large as a threepenny-piece. More often it is covered by recently formed lymph, and the only way of detecting it is to inflate the lung with bellows through the trachea. Or it may have become completely closed by adhesions during the interval between the occurrence of the pneumothorax and the death of the patient, so that there is no possibility of discovering it. It is most commonly situated upon the lateral surface of the lung, in the upper lobe near its lower border, or in the lower lobe near its upper border.

The chemical nature of air withdrawn from the pleural space was inves-

tigated by Dr John Davy many years ago,* and analyses have since been made by other chemists; it has always been found to consist mainly of nitrogen, and the amount of carbonic acid in it has generally been greater than that of the oxygen; sulphuretted hydrogen has been present when the other contents of the cavity were putrid. Dr Walshe explains the difference of this pleural air from that of the atmospheric by the fact that it traversed the lung before reaching the pleura. But it can hardly be said to have passed through pulmonary tissue, and, moreover, its composition is far more altered than that of normally expired air. Obviously, therefore, it must have undergone change while in the pleural space, either from the solvent action of the liquid effusion, or from absorption by the pleural membrane—*i. e.* by the lymph which fills its stomata and lymphatics, and by the subpleural veins.

The secondary pleurisy.—When death occurs within a few hours after the development of pneumothorax, the cavity of the pleura is of course found empty, there having been no time for the occurrence of effusion. But in other cases, at least such as are seen by physicians, an empyema is, as a rule, formed in a few days. Dr Walshe has discovered signs of liquid effusion within twenty-four hours. A striking instance to the contrary is afforded by Vogel's case, already quoted (p. 1043). He repeatedly examined his patient during the month after she was attacked, and could never detect the slightest indication of pleurisy.

There is no doubt that the power of the air to set up suppuration depends upon its containing septic microbes. But, as we have seen, pneumothorax is, in most cases, due to the rupture of a phthisical vomica into the pleural space. The contents of the vomica must generally escape with the air, and they are probably the cause of the pleurisy which follows. When the original affection is a sloughing block in the lung, or when the pleura is perforated by a malignant œsophageal growth, or by a hypophrenic abscess communicating with the stomach, the consequent inflammation is peculiarly severe and rapid in its course.† On the other hand, in surgical practice, when a healthy lung is wounded by fractured ribs, we are told that pleurisy is often absent. Probably very much depends upon whether or not the aperture in the lung becomes quickly closed again; for the risk of the entrance of germs must be greatly diminished if no air is admitted beyond that which immediately fills the serous cavity.

It is clearly impossible for subcutaneous emphysema to be produced by fracture of the ribs without there being also pneumothorax, unless the pleural space at the seat of injury happens to have been closed by former adhesions. But in cases of this kind we have often failed to detect any signs of the presence of air in the serous cavity when a day or two had passed before an opportunity of examining the patient was afforded. So that air must often be absorbed very rapidly from the pleural space; and this conclusion is quite in accordance with the results of experiments on animals. Cohnheim says that in rabbits it is not possible by injection of air into the pleura to cause compression of the lung so as to study the effects of pneumothorax, because the air is so quickly absorbed.

Locality.—Dr Walshe states that of eighty-seven cases of perforation

* 'Phil. Trans.,' 1823; and in his collected 'Researches,' vol. ii, p. 249.

† Of this we have recently had an instance (June, 1890) in a patient with extensive sero-purulent effusion on the right side caused by a sloughing abscess perforating the diaphragm, which originated in cancer of the stomach.

of a tuberculous lung collected by him from various sources, fifty-five affected the left and thirty-two the right pleura. But among twenty-six cases of pneumothorax taken from the records of Guy's Hospital without selection, the number on each side of the chest was exactly equal.

Physical signs.—Whether pneumothorax is easy or difficult of diagnosis, depends upon a variety of circumstances. Its recognition must always be the result of physical examination of the chest, although the patient's symptoms, and the way in which they develop themselves, often enable one to guess the nature of the case.

In general, pneumothorax is to be suspected whenever, over a large part of the chest, but on one side only, marked deficiency of vesicular murmur is associated with tympanitic resonance on percussion.

Enfeeblement or *absence of vesicular murmur* is a very important indication; and sometimes the faint sound which accompanies the breathing gives one an impression of being conveyed from a distant part of the chest. But in many cases there is marked *amphoric breathing*. Sometimes, no doubt, this is due directly to the passage of air backwards and forwards into the pleural cavity; but it is often present when the aperture is closed, and when, as Dr Gee remarks, it must acquire its peculiar quality by transmission through the pneumothorax.

The *voice* may either be less audible than on the healthy side, or it may be conveyed so as to produce bronchophony or even pectoriloquy. As a rule, *vocal fremitus* is either absent or greatly diminished.

The *percussion-note* is altered in the direction of over-resonance, with, as a rule, a tympanitic quality. When air escapes into a healthy pleural sac the sound is, as a rule, musically clear and resounding. But if the air should accumulate so as to cause extreme distension, it may, in the words of Dr Walshe, at length become "muffled, toneless, almost dull," like that of a drum tightened to the highest possible point, and with all escape of air from its cavity prevented. More frequently, the reason why the percussion-sound in pneumothorax is imperfectly resonant is that the pleura itself is thickened; in all probability not only do the chest-walls themselves fail to vibrate, but they are incapable of transmitting the percussion-note to the air within, so as to throw it into free vibration. In such cases one may obtain any one of the modifications of percussion-sound mentioned at p. 930 —osteal, tracheal, or subtympanitic.

Metallic sounds.—There still remain certain physical signs, which when present are striking and characteristic, although their absence is no argument against the existence of pneumothorax. They may be grouped together as "metallic" sounds.

Laennec described *metallic tinkling** as a sound like that "produced in a metal cup, or in one made of glass or of porcelain, by gently striking it with a pin, or by dropping into it a grain of sand." He described it as being heard when the patient either breathed, or spoke, or coughed. There was afterwards much discussion as to the origin of this sound, but there is now general agreement that it is caused by the bursting of bubbles of fluid in a large space, which is filled with air and has a smooth surface. In other words, metallic tinkling is a moist sound, or râle, modified by the vibrations of the walls of a large air-containing cavity. One way in which the bubbling necessary to give rise to such a sound may be produced was noticed by Laennec himself; namely, by the

* *Fr.* Tintement métallique.—*Germ.* Metallklang.

dropping of liquid from the upper into the lower part of the pleural space when it contains air as well as pus. Thus metallic tinkling may be due to the patient's changing from the recumbent to the sitting posture; but one is hardly likely to hear this unless one has the stethoscope applied to his chest before he begins to move. Again, there is no difficulty in understanding how the sign may be produced by coughing, as well as by drawing in the breath, especially if there be a free communication between the space in which it is found and a bronchus.

True metallic tinkling does not seem to arise as a mere result of speaking. What is then heard is rather an *echo* of the voice, which acquires a metallic quality from the conditions under which it is produced. So also the heart-sounds, and even the sound produced by percussion of the chest, may be reverberated with a similar musical quality. The thin, clear "tick-tack" of the heart in pneumothorax is like the sound of a watch or a child's "musical cart," and is a most characteristic sign. To these phenomena it would be well to give the name of "metallic echo;" reserving that of "metallic tinkling" for sounds which in their origin resemble râles. Coughing may be attended either with echo, or with tinkling.

A particular kind of metallic echo was described by Trousseau as *le bruit d'airain*. Among all the "metallic" signs it has the advantage of being completely under the control of the observer. Metallic tinkling is well known to be exceedingly capricious, accompanying certain respiratory movements and being absent with others, according as bubbles happen or do not happen to burst. Even a metallic echo of the patient's voice may probably fail to be heard unless he speaks distinctly and with a certain pitch and loudness. But the *bruit d'airain* can not only be determined as to the time, but the sound which is to produce it can be varied, until one obtains the best possible result. The method of eliciting it was originally given in the 'Gazette des Hôpitaux' for 1859. It consists in applying one's ear to the back of the patient's chest, while a third person strikes the front of the chest, either with the hammer upon the plessimeter, or with one coin upon another. The metallic echo which results is sometimes wonderfully distinct, and there are probably few cases of pneumothorax in which it is absent. One may sometimes, however, fail to obtain a "bell-sound" by percussion during life, and yet have no difficulty in eliciting it from the dead body of the same patient. Traube attributes this fact to lowering of the tension of the air in the pleural space, as the result of cooling of the tissues after death.

Another sign of pneumothorax, which is of great historical interest, is termed the *succussion-splash*. It was well known to Hippocrates, so that it is sometimes spoken of as "Hippocratic succussion."* To obtain it, one may shake the patient's body while one has one's head pressed against his chest. But sometimes it can be heard at a little distance off,

* "Another malady. When the time grows long [after an inflammation of the chest has appeared], then the fever becomes higher and the cough increases, and the patient's side pains him, and he can no longer bear to lie on the sound side but only on the diseased one, and his feet swell and the hollow of his eyes. Then, when fifteen days have elapsed since the rupture [*i. e.* the bursting of an abscess into the pleura as the result of peripneumony, for that was the Hippocratic pathology of empyema], give the patient a warm bath [or, possibly, bathe the affected side with hot water] and set him upon a good steady stool. Then, while a friend holds his hands, do you shake him by the shoulder and listen, so as to tell on which side of the chest there is a splash (ψοφεῖ). [The word ψόφος denotes a noise, *strepitus*, as opposed to a musical or an articulate sound: it is applied to doors banging, armour clanging, and streams splashing.]"—Hipp., 'De Morbis,' lib. ii, cap. xvi.

and the patient himself may be conscious of it every time he makes any abrupt movement, as in stepping downstairs, or in riding on horseback. It is literally nothing else than the splashing of pleuritic effusion against the sides of the serous cavity, and of course it is never audible unless there is liquid present as well as air.

In such cases, accordingly, the signs of pleuritic effusion are to be observed, as well as those of pneumothorax; it may also be noted that alterations of the level of dulness when the patient changes his posture are generally very conspicuous, whereas in uncomplicated pleurisy they can seldom be made out satisfactorily (cf. p. 1022).

Dislocation of viscera.—In most instances pneumothorax is attended with lateral displacement of the heart. Dr Douglas Powell showed in vol. lix of the 'Med.-Chir. Trans.' that the mere elasticity of the opposite lung drags the mediastinum over whenever air has free entrance into one pleural space, without there being of necessity any excess of pressure above that of the atmosphere. He remarks, however, that, in some cases of phthisis, consolidation of the lung on the side opposite to the pneumothorax prevents the mediastinum from being thus displaced; and probably a like effect is also produced by consolidation and adhesion of any considerable part of the lung on the side of the pleural affection, or, again, by the rigidity and thickening of the pleura, which so often occur in cases of empyema before perforation takes place. Thus one must not expect to find the heart beating in an abnormal position in those cases of chronic disease of the chest in which it is sometimes so difficult to determine whether pneumothorax is or is not present.

Even in such cases it is possible for the pressure of the air in the pleural cavity to be considerably increased. The way in which this is brought about is by the action of a piece of false membrane lying over the aperture; this plays the part of a valve, and allows air to enter the cavity during inspiration, but hinders its escape during expiration. Cohnheim, indeed, declares that for air confined in the pleural space to retain for any length of time a high pressure after closure of the opening by which it entered is impossible, on account of the rapidity with which it undergoes absorption. But it is certain that among 17 cases collected by Dr Powell there were 12 in which, after death, the pressure was found to be above the atmospheric pressure, the difference amounting in these cases to that of a column of from five and a half to seven inches of water. When the adjacent organs are capable of yielding to it, one cannot be surprised that the elastic force exerted by air in the pleural cavity should displace them even more than they are displaced by liquid effusion. Thus Dr Gee speaks of the diaphragm as being pushed down so that the upper surface of the liver lies below the level of the anterior costal margin, and percussion yields a tympanitic sound in the right hypochondrium. The intercostal spaces, too, may be flattened or bulging; and the affected side of the chest may be obviously enlarged as well as motionless.

Diagnosis.—There are few affections of which the physical signs may be mistaken for pneumothorax, or *vice versâ*.

The limitation of the signs to one side of the chest obviously suffices to exclude the possibility of their being due to emphysema, which from the time of Laennec has been given as the disease most needing distinction from pneumothorax; in practice the two are not in the least likely to be confounded.

When distension of the pleura with air is very extreme, the percussion-sound may become muffled and toneless; but this probably never reaches such a point that the case could be supposed to be one of liquid effusion.

In vol. xi of the 'St Bartholomew's Hospital Reports,' Mr Butlin has recorded an example of rupture of the diaphragm, with escape of the distended stomach and colon into the left pleural cavity; it was the result of a severe crush between the buffers of two railway coal-waggons, and was diagnosed during life as traumatic pneumothorax.

But in general the only cases which are attended with doubt are those in which, if air is present in the pleura at all, it is confined to a limited portion of the serous space. Thus at the upper part of the chest it might very likely be impossible to diagnose a localised pneumothorax from an exceedingly large vomica. It is doubtful, however, whether limited pneumothorax ever occurs in that position, and in all probability the cases that have been admitted as open to question have been really examples of vomicæ attended with unusual signs, such as metallic tinkling or Hippocratic succussion-splash. On the other hand, at the base of the chest, a cavity within the lung of sufficient size to be mistaken for pneumothorax is a thing almost, if not quite unknown.

It is possible that during the contraction of an empyema on the left side the diaphragm, with the stomach, may be drawn upwards so far that percussion may yield a tympanitic sound over a considerable area, where complete dulness might have been expected. A similar condition may also arise when the lung is affected with cirrhosis. Probably one might avoid an error of diagnosis by re-examining the patient after having made him swallow a large quantity of fluid. In a case of Wintrich's a subdiaphragmatic abscess, which arose from a perforating ulcer of the stomach, and consequently contained air, was mistaken for pneumothorax.

It is necessary to bear in mind the fact, mentioned at p. 990, that in some rare instances the percussion-note is tympanitic over part of a lung affected with pneumonic hepatisation. The other physical signs would negative the suspicion that there is air in the pleural cavity.

After all, the mistake which is most apt to be made in regard to pneumothorax is not that it is taken for any other affection or any other affection for it, but that its presence is overlooked. This is due to the fact that the symptoms of pneumothorax, though often of the most striking character, are sometimes altogether absent.

Symptoms.—The amount of dyspnœa produced by the escape of air into the pleural sac depends upon two conditions: first, upon whether the patient's vital functions are or are not being actively carried on at the time; and, secondly, upon whether he has or has not been accustomed to make full use of the lung on that side in breathing. A healthy person always experiences great distress when attacked with pneumothorax. Among those who are the subjects of disease the distress is greater in proportion as they are well nourished and able to take food and to bear exertion. It is also greater among those who have chronic pulmonary disease, in proportion as the lung on the side of the pneumothorax took a greater share of the work of respiration before the supervention of the accident. It accordingly reaches its maximum when a man who has one lung extensively diseased but whose health is nevertheless pretty good, becomes attacked with pneumothorax on the opposite side. A directly fatal result is then inevitable. Accordingly pneumothorax has to be remembered among the possible causes of sudden

death in persons who are walking about and earning their living. One morning, in the year 1874, there was brought into Guy's Hospital the body of a man who had fallen dead while on his way to his work; he was found to have pneumothorax on the right side, and chronic phthisis of the left lung.

On the other hand, if air escapes into the pleura of a person who is wasted, and whose functions are already at a low ebb, and especially if the lung on that side has before been rendered almost useless by advanced tubercular disease, the supervention of the pneumothorax may give rise to no symptoms whatever. This fact was stated in the 'Medical Gazette' for 1844, by Dr Hughes, who was one of the best auscultators of that day.

In persons who are in the last stage of phthisis, it is possible for pneumothorax to produce a shock that may be directly fatal, without any warning symptoms. The patient is perhaps found dead in bed, and nothing has occurred to attract the attention of the nurse.

Between the two extremes just described there are all degrees of severity in the symptoms of pneumothorax. The most typical cases are those in which the patient is suddenly seized with an agonising pain in the side, and has a sensation of something having given way or possibly of a stream of air or of water trickling down within his chest. His dyspnœa is extreme; the respirations may reach forty or even sixty in the minute, while the beats of the heart, although accelerated, are not so to any proportionate extent. The pulse is small, the radial arteries being imperfectly filled as a consequence of the deficient flow through the obstructed lungs. The hands, the feet, the cheeks, the lips, and the visible mucous membranes become cyanosed; the extremities and even the tongue feel cold; a cold sweat breaks out over the body; the temperature, even in the rectum, falls considerably. The voice is weak, or reduced to a whisper. There may be complete inability to cough. The patient is usually obliged to sit up in bed; sometimes he finds it more comfortable to incline towards the affected side, sometimes towards the healthy side.

Prognosis.—In some cases of pneumothorax the symptoms continue unabated until the death of the patient, which may take place after a few hours, or in a day or two. But in other cases they subside as the shock of the accident passes off; the breathing may remain rapid and yet the patient may experience little or no distress, as in a case in which Dr Walshe counted fifty-two respirations in the minute. In some very exceptional instances the air gradually undergoes absorption, and complete recovery takes place. Vogel's case, referred to at p. 1043, was one of these.

What usually happens is that after a few days pleurisy sets in. Even then it is not impossible for the disease to subside. Dr Walshe says that he has seen two cases in which in the course of two months all signs of air and fluid in the pleura disappeared; in all probability the exudation was sero-fibrinous. As a rule, when an empyema is developed, one can give the patient a chance of recovery only by making a free external opening, so as to allow the cavity to become obliterated by the process of granulation. The same treatment is of course necessary when the entrance of air into the pleura is secondary to pleurisy. But in cases in which there is already advanced phthisis it is scarcely ever right to perform such an operation, as the resulting inflammation of the pleural space is apt to assume a putrid character, and carry off the patient very rapidly. Nevertheless, Czernicki pointed out ('Gaz. hebd.,' 1872) that in some phthisical patients the supervention of pneumothorax with consequent spontaneous

pleuritic effusion actually leads to an improvement in the general symptoms and to cessation of expectoration, effects which can only be ascribed to anæmia of the affected lung, resulting from its collapse.

As a rule, death occurs within two or three weeks after perforation of the pleura. Traube insisted on the rapidity with which emaciation advances in many cases. Œdema of the limbs, and even of the face, sometimes develops itself; and the urine is sometimes albuminous.

On the other hand, it is surprising how long a pyo-pneumothorax may be tolerated, and how little discomfort it causes. The patient is sometimes able to take horse exercise, and thus, as already mentioned, may hear the fluid splashing within his chest while he is riding.

Traube relates a case of pneumothorax occurring in a woman who had been attacked by it some years before he first saw her, and in whom seven years later scarcely any physical signs were discoverable. She looked well, and could even walk uphill without discomfort. The history appeared to indicate that the affection arose as a complication of phthisis; for she had previously had a febrile illness, with night-sweats, cough, and hæmoptysis.

Treatment.—In cases of pneumothorax a great deal can be done to diminish the patient's sufferings, and perhaps to avert a fatal termination. Cupping, dry or wet, often gives remarkable relief, and venesection is probably still more efficacious. A small dose of morphia should be injected subcutaneously, or, as Dr Walshe recommends, a very little chloroform may be given by inhalation from time to time. He also says that he has seen musk in five-grain doses afford much relief.

If great enlargement of the side and depression of the diaphragm suggest that the pressure of air within the thorax is greater than the atmospheric pressure, paracentesis should be performed with a fine trocar. But displacement of the heart alone is not evidence of increased pressure; it may be due to the elasticity of the mediastinal tissues. Fräntzel appears to have tapped for pneumothorax rather frequently: he quotes a dissertation by Bärensprung, in which are recorded a number of cases treated in this way with success. He says that, if possible, it is well to postpone the operation until three or four days have elapsed, so that the aperture in the pleura which allowed the escape of the air may have closed. Between the fourth and the eighth day it may be advisable to introduce a trocar, even when the symptoms are not very urgent. He has often found the pressure of the air such that a considerable quantity passed out through the instrument; and, even when this is not the case, it is easy to close the wound, and no harm is done. An aspirator ought never to be employed, on account of the risk of reopening the original aperture. Fräntzel uses an ordinary trocar with a valve of goldbeater's skin (p. 1038, *note*). If cough arises during the operation, which is not uncommon, he either gives an injection of morphia, and waits for a time before withdrawing the trocar, or else he keeps up pressure upon the seat of puncture until the cough has ceased. By either of these methods the escape of air along the track of the trocar may be prevented, which would otherwise lead to subcutaneous emphysema.

PHTHISIS*

"While meagre Phthisis gives a silent blow,
Her strokes are sure, but her advances slow;
No loud alarms nor fierce assaults are shown;
She starves the fortress first, then takes the town."

GARTH.

History and definition—Pathology—unity of phthisis—localisation—phthisis always tubercular—Histology: miliary, caseous and infiltrating tubercle—Tubercular pneumonia and ulceration—vomicæ—adhesions: involution——Symptoms: wasting, pyrexia, cough, sputum—The bacillus—Hæmoptysis—Physical signs of the three stages and of involution—Diagnosis—Course and duration—mode of death—recovery—Prognosis—Ætiology—the question of contagion—hereditary taint—diathesis and conformation—overcrowding, &c.—inhalation of dust—damp soil—Age and sex—Distribution—Treatment: preventive, curative, and palliative—diet and hygiene—climate—drugs.

ACUTE PULMONARY TUBERCULOSIS—*Distinction from phthisis—Morbid anatomy—Physical signs—Clinical symptoms and course—Diagnosis by concomitant tuberculosis, especially of the choroid—Ætiology—Prognosis.*

OF all diseases which attack adults, Phthisis or pulmonary Consumption is in our country, and in the temperate regions of Europe and America generally, by far the most fatal. It is estimated that a third of the deaths between fifteen and forty-five in England is due to this terrible disease.

From an early period in the history of medicine it has been known that progressive loss of flesh often accompanies cough, spitting of pus and blood, and other signs of disease of the lungs. This was distinguished as *phthisis pulmonum;* but the qualification was very early dropped, and in modern as well as ancient medicine phthisis or consumption means phthisis pulmonum, or pulmonary consumption. The term is now applied even to exceptional cases unattended with emaciation, but is never used when the lungs are believed to be healthy, even though wasting may be extreme.

There is, however, a secondary meaning of the word. Since it has been shown that most, if not all, cases of phthisis are tuberculous, such expressions as "renal phthisis," "intestinal phthisis," "laryngeal phthisis," have

* *Synonyms.*—Phthisis pulmonalis—Tabes pulmonum—Consumption of the lungs—Decline.—*Germ.* Lungenschwindsucht.—*Fr.* La phthisie. Φθίσις (from φθίνω, I waste = *tabes, consumptio*) occurs frequently in the Hippocratic writings, and there, as well as in those of Celsus and of Aretæus, means pulmonary phthisis. Celsus divides *Tabes* into Atrophy, Cachexia, and Phthisis. The earlier Greek physicians distinguished between Empyema, as suppuration outside, and Phthisis, as suppuration inside, the lung.

The following quotation from Celsus shows how good a clinical knowledge of the disease was formed by the Ancients:—"The third kind of decline (*tabes*) and by far the most dangerous is what the Greeks have named φθίσις. It usually arises from a cold in the head, whence it settles on the lungs, and there causes ulceration. There follows a slight feverish movement, which remits and comes again. There is a constant cough, raising of yellow matter, and sometimes of blood."

been introduced, to imply that the kidneys, the bowels, or the larynx present tuberculous lesions. But to make the clinical term phthisis synonymous with the histological term tuberculosis is most undesirable.

Pathology.—In proof of the extent to which opinions have differed as to the nature of phthisis, the words of Laennec in 1819 may be contrasted with those of Niemeyer in 1867. The former taught that "the existence of tubercles in the lungs is the cause, and constitutes the true anatomical character of consumption;" the latter, that in the majority of cases tubercles, if found in the lungs after death, "have been of recent origin, and have complicated the disease when it was already in an advanced stage." The older writer was, we may now affirm, undoubtedly right.

Phthisis one disease.—The appearances presented by the diseased lungs in cases of phthisis differ exceedingly; and its clinical symptoms and course are subject to no less wide variations. One cannot be surprised, therefore, that both pathologists and physicians have endeavoured to divide it into several diseases. Addison led the way in this direction by insisting that much of what was commonly regarded as tubercular disease in the lungs was in reality pneumonic, and that softening of the organ with excavation of its substance might occur without any tubercle being present; but he was very far from maintaining that an absolute distinction could be drawn. In his well-known essay, read before the Guy's Hospital Physical Society in 1845, he described first a "pneumonic" and then a "tuberculo-pneumonic phthisis;" and the final sentence of this work is, that "in every form of phthisis, inflammation constitutes the great instrument of destruction." Thus, after all, it may be said that Addison's teaching bore upon the question whether tubercles should be regarded as distinct from what Laennec used to term tubercular infiltration quite as much as upon the practical question whether a pneumonic kind of phthisis is to be recognised apart from the tubercular. Since his time other pathologists, particularly in Germany, have asserted in the most positive and dogmatic manner, that "catarrhal" or "caseous" pneumonia is the essential morbid change in many, if not in most, cases of phthisis. Another form which has also been declared non-tuberculous is the so-called "fibroid phthisis;" but there was never consensus of opinion among the most advanced pathologists with regard to these questions. Rindfleisch, who at one time maintained that the "tubercular granulations of Laennec" consisted in an inflammatory infiltration of the alveolar parenchyma round the smallest bronchi, afterwards admitted that they are true tubercles; and of late years the classical doctrine of Morton, Cullen, and Laennec is almost universally accepted, that phthisis is only one disease, and that it is always tubercular.

The varied appearances which may be found in the lungs after death depend mainly upon whether the tubercles and the tuberculous infiltration become caseous or undergo fibrous changes; and this, to a great extent, depends on the degree of rapidity with which the disease has advanced during life. Thus, pneumonic phthisis is generally equivalent to phthisis which has advanced quickly; fibroid phthisis to one of which the course has been very slow. No case of phthisis is without bronchitis and pleurisy, and few are without tubercular lesions elsewhere; but none are without the essential lesions of tubercle, catarrhal pneumonia, caseous degeneration, and more or less attempt at fibroid cicatrisation.

There is reason to believe that the tubercle bacillus (p. 312) is present in cases of phthisis of whatever form at some period of their course. In his earliest communication on the subject, Koch stated that he had found bacilli in twelve cases of caseous bronchitis with pneumonia; and these appear to have been all the examples of that variety of phthisis he had then examined. In fibroid phthisis one must look for them in the parts of the lungs most recently affected, and in the caseous form also they are generally limited to the edge of the infiltrated tissue. Sometimes nests of bacilli occur in the midst of infiltration. In tubercular vomicæ they are often present in great numbers. The little cheesy fragments, which are so commonly found in the cavities of phthisis, consist, according to Koch, almost entirely of masses of bacilli.*

Locality.—Phthisis affects both lungs. Clinically we constantly meet with early cases of consumption, in which the physical signs are confined to one side of the chest; but, as the disease advances, the opposite side becomes also involved. The evidence of autopsies proves that it is the rarest event for a patient to die of phthisis with one lung only affected. Nevertheless the disease is not perfectly symmetrical; it is almost always earlier and more advanced in one lung than in the other. It is much more symmetrical than pneumonia or pulmonary cirrhosis or pleurisy; rather more so than tubercular disease of the testes or adrenals, and less symmetrical than chronic tubal nephritis or than psoriasis.

It has long been known, both to physicians and to pathologists, that the upper parts of the lungs are almost invariably affected with phthisis, in whatever form, before the lower parts; and that in all but the most exceptional instances, the disease spreads downwards from apex to base, often with almost perfect regularity.

It is difficult to find a satisfactory explanation of this proclivity of the upper lobes of the lungs to phthisis. The same thing is observed in miliary tuberculosis, in which disease the pulmonary affection is believed to be due to an affection of the tissues at a number of different points through the blood-stream. This fact is opposed to Dr Hamilton's view, expressed in the 'Practitioner' for 1880, that the proclivity of the apices depends upon their being the driest parts of the lungs, so that caseation of catarrhal products is more apt to take place there than elsewhere. He also maintains that there is less expansion of the apices during breathing, and that catarrhal products are consequently more likely to accumulate in them than in other parts of the lungs. Almost exactly the same line of reasoning is adopted by Rindfleisch in 'Ziemssen's Handbuch.' He insists that the upright position of

* It is worthy of notice that, after speaking of the presence of the bacillus in the "Perlsucht" of cattle (cf. p. 315) Koch goes on to say that he detected it in cases in which there were round smooth-walled nodules filled with a cheesy pulp, such as are not generally reckoned to belong to Perlsucht, but to bronchiectasis. It would thus appear that pathologists have too narrowly defined tubercular diseases of the lungs, in animals as well as in man. But my own opinions with regard to phthisis have been based not so much upon microscopical investigations as upon the results of careful study of the appearances seen in the *post-mortem* room of Guy's Hospital during a long period of years. And I cannot help thinking that any unprejudiced observer would inevitably be driven to the same conclusion. Without wishing to detract from the importance of histological inquiries, I am under the impression that the practice of setting aside minute fragments of diseased organs for study at a future time, when the general morbid anatomy of the case has been forgotten, is very apt to lead to one-sided and partial views. What I have found is, that in the same body lesions which would be universally admitted as tubercular are associated inextricably with other lesions, of which the tubercular nature would by many pathologists be denied.—C. H. F., 1883.

the body in man and in the Quadrumana causes the weight of the shoulders and arms to fall upon the upper ribs, and so interferes with their play and leads to a deficiency in the movement of air in the apices as compared with that in the lower lobes. On the other hand, it is certain that the proclivity of the apices is no greater in men than in women, who use those parts of the lungs far more than men do. According to Dr Moxon, the regions which become the earliest seats of tubercle in persons who are confined to bed are the anterior edges; a fact which he explained by supposing that in a bedridden patient these parts are the most, not the least, active in respiration.

The general rule of the proclivity of the apex is liable to some other exceptions. In certain cases the tubercles appear a little lower down, leaving one or two cubic inches at the extreme summit of the upper lobe free, and often the seat of emphysema. Occasionally the middle of the organ is first affected, or even the lower lobe, the upper angle of which is frequently the seat of a vomica in ordinary phthisis. But the tubercular process seems never to spread upwards from the base of a lung into and through the upper lobe. What has sometimes been called "basal phthisis" is a distinct affection, which has been described above under the name of "chronic pneumonia" or cirrhosis of the lung (*supra*, p. 1007).

Phthisis tuberculous.—A common appearance is for the upper lobe to present a dense fibroid mass (perhaps containing more or less numerous cavities), while in the middle of the organ there are cheesy patches and in the lower lobe grey tubercles, scattered or in groups. Or, again, the affection in one lung may appear to be typically fibroid or typically pneumonic throughout; yet in the opposite lung, in which the disease is of more recent origin, there may be clusters of tubercles; and these may themselves be caseating, whatever the character of the change in the lung first affected. Lastly, the pulmonary lesion may appear to be pneumonic, or to be fibroid, not a single tubercle being discoverable, even in a state of caseation; and yet in some distant part of the body there may be tubercular lesions of the most characteristic kind in the ileum, or ulceration at the back of the vocal cords.

The following facts illustrate this point. In 1876 the author examined the body of a girl, aged sixteen, who died of what was regarded as pneumonic phthisis; in one kidney, traversing its cortex from the surface to the medulla, was a single linear tuberculous mass. In 1878, in a case of pneumonic phthisis, in which the affected part of the lung showed only a cheesy infiltration breaking down into sinous cavities without any distinct walls, there were not only small caseating points and ulcers in the intestine, but in the liver several tubercles as typical as possible. In 1876 the lungs of a child, aged six, presented a remarkable example of "fibroid phthisis;" in her intestine there were a large number of ulcers, with most abundant subserous tubercle. In 1879 a typical case of fibroid phthisis of the apices of the lungs showed indurated tubercles lower down; in the kidneys and the prostate of the same patient there were caseating vomicæ. In 1878, in a woman of thirty-three, the disease consisting mainly of grey induration of the pulmonary tissue, there being very little tendency to caseate, both adrenal bodies contained cheesy nodules, and there were yellow tubercles in the liver.

The way in which phthisis begins confirms the belief that it is from the

very outset tuberculous. The following are nine cases of very early phthisis found in persons dead from other causes. In 1881 a man, aged thirty, died of delirium tremens in Guy's Hospital; in the upper parts of both lungs there were miliary tubercles in groups. In the same year a man, aged thirty, died of caries of the spine with psoas abscess; in the apex of each lung there were grey translucent tubercles, some scattered, some in clusters, occupying only a square inch of the cut surface; the other parts of the lungs were quite free. In 1879 a youth of nineteen died of spinal disease, with a scrofulous kidney; in the apex of the right lung there was a single cluster of the most typical firm grey tubercles, none of which showed any tendency to caseate. In the same year a man, aged thirty, died of "sacro-iliac disease," which, although the result of injury, was accompanied with tuberculous affections of the prostate, kidney, spleen, and lymph-glands; the extreme apex of the right lung contained scattered grey miliary tubercles, without the slightest caseation, and with no induration of the surrounding pulmonary tissue. In 1877 a boy, aged ten, was killed by fracture of the spine; he appeared to have been strong and healthy, but in the apex of each lung there were miliary tubercles. These cases were observed by the author; and Dr Moxon's experience at Guy's Hospital was very similar. In 1869 a child, aged two years and three months, died of croup; in the left lung, below the apex, there were found several clusters of grey tubercles, one of them with caseous material in its centre. In 1867 a man, aged twenty-seven, was killed by accident, with fracture of the skull; at both apices, especially the right, there were recent miliary tubercles, in smaller or larger clusters. In the same year a man, aged twenty-two, died of typhus: in the right upper lobe there were many clusters of miliary tubercles, some already softening. In 1868 a woman, aged twenty-one, died after amputation of the thigh for disease of the knee-joint; in each apex there was early phthisis with clustered tubercles, some caseating; there was also a small vomica.

Even when the tuberculous character of an incipient pulmonary lesion might fairly be doubted, one may discover elsewhere morbid changes the nature of which is indisputable. Thus, in 1874, in examining the body of a girl, aged eighteen, who had died after excision of the knee-joint, the author found in the apex of the right lung a mass, the size of a marble, consisting of a cluster of yellow softening granules, which might naturally have been set down to catarrhal pneumonia; but the bronchial glands were caseating, and one of them contained the most typical grey tubercles.

Seat of the tubercles.—If we consider that there is scarcely a structure in the human body which is not liable to the growth of tubercles, we shall surely think it very improbable that in the lung their development should be limited to any one rather than another of the various tissues which make up the organ. Rindfleisch, however, maintained that the morbid process in phthisis begins definitely just where the bronchioles open into the alveoli, the earliest change being a "tuberculous infiltration of all the edges and processes" which exist at these points, and which contain muscular and elastic tissues, as well as fibrous. The occurrence of such a change at the extremities of several adjacent tubes, and its extension along the walls of the tubes themselves, would no doubt account satisfactorily for the "racemose" distribution of pulmonary tubercles on which Carswell used to insist, so that the phrase "Carswell's grapes" was invented to keep it in recollection. But his drawings illustrating this point are diagrammatic and accom-

modated. It is very seldom that one sees anything like a "peribronchial" distribution of clustered tubercles. And Dr Hamilton, in the 'Practitioner' for 1880, describes tubercle in the lung as generally beginning in a little cellular projection on one side of an alveolus, which afterwards becomes somewhat pedunculated and hangs into the alveolar cavity. When these alveoli lie adjacent to one another it may project into all of them at once. At first it pushes before it the epithelium and even the alveolar capillaries. But soon it breaks through and destroys the alveolar wall, so that a uniform rounded mass results, in which the outlines of the original air-vesicles are barely recognisable. The cells of the tubercle may, Dr Hamilton thinks, be derived either from the connective-tissue elements of the alveolar wall, or from the endothelium of certain of its capillaries, or both sources at once. Sometimes a tubercle sprouts from the inner coat of a branch of the pulmonary artery, starting perhaps from the endothelium, but soon involving the rest of the *tunica intima*, and almost occluding the channel of the vessel. Other tubercles lie in the course of the pulmonary lymphatic vessels contained in the periarterial and peribronchial sheaths, the interlobular septa and the deep layer of the pleura.

In some instances of phthisis, tubercles, scattered or in clusters, spread slowly through the lung, with little or no change in the intervening tissue. But, as a rule, this undergoes early consolidation, so that the tubercles come to be embedded in a more or less homogeneous mass. Sometimes, the substance of the lung is involved uniformly from the apex downwards, the edge of the consolidated area having a festooned outline, not unlike that of the border of a malignant new growth. Much more frequently, even when part of the upper lobe is universally affected, there are more or less numerous independent nodules of various shapes and sizes, lower down; and between and below these again, scattered tubercles may generally be seen in abundance. The character of the infiltrating material varies widely in different cases. It may be a soft, semitranslucent, pinkish substance, or a yellow friable tissue, the result of caseation. In other cases it is firm, dark, and tough, constituting one form of Addison's iron-grey induration, or it may have a "marbled" aspect, crossed by bands and seams of well-developed fibrous tissue, and darkened in all degrees of depth, up to perfect blackness. There are comparatively few cases in which caseation may not be found in some part of the lungs; and when the parts earliest affected are fibrous and of an iron-grey colour, those recently involved are often soft and yellow.

Vomicæ.—In all but very exceptional cases of phthisis the process of consolidation is followed more or less quickly by one of *ulceration*, leading to the formation of cavities technically called *vomicæ.** The tubercles no doubt soften in their centres, as they do elsewhere, and there is no reason why a vomica should not result from the breaking down of caseous material derived from tubercles alone; but, as a matter of fact, the formation of cavities involves the destruction of infiltrated lung-substance as well.†

* *Vomica*, a foul sore, an internal abscess: an old Latin word ('Liv. Hist.,' xxv, 72), applied by Celsus (lib. iv, cap. 8) to abscess of the liver as well as of the lung.

"Et phthisis et vomicæ putres."—Juv., xiii, 95.

† Sir Robert Carswell used to declare that sections of bronchial tubes with pus in their interior were frequently mistaken for softening tubercles. In this I feel confident he was wrong. In two or three exceptional instances I have thought, on first glancing at the cut surface of a lung, that I saw tubercles, when there were really only the open mouths of swollen tubes; but an instant afterwards I have perceived my error, because the slightest pressure below has made pus well up from them in large quantities.—C. H. F.

One characteristic appearance, which may be observed in various other organs besides the lungs, is the presence of a caseous zone of definite thickness between a vomica and the surrounding normal tissue. Such a zone is generally an indication that the disease is still spreading, and it is the chief means by which vomicæ increase in size. As they enlarge, cavities originally distinct are very apt to open into one another, and thus a single cavern of irregular form may be produced. Sometimes the destructive process remains limited by the lobar septum; sometimes this becomes ulcerated through, so that both the whole of the upper and a large part of the lower lobe may form one huge sac.

Sooner or later, if the patient should survive long enough, the further extension of a vomica becomes arrested. The indication of this change is that the interior of the cavity ceases to be rough and shaggy with adherent cheesy débris. A fibrous wall becomes developed, and its inner surface gradually assumes a smooth, polished appearance, exactly like that of a mucous membrane.

Such smooth-walled vomicæ are often crossed by fibrous bands or *trabeculæ*. Each consists of a mass of condensed pulmonary substance, with fibrous tissue that perhaps originally belonged to interlobular septa. In all probability some trabeculæ are remains of partitions that at one time separated vomicæ which have since coalesced. Others contain obliterated branches of the pulmonary artery; and sometimes several can be seen to spread away from a point situated on that side of the cavity which is nearest the root of the lung, so that their formation has obviously been the result of the resistance offered by the arterial walls to the process of ulceration. Ultimately the trabeculæ themselves give way, and their loose ends may then be seen hanging into the interior of the vomicæ. In very large cavities a bundle of such ruptured trabeculæ may sometimes be seen, the relation of which to the pulmonary artery is at once shown if a probe is passed into that vessel from the heart. Sometimes a pervious channel persists for some little distance in a trabecula, a fact which we shall see to be of clinical importance.

According to many observers of authority, certain smooth-walled cavities have an entirely different origin from that just assigned to them, being dilatations of bronchial tubes, instead of being formed by ulceration (cf. p. 973). Tubes do, indeed, almost invariably open into them more or less freely, the branches of the bronchial tree possessing no such power of resisting ulceration as belongs to the arteries. The idea of regarding the cavities in question as "bronchiectases" seems to have originated with Laennec. In all probability what first suggested it was the difficulty of understanding how a vomica formed by ulceration could acquire anything like a mucous membrane. But this goes for very little now that we know how readily such a structure can be pushed forwards over a raw surface from an edge of skin or of mucous membrane, as, for example, in the case of a rectal fistula. As a matter of fact, moreover, it is doubtful whether smooth-walled pulmonary cavities ever have a continuous epithelial lining. Dr Ewart, in his Gulstonian Lectures for 1882, says that this is wanting, except where there are "scattered islets of mucous membrane," the remains of "outlying bronchi intersected by the cavity wall."

Dr Hamilton, in the 'Practitioner' for 1879, declares that cavities, which cannot be dilated bronchial tubes, often have an epithelium "most typically columnar and ciliated;" and he explains the occurrence of bronchiectasis, and the sinuous and irregular outlines of the cavities which he

believes to be of such a nature, by referring it to the traction of bands of fibrous tissue radiating away from the sides of the cavity at different points. What seems to prove that the cavities in question are really vomicæ is that the earlier stage of the process of dilatation is never seen. If the view adopted by Dr Hamilton were correct, one ought, towards the margin of the affected part of the lung, to see tubes which could still be traced on to their extremities, but the sides of which were beginning to bulge out here and there. On the other hand, what one does commonly find are all possible transitional varieties between smooth-walled cavities and unmistakable vomicæ. The former are seen towards the apex, where the mischief is of oldest date; the latter lower down, where it is of more recent origin. Moreover, smooth-walled cavities often riddle the substance of a diseased lung in all directions, communicating freely with one another on every side, so that an ulcerative process must clearly have been concerned in their formation.

The contents of vomicæ vary widely in kind and in amount. When they are recent they often show masses of cheesy débris. Such masses, even if they are at first too large to pass out through a bronchial tube, probably crumble into fragments in the course of time, and are expectorated. Cavities of old date usually have pus in their interior. This of course implies that there is no very free communication with the bronchial tubes, and the fact is that in old vomicæ there is a tendency for the orifices of the tubes to contract until they become very narrow. One often finds that a tube of considerable size, into which a large catheter might be passed, has an opening into a cavity that will but just admit a probe. When such is the case, a turgid condition of its lining membrane may easily block it altogether.

It is remarkable how seldom the contents of phthisical cavities putrefy; they often have a faint, sickly odour, but they very rarely become fœtid, nor do they often undergo that peculiar acid fermentation which is so apt to arise in cases of chronic bronchitis with dilatation of the tubes (p. 975). When there is free escape of pus from vomicæ, their lining membrane may continually pour it out with large daily expectoration. But sometimes the walls are found at an autopsy perfectly dry, and the interior empty; in such cases there may be no expectoration whatever during life. In 1854 Dr Bristowe showed to the Pathological Society a specimen of such a quiescent cavity having adherent to its inner surface a soft, greenish, powdery mass of fungus, consisting of a branching mycelium, and of spores arranged upon rounded heads with thick stalks (*Aspergillus fumigatus*).

Adhesions.—A constant attendant upon phthisis is a local pleurisy, which leads to a gradual closure of the upper part of the serous space on the affected side. As a rule, these pleuritic adhesions are non-tubercular, and Rindfleisch insists on the extreme vascularity of the "false membranes" which unite the two surfaces, as contrasting with the deficiency of vessels in the substance of a fibroid lung. Not infrequently the adhesions are so thick and dense that it is impossible to remove the lung without free use of the knife.

Involution.—To complete the morbid anatomy of phthisis we have still to discuss the processes by which tuberculous lesions in the lungs become obsolete, so that they cease to threaten the patient's life, or even to impair his health; for tubercular inflammation is not an incurable process.

Relics of former mischief are frequently discovered in the lungs of persons who have died at various periods of life and of every kind of disease

or injury. In May, 1880, Dr Heitler, of Vienna, brought before the Medical Society of that city an analysis of all the cases of this kind that had been met with in a series of 16,562 autopsies between the years 1867 and 1879. Excluding all cases in which death was due to phthisis (among which there must of course have been many other instances of a previous attack of the same affection), he found that there were no fewer than 780 (or almost exactly 5 per cent.) in which obsolete tuberculous masses were present. Of the patients 503 were males, 277 females. The number of those who died of tuberculous affections of other organs was 101. A point of great interest is that the proportion of cases at different ages went on regularly increasing for each decennial period up to sixty years of age. Among persons aged from ten to twenty there were 12; from twenty to thirty, 105; from thirty to forty, 131; from forty to fifty, 156; from fifty to sixty, 157; from sixty to seventy, 36; from seventy to eighty, 153. It is true that no positive conclusion can be drawn from this fact, in the absence of information as to the proportion of persons at different ages in the total number of autopsies, but it is difficult to escape the inference that the time at which the pulmonary lesions were originally developed must, in a considerable number of instances, have been during adult life. In no fewer than 651 cases both lungs showed signs of past disease, though generally to an unequal extent; in sixty-eight the right lung was alone affected, in sixty-one the left.

There is a definite history of a former pulmonary affection in a case cited by Rindfleisch in 'Ziemssen's Handbuch.' It occurred in a man over fifty years of age, who died of enteric fever, fourteen years after having been treated in the same hospital for serious disease of the lung, attended with hæmoptysis and infiltration of the right upper lobe. He completely recovered, resumed his former occupation, and remained well until he took the fever a week before his death. The part of the lung that had been diseased was found to be indurated and shrunken, with surrounding emphysema and dilatation of bronchial tubes.

That such relics of long-passed pulmonary mischief belong to the same affection which, when it goes on and destroys life, is called phthisis, cannot be doubted. For their seat is in or near the apex of the lung, the affected part is more or less indurated, and it is often puckered on the surface or adherent to the chest-wall. On section it presents fibrous bands, or tough masses of fibrous tissue, parts of which are generally deeply pigmented, and in which there are often embedded cheesy or calcareous nodules. Kurlow has found by experiment that such obsolete tubercle is sometimes still infectious when injected, and reproduces tubercle in animals. The cheesy nodules may look very like gummata; they are enclosed in fibrous capsules; not infrequently they are gritty from the deposition of lime-salts in them, or this process may have gone on until they have become converted into hard, smooth calculi made up almost entirely of mineral constituents. In many instances one also observes grey or black indurated tubercles, long obsolete, and probably as old as the cheesy masses. When the fibrous bands have given rise to much puckering of the pulmonary tissue they often look very like cicatrices, and it has been thought that they represent former vomicæ which have undergone obliteration. Although there is no proof of this, there is no doubt that the walls of a cavity may shrink, so that in time it becomes reduced in size. Dr Theodore Williams has shown that this process of contraction of a vomica is often attended by shifting of its position. Unless its anterior surface is closely in contact with a firmly

adherent pleura, the more fixed part of its wall is that which contains the openings of bronchial tubes; consequently it often shrinks away from the front of the lung towards the root. Dr Ewart, in his 'Gulstonian Lectures' for 1882, gives diagrams showing that the pulmonary pleura, if not too extensively fixed by adhesions, may be drawn inwards over such a receding cavity until it forms a deep chink or fissure. The space created by the shrinking of a vomica may be filled up by the adjacent pulmonary tissue becoming emphysematous, the bullæ having been probably formed during inspiration, after the manner suggested by Dr Gairdner (p. 964). More frequently, however, the lower part of the upper lobe of the lung, or (in the case of the right lung) the fore-part of the middle lobe, is uniformly enlarged; if there is a considerable amount of contraction of one, the upper lobe of the opposite one may increase in size until it passes across the median line. Other organs at the same time undergo displacement. The liver or the stomach is dragged upwards, according as the right or the left lung is the one which is diseased; and the heart may be pulled over either to the right, or beyond its natural position to the left. Lastly, the upper ribs are drawn inwards, so that the chest-wall, especially below the clavicle, appears flattened or even hollowed.

Summary.—We may conclude our account of the anatomy of phthisis by saying that one of its characteristic marks is its multiformity. In every case there is more or less evidence of bronchitis, in every case more or less pleurisy, in every case discrete tubercles, grey or yellow, and infiltrating caseous tubercle. In every case there is cartarhal inflammation ("pneumonia"), with softening and destructive ulceration, causing more or less developed vomicæ; and in almost every case there is some attempt at repair, shown by fibrous induration, contraction, and cicatrisation.

On the other hand, pleuritic effusion is rare except occasionally in the earliest stage, and empyema is still rarer. True lobar fibrinous pneumonia is an infrequent and apparently an accidental complication; and neither abscess nor gangrene is met with. Pneumothorax sometimes occurs, and must always be remembered as a possible event. Emphysema, chiefly of the anterior edge of the lungs, is present in almost all chronic cases.

The anatomical lesions which most frequently accompany pulmonary phthisis are ulceration of the *ileum* and of the *larynx*. The latter has been already described (p. 894); the former will be noticed among the diseases of the intestines. Next in frequency comes tubercular pleurisy, then peritonitis, tubercular meningitis and tuberculosis of the spleen, liver, and kidneys, of the testes in men, and the Fallopian tubes in women.

Symptoms.—The clinical recognition of phthisis, as of pulmonary diseases in general, is based partly upon symptoms, partly upon physical signs. But there is no other disease in which diagnosis depends so completely upon the concurrence of the two kinds of evidence. Symptoms alone, when no signs can be detected, may justify a strong suspicion that phthisis is present; but, unless it is confirmed by their subsequent appearance, this suspicion never reaches certainty. On the other hand, when one discovers physical signs of the disease in a person whose health appears perfect—as sometimes happens, for example, in a candidate for life insurance—the inference is, that they depend upon a lesion which, although it was phthisical, yet is now obsolete, at least for the time. Probably physical signs never develop themselves in phthisis without symptoms having preceded them, although the patient

may fail to notice or may wilfully conceal them. A description of the symptoms of the disease will therefore best precede that of the signs.

The symptoms of phthisis fall into two groups. One group includes those which point directly to the lungs; the other those which concern other organs, or belong to the whole body. We will take the latter first.

Emaciation.—Of the general symptoms, one of the most important is progressive loss of flesh. This often occurs with extreme rapidity. Rühle mentions the case of a very bulky woman, who had weighed 240 lbs., and who lost 40 lbs. in the four weeks before she came under his care for hæmoptysis, at which time no physical signs of mischief in the lungs could be detected. Ultimately phthisical patients often lose a quarter or even a third of their weight. The explanation of the wasting is not always obvious. There is often great loss of appetite, and especially distaste for fat in every form, while in some cases vomiting is added to anorexia. But many patients who eat well and appear to digest what they eat, still lose flesh steadily. Nor does the wasting correspond to the degree of pyrexia or to the amount of sweating. There is no question that diminished appetite and imperfect assimilation of food, hectic fever and continued colliquative diarrhœa, profuse purulent expectoration and excessive sweating, each tend to produce loss of flesh; but it almost seems as if the growth of tubercle, like that of cancer, has a certain wasting effect, independently of diminution of the income or increase of the expenditure of the organism. Atrophy probably affects all the tissues more or less; but the heart becomes much less reduced in size in phthisis than in other wasting diseases, as, for example, cancer, probably because the right ventricle has increased work thrown upon it by the obstruction in the lungs.

With the emaciation there is often failure of strength and energy. The patient is no longer able to walk far without fatigue. The duties of the day tire him, so that he is glad to get home and to lie on the sofa until he goes to bed; and in the morning he gets up feeling weary and unfit for the day's work. But in many cases the mental activity and the muscular strength persist to a surprising degree, even in the advanced stages of the disease.

The *skin* is usually moist, sometimes greasy to the touch, but occasionally harsh and dry, with diminished sebaceous secretion—the state known as *xerodermia* or *pityriasis tabescentium.*

The growth of the *hair* is also changed. The straight lanky whiskers and beard of consumptive patients, and the long thin hair upon the chest, often suggest the nature of their disease.

Anæmia is often an early symptom of phthisis. The face becomes pale, the hands are white and transparent. In women scantiness or suppression of the catamenia may be one of the first indications that the health is failing. Œdema of the ankles often occurs as the disease advances, but it is seldom considerable unless there be venous thrombosis. It depends rather on anæmia than upon pulmonary obstruction; and there is never general dropsy or albuminuria, as in cases of chronic bronchitis.

Pyrexia.—When one is consulted by a person who has thus become thin and weak, the first thing to do is to ascertain whether the temperature is raised, particularly in the evening. One should notice whether the palms of the hands are hot, whether the cheeks are flushed and the pulse quickened, and whether there is unusual perspiration at night. The pyrexia of phthisis is altogether atypical, and in different cases it

varies widely in character and degree. It is scarcely ever altogether absent.*

In the most acute cases, which in Germany are called "phthisis florida," the pyrexia may be continuous throughout the twenty-four hours; the temperature may reach 104°, and never fall below 102°, unless profuse sweating should occur, when it usually falls one or two degrees Fahr. It is remarkable that, even when there is high fever, delirium and other cerebral symptoms are often absent; and it is quite an exception for the patient to pass into a "typhoid" condition, with stupor, sordes on the lips, and a dry, brown tongue. Moreover, phthisical patients often retain a much better appetite than would be present in other diseases with a like degree of pyrexia; nor is there generally much complaint of thirst.

Scarcely less acute is the course of other cases in which the daily range of the thermometer is very wide, the maximum perhaps reaching 103° or 104°, while the minimum may be 98·4°, or even lower still. Rühle says that the occurrence of a subnormal temperature, alternating with a high temperature at different periods of the day, is more unfavourable than when the fall is nearly to the normal point. Sometimes the patient experiences a slight rigor or a sensation of chilliness, and then passes through hot and sweating stages, very like those of a paroxysm of ague; and for this phthisis has actually been mistaken by a careless observer.

In other less severe cases the range of the temperature is comparatively slight; the thermometer may indicate 100° or 101° towards evening, but during the rest of the day it is perhaps scarcely, if at all, above the normal point. In the same patient there may be all possible variations in the thermometric readings. Even when pyrexia is generally present it sometimes happens that none can be detected during intervals of days or weeks. Of the cause of the differences in degree of pyrexia in different cases of phthisis no satisfactory account has yet been given. Dr Wilson Fox ('Med.-Chir. Transactions,' vol. lvi) thinks that it is generally proportioned to the extent of the intercurrent inflammation; but he admits that there are many exceptions. Lebert concluded, from an elaborate series of investigations, that the temperature is more influenced by individual idiosyncrasy than by anything else. This, of course, is no explanation at all.

Sweating comes on most during sleep, and some patients cannot doze for half an hour during the day without their clothes becoming saturated. A paroxysm of cough is sometimes the starting-point of such fits of perspiration.

Pulse.—The heart's action is nearly always accelerated in phthisis, and its rate is almost as valuable an indication of the activity of the disease as the temperature itself. Like the pyrexia, it is highest in the evening. It is apt to be much affected by slight exertion, and even by a change of position from sitting to standing. The frequent pulse is generally soft and feeble in quality. Sometimes its rapidity is out of proportion to the degree of fever, perhaps due to the nervous sensibility which forms so striking a feature of many cases of phthisis.

Vomiting is sometimes a conspicuous and early symptom of phthisis, and instances are known of grave error being committed in supposing that the

* Dr Theodore Williams, however, in vol. lviii of the 'Med.-Chir. Transactions,' says that in several of his cases in which active disease was going on in one or both lungs, no rise of temperature took place. And he gives details of an instance in which, although five observations were made every day for a week, the thermometer was never found above 99°.

patient's complaints were all due to disorder of the stomach. The suggestion was many years ago made by Mr Hilton that severe vomiting in phthisis may be due to irritation of the trunk of the pneumogastric nerve by tuberculous bronchial glands. In other cases this symptom is the direct result of a violent fit of coughing.

Diarrhœa is another symptom which often attracts attention in phthisis. It is often due to the presence of tubercular ulcers of the small intestine, or rather, perhaps, to a catarrhal state of the mucous membrane which precedes and accompanies such ulcers. Sometimes diarrhœa from this cause persists for many weeks before any physical signs of pulmonary disease can be detected. In advanced cases another cause of diarrhœa is the development of lardaceous disease in the intestinal mucous membrane.

A minor point, on which French writers have insisted, is the presence of a pink line on the gums close to the teeth. Whether it is seen more often in persons who are consumptive than in others is doubtful.

Aspect.—A phthisical patient often betrays the nature of his disease to the experienced physician at the first glance. Apart from the question (to be considered presently) of there being a special configuration indicative of a phthisical tendency, a bright eye, and a flushed cheek, associated with a pale face, wasted frame, slender fingers, and lank hair, at once suggest consumption.

It is a well-known peculiarity of consumptive patients that they generally remain hopeful throughout their illness, and, though they often suffer much, are as a rule cheerful and uncomplaining. This is in contrast with those who suffer from disease of the heart, but still more with patients who have chronic affections of the abdomen and particularly of the rectum.

Dyspnœa is a much less marked symptom in phthisis than might perhaps have been expected. The gradual onset of the disease and the loss of muscular power and of body-weight keeping pace with the destruction of pulmonary tissue, probably accounts for the fact that a patient, even with advanced phthisis, is often able to breathe quietly, and to carry on conversation with comfort, so long as he is sitting still. As Sir Thomas Watson observes, persons who fear, but will not believe, that they are consumptive, will fetch a deep breath, and bid us remark how thoroughly they can distend their lungs. But any effort or exertion is almost always attended with obvious hurry of breathing in patients who have passed beyond the earliest stage of the disease; and towards the last, orthopnœa is sometimes present in the most extreme degree, so that the patient gasps for breath, while his face and hands are livid and bathed in sweat.

It is no doubt as a consequence of obstruction to the pulmonary circulation that in the more chronic cases of phthisis we see clubbed finger-ends with incurved nails (*ungues adunci*). This affects the toes also, as it does in cases of chronic bronchitis, and of heart disease.

Pain is not commonly distressing or troublesome in cases of phthisis. There may be pain in the shoulder, or beneath the collar-bone, or lower down. But in many cases even this seems to be muscular rather than deeply seated. The pleurisy which invariably fixes the lung to the surrounding structures as the disease advances must be painless, for it is a constant complication. But pleurisy lower down, where there is more movement of the parietal upon the pulmonary layer, is less frequent, and is not uncommonly attended with sharp and piercing pain.

Cough and sputum.—In certain cases a *cough* is the earliest indication

of phthisis. At first it may be very slight, hardly more than a clearing of the throat; or it may occur only in the early morning, or after exertion during the day. It sometimes disappears for a time, to return later on. But ultimately it becomes more and more frequent, until it may cause great distress. It is when cough has been the first symptom noticed, that the disease is said to have arisen out of a "neglected cold."

A dry cough (*tussis sicca*, βῆξ κενή) is supposed to be characteristic of phthisis, hence the phrase "a dry phthisicky cough." After a time, however, there is more or less *expectoration* of a frothy fluid, watery or slightly viscid. The sputa may consist of a glairy greyish material, in which the microscope shows large round granular cells. As the local process advances, the expectoration becomes muco-purulent and occasionally streaked with blood; and it may ultimately be almost pure pus, or pus so intimately mixed with blood that it has a uniform brick-dust colour. In other cases the expectoration consists of round or disc-like pellets that remain distinct from one another in the mucus; they are called "nummular sputa," from their resemblance in size and shape to coins. If received into water they are seen to have a loose flocculent surface, as if they were portions of wool, or as if they had been "nibbled at," to use an expression of German writers. They consist of inspissated mucus or muco-pus, and contain no air, so that they fall rapidly to the bottom, unless they are held up by stringy mucus. They are probably formed in a vomica of some size, not in a narrow tube through which air was constantly passing backwards and forwards, and have become inspissated by absorption of their liquid constituents, until dislodged by a more than usually violent cough. Accordingly, the general opinion that nummular sputa are distinctive of phthisis is not without reason. But it must be remembered that the necessary conditions for their production are afforded by dilated bronchial tubes, as well as by pulmonary vomicæ. This is probably the explanation of a case mentioned by Sir Thomas Watson, in which he wrongly diagnosed phthisis when chronic bronchitis was the patient's disease.

Under the microscope, beside pus-corpuscles and epithelial cells, red blood-discs, elastic fibres and bacilli are the characteristic elements of the sputa in phthisis.

It has long been known that *fragments of pulmonary tissue* sometimes occur, in which the shape of the alveoli is still plainly visible. Dr Fenwick, in the 'Med.-Chir. Trans.' for 1866, showed that their detection is much facilitated by boiling the sputum with an equal part of a solution of caustic soda (gr. xv to ℥j). This dissolves the mucus in three or four minutes. The resulting liquid is then poured into a conical glass which is filled up with water, and the deposit which forms is carefully examined in a shallow cell. Dr Fenwick in one case found 800 fragments in the expectoration of twelve hours. He did not discover them in any case which was at so early a stage that there were no physical signs, but he often succeeded when no signs had yet pointed to ulceration or softening of the lung. The method is of great value in cases in which phthisis supervenes upon chronic bronchitis and emphysema, when the physical signs are apt to be ambiguous. At an advanced stage, when cavities are present, *elastic fibres* may always be found in the expectoration, even though the disease appears to be quiescent.

The detection of the *bacillus* of tubercle (cf. p. 312) in the sputum has now become an important means of diagnosing phthisis. There are many

methods of staining these organisms.* The following procedure devised by Neelsen has been found the most convenient in our wards. A minute quantity of the most purulent portion of the sputum is spread in a very thin layer upon the centre of a glass slide and thoroughly dried over a spirit lamp without charring. The slide is then immersed for five minutes in a beaker containing some carbolic fuchsine solution† which has been previously warmed to about 100° F., or till it begins to steam. The slide is next washed alternately in two beakers, one containing dilute sulphuric acid (20 p. c.) and the other tap water, until the colour, which speedily disappears from the preparation when dipped in the acid, does not return when it is placed in the water. By this means the fuchsine is washed from all parts of the sputum except from the bacilli of tubercle, which hold the stain with great tenacity. To produce a contrast-stain the slide is now immersed for two or three minutes in a concentrated alcoholic solution of methylene blue, washed rapidly in methylated spirit, and thoroughly dried over a lamp. The process is completed by placing a drop of Canada balsam on the sputum and covering with a thin cover-slip. The tubercle bacilli, stained red, are now easily seen in the blue field under a sixth objective, with a bright light concentrated by means of a substage condenser.

Hæmoptysis.—This important symptom is present whenever mucous or purulent sputa contain streaks of blood, or are uniformly discoloured by it as with the rusty sputum of lobar pneumonia. But in practice it is necessary to distinguish from these conditions the expectoration of pure blood liquid or frothy with air. In a large number of cases this occurrence is the first thing which suggests that there is anything wrong with a patient's lung, or indeed that he is otherwise than perfectly well. He perhaps feels a little tickling in the throat, and finds that his mouth contains a fluid which has a salt taste. He looks at his handkerchief, and is horrified to see that it is stained with blood. In some cases he brings up a large quantity at once and for the first time, but this is not the rule. Almost always hæmorrhage continues more or less for some hours, often recurring with each bout of coughing, and very often if unchecked by treatment will go on with more or less interval for several days.

From the days of Hippocrates it has been thought that the hæmoptysis is in such cases the *cause* of the consumption which ultimately develops itself; and two centuries ago Dr Richard Morton included a *phthisis ab hæmoptoë* among his species of that disease.‡ Recently the same doctrine has been revived by Niemeyer. But there is no sufficient evidence that

* See Dr Heneage Gibbes's paper in the 'Lancet' of August 5th, 1883; and Dr Klein's account of Koch's original method, with those of Ehrlich and Weigert, in his 'Micro-organisms and Diseases,' 3rd ed., p. 163; also a full account by Dr Crookshank in his 'Bacteriology' (1886), pp. 162—167. Like all aniline dyes, the colour is apt to fade, but if the slides are thoroughly washed and treated with nitric acid this may be overcome. Specimens still show perfectly well which were made seven or eight years ago.

† Fuchsine, 1 gramme; absolute alcohol, 10 c.c.; carbolic acid, 5 grammes; water, 100 c.c.

‡ "Hoc tamen perpetuo fere observare licet: quoties scilicet hæmoptoë præcedit, phthisin pulmonarem subsequi solere."—'Phthisiologia,' lib. iii, cap. v. He gives three illustrations.

Herodotus relates the following history of *phthisis ex hæmoptoë*, and it is probably the earliest on record. One of the generals of cavalry in the great host with which Xerxes invaded Europe B.C. 480, was Pharnuches, but he never crossed the Hellespont. For as the army was defiling out of Sardis a dog chanced to run under his horse's feet, and the horse, being frightened, reared and threw Pharnuches. After his fall he brought up blood, and the sickness ended in consumption (πεσὼν δὲ αἷμα ἤμεε καὶ ἐς φθίσιν περιῆλθε ἡ νοῦσος), lib. vii, cap. 88.

the extravasation of blood into a healthy lung is ever the starting-point of disease spreading through its substance and destroying it. Under various other conditions—as, for example, after injuries to the chest, and in chronic heart disease—we have frequent opportunities of observing the effects of pulmonary hæmorrhage and hæmoptysis; and few pathologists will assert that they have ever seen it give rise to phthisis. Nor does anatomical experience lead to the belief that blood extravasated into the air-passages is capable of being inhaled into the pulmonary tissue, and there forming solid nodules, as was maintained by Dr Reginald Thompson in the 'Med.-Chir. Transactions' for 1878. According to him they are most often found in three situations—in the upper lobe, in the axillary region, and towards the base, but not posteriorly,—which, he says, are notably those where inspiration produces the greatest expansion of the lungs. Cases are often met with in which inhalation of blood into the lung has obviously taken place; and what is observed is mottling of the cut surface of the organ with red or purple spots, impalpable, devoid of induration, and offering not the slightest resistance to the finger when passed over them. The formation of blocks or nodules of pulmonary apoplexy is a different process, and occurs only when a vessel is obstructed by an embolus. Hence it seems most probable that the congested cheesy bodies to which Dr Thompson refers are really relics, not of hæmorrhages, but of tubercular lesions.

A point mentioned by Niemeyer, to which a certain importance has since been attached, is that in one case, four weeks after an attack of hæmoptysis, he found a bronchial tube filled with adherent softening clot, giving it exactly the appearance of a vein obliterated by thrombus. A similar case has since been recorded by Dr Weber in vol. ii of the Clinical Society's 'Transactions.' In each instance the tube so affected was situated in the lower lobe of the lung. Now, as Traube remarked, such an appearance is so exceptional that very little significance can be attached to it. As a rule, unless a patient has actually been suffocated by hæmoptysis, one does not find any clots in the bronchial tubes after death. Sometimes a clot of considerable size, with branches that had evidently extended into a number of the bronchi, is expectorated a few days after an attack of pulmonary hæmorrhage. There is such a specimen in the museum of Guy's Hospital.

The other evidence brought forward by Niemeyer in support of the existence of *phthisis ab hæmoptoë* was mainly clinical. It consisted partly in the fact that hæmoptysis in patients who subsequently die of consumption often takes place at a time when no signs of mischief in the lungs can be detected on the most careful examination; partly in the fact that the hæmorrhage is frequently followed by fever, acceleration of the pulse, and signs of inflammation of the pulmonary tissue and of the pleura.

The first point is surely worth nothing. We shall presently see that auscultation and percussion frequently fail to reveal lesions which are really present in the lungs, if they happen to be situated deeply or to be scattered widely apart from one another. Take, for example, the case of a patient who is attacked with hæmoptysis, but who recovers from it completely without the subsequent development of any disease, so that the origin of the hæmorrhage remains a mystery. The author once had under his care a lady, about seventy years old, who on two successive occasions brought up several ounces of blood, but who got quite well afterwards, and at no time had any signs of mischief in the lung. The probability is that she really had a small cavity or other relic of former

phthisis, and that this was the seat of the hæmorrhage. Dr Weber has remarked that some of the patients who appear to get a *phthisis ab hæmoptoë* have had a tendency to epistaxis, and asks why blood should not come from the mucous membrane of the bronchi in such persons, as well as from that of the nose. But this appears unlikely.

It often happens that hæmoptysis is directly traceable to some violent effort or strain, such as rowing, running a race, or lifting a heavy cask; but of course that fact is quite compatible with the existence of disease in the lung at the time, and in most cases there is little doubt that early disease was present. Until we get a case of a presumably healthy young man, who has lately spat blood without other signs of phthisis, and has been killed by some accident shortly after, we cannot say that the lungs were in a healthy state. At present we admit hæmoptysis as a first symptom, but not as a first pathological event in the course of phthisis.

Niemeyer's other point was that hæmoptysis is often followed within two or three days by an increase in the temperature of the body and in the frequency of the pulse, and by signs of inflammation of the lung and pleura. Traube remarks, in reference to this, that none of the cases cited by Niemeyer show the absence of pyrexia at the time when the hæmorrhage occurred. But a chart given by Bäumler in vol. ii of the Clinical Society's 'Transactions' does show a rapid rise of temperature from the second morning after the commencement of the bleeding until the sixth day, when it reached 103·8°, and then a gradual fall until the eleventh day, when it became normal. And it must be in the experience of every clinical physician that such a febrile attack of variable duration is of frequent occurrence after an attack of hæmoptysis, and that before it subsides one can often make out distinct signs of consolidation of one apex, which were absent when it began. Still, Niemeyer's interpretation of these facts is not the most probable. It seems far more likely that the hæmoptysis is itself a direct effect of the development of tubercles in the pulmonary tissue. Hæmorrhage is no uncommon symptom of miliary tuberculosis of the lung, and may be immediately fatal at a time when there is neither ulceration nor obvious consolidation of the lung-substance, and when the only lesions found after death are recent miliary tubercles, which had apparently produced no other symptoms whatever. There is no doubt a difficulty in saying how the bleeding is brought about, but it seems very likely that the growth of tubercles in the walls of the alveoli may be attended with an invasion and softening of the coats of many of their capillaries, while at the same time the blood-pressure in them is augmented in consequence of compression of other capillaries. Rindfleisch, in 'Ziemssen's Handbuch,' gives a microscopical drawing, showing the coats of a minute artery actually perforated by a tubercular cell-growth.

In certain cases of phthisis, hæmoptysis is due to a very different cause, namely, to the rupture of the wall of a branch of pulmonary artery crossing the side of a vomica or enclosed in a trabecula. Rasmussen, of Copenhagen, first made known the fact that in many instances of this kind the hæmorrhage is preceded by an aneurysmal bulging of the coats of the vessel. A translation of his paper appeared in the 'Edinburgh Medical Journal' for November and December, 1868, and for August and September, 1869. Since that time the occurrence of such aneurysms in vomicæ has been noticed by many observers. In the 'Pathological Transactions' for 1871 Dr Douglas Powell tabulated a number of cases that had been inspected by him. We have had a specimen in a child under three

years of age. This instance is in itself sufficient to show that the formation of the aneurysm is not the result of atheroma, like that of an ordinary aortic or popliteal aneurysm. Rasmussen was inclined to attribute it to the unsupported state of the walls of the vessel when one side of it is exposed in a vomica. But Dr Powell points out that the coats are much swollen, semigelatinous, and glistening; and it therefore seems clear that their yielding to form a pouch depends on a previous inflammatory change, more or less like that which causes aneurysm in an artery occluded by an embolus. This, indeed, was Rokitansky's account of the lesions which precede large bleedings in phthisis, in opposition to Laennec's theory of diapedesis and Andral's of bronchial oozing. The size of an aneurysm in a vomica is commonly from that of a pea to that of a nut; but Dr Powell speaks of one which was as large as a Maltese orange. The vomica in which it is found is usually an old one with fibrous walls. The point of rupture is a little hole or fissure just large enough to admit a probe. Hæmorrhage may have recurred on several different occasions, at intervals of days or weeks, before the fatal issue. Indeed, death is not by any means always the direct result of an attack of bleeding, and the patient may sink exhausted after having ceased to spit any blood for several days; but in other cases he dies almost instantaneously, with a rush of blood from the mouth and nose; or he may be choked by the blood before it appears externally, so that the hæmorrhage is not suspected until an autopsy is made. On the whole, it is remarkable how rarely even profuse hæmoptysis is the immediate cause of death in phthisis.

Instances are not uncommon in which, without having formed an aneurysm, the branch of pulmonary artery from which fatal hæmorrhage had occurred is found to be perforated by a process of ulceration. At Guy's Hospital the one condition has been as frequent as the other.

Lastly, in some cases, even of advanced phthisis, in which the lungs contain many vomicæ, it is not possible, after the most careful research, to discover what has been the source of the hæmoptysis. No part of either lung may seem to be more deeply stained with blood than all the rest, even though death may have occurred almost immediately.

A point of some importance in regard to cases of ruptured aneurysm, or laceration of a branch of the pulmonary artery, is that the blood which is expectorated by the patient is usually of a bright red colour. For some writers have insisted that this appearance proves it to have been derived either from a bronchial artery or from a pulmonary vein; but it is extremely rare for blood from the lungs to be dark coloured.* In all probability the bright red, arterial appearance, which is usually seen depends not on the blood being derived from a vessel containing arterial blood, but on its having become aërated while it is in the bronchial tubes, where it certainly often is freely exposed to the air, as is shown by the frothy state in which it reaches the mouth. At any rate it is clear that in no case of hæmoptysis can the fact of the blood being bright red be taken as proving that it came from one kind of vessel in the lung rather than another.

* One such case is related by Niemeyer, in his 'Clinical Lectures. The patient had brought up enough blood to fill three basins within a few minutes; it was found to have a thin frothy layer on the surface but below this it was coagulated into a dark, almost black, cake. Anyone might, says Niemeyer, have supposed that it came from a profuse venesection. Probably the appearance depended on the known physiological fact that effused blood gradually becomes reduced below the surface, the oxyhæmoglobin yielding its oxygen to the white corpuscles.

A further point of great interest, on account of its bearing on the question of a *phthisis ab hæmoptoë*, is that in none of Rasmussen's cases of hæmorrhage from aneurysms of the pulmonary artery was any recent pneumonia found at the autopsy, even when the patient had lived for some weeks. If it should be found that no pyrexia develops itself when, in a patient previously free from fever, hæmoptysis results from ruptured aneurysm, or from laceration of a branch of pulmonary artery, it would give the last blow to Niemeyer's theory.

In almost all cases of hæmoptysis, if the bleeding should cease, there is for some little time afterwards a continuance of expectoration of a deeply blood-stained material—clotted blood, or mucus intimately mixed with blood. This is gradually found to alter in appearance, becoming reddish-brown, or brownish-black in colour. Such a change in it should be carefully noted, because it shows that the hæmorrhage is in reality no longer going on; and perhaps this may affect the treatment. But of course there is still reason to fear that fresh oozing may at any time occur.

Concretions.—When the tubercular process in a part of the lung has become quiescent, and calcification of some of the cheesy material has occurred, it not uncommonly happens that the patient ultimately spits up the concretions which are thus formed, and which may be of all sizes up to that of a pea. Sometimes their detachment from the tissues in which they had been embedded is attended with a little hæmorrhage, and Rühle seems to think that there must necessarily be at the time some fresh softening, so that a further advance of the disease may be anticipated. Indeed, that the expectoration of pulmonary concretions is unfavourable was long ago stated by Morgagni. But this occurrence in many cases has not been followed by any serious consequences. In one case it took place at a considerable interval of time after the subsidence of all active symptoms; and the patient in question is living at the present time. It must be remembered, too, that exactly similar concretions may come from the substance of a mediastinal gland having reached the trachea, or one of the bronchi by ulceration. A case in point occurred at Guy's Hospital in 1874; the man, who had been spitting up pieces of calcareous matter every two or three weeks, was admitted into the hospital and died there; and at the autopsy it was found that round the affected gland there was an abscess which had opened into the œsophagus as well as into the right bronchus.

Physical signs.—The examination of the chest in a case of phthisis reveals slow and progressive but usually imperfect consolidation, followed by excavation of the affected parts of the lungs. There are also the signs of concomitant bronchitis and pleurisy.

(1) *Initial stages.*—At the commencement of the disease the signs may be very slight and doubtful, and repeated examinations at intervals of some days or even two or three weeks, may be required, before one ventures to express a positive opinion as to whether mischief is developing or not.

Among the earliest changes to be detected is diminished mobility of the upper part of the chest on one side. Standing behind the patient, with one hand placed lightly below each of his clavicles, the physician can feel that the expansion of the two sides is unequal; one lags slightly behind the other, or one stops in its movements while the other continues to rise.

On percussing with great care, and comparing closely corresponding regions of the chest, he may make out that there is more or less deficiency of

resonance, either in front or behind. A good plan is gently to flick the two clavicles in turn with the finger; the resulting "osteal" sound may then be mixed with unequal degrees of pulmonary resonance on the two sides. It is important to examine the spaces above the clavicles as well as those below them; and by employing different amounts of force in succession one may sometimes find that a particular kind of stroke elicits an impairment of resonance better than others. The suprascapular regions must also be carefully percussed; deep percussion is required to bring out differences of sound here.

On auscultation it may be found that the vesicular murmur is not alike on the two sides. If over one apex it is permanently deficient or even absent there can be no doubt that that is the lung which is affected. In other cases the presence of early phthisis causes the vesicular murmur to be coarser than natural. It is then difficult to determine by auscultation alone which of the two lungs is most likely to be the seat of disease, for an abnormally loud vesicular murmur, instead of indicating a lesion where it is heard, may be compensatory of one on the opposite side. Various modifications in the character of the vesicular murmur may indicate early phthisis. Inspiration may be harsh in quality, expiration may be too distinct and prolonged, or there may be a slight pause between the two.

Again, the inspiratory sound may be interrupted, in accordance with irregularities in the play of the chest walls. These interruptions may be so frequent that the inspiratory murmur has been compared with the sound produced by a revolving cog-wheel (*respiration saccadée*). This must not be taken as necessarily showing that disease is present, for Dr Walshe has "observed it at one or both apices when free from consolidation of any kind." In one of his cases the cog-wheel rhythm was probably due to the action upon the healthy lung of an irritable heart; for the separate sounds which make up cog-wheel breathing may be synchronous with as many cardiac pulsations, as pointed out in the 'Revue mensuelle,' 1877. It seems likely that when a portion of the lung is partially solidified the shock given to it by the beating of the heart, whether directly or through the blood-vessels, may produce a greater effect than on the normal lung-tissue.

One of the signs of tubercular disease of the anterior edge of the lung is an increased loudness of the cardiac sounds in the corresponding subclavian region. In some cases, too, there is heard over the pulmonary artery a systolic murmur. In later stages it may be due to traction by adhesions, or to anæmia, but its early occurrence is not easy to explain.

A somewhat later sign of phthisis is the presence of non-consonating moist sounds at the affected apex. In some cases they are audible only just after the patient has coughed. If limited to the upper lobe, moist sounds are of special diagnostic significance, since a primary bronchial catarrh is probably never thus localised.

(2) *Consolidation.*—No doubt a considerable amount of consolidation may take place in the apex of a lung without any physical signs being audible beyond those which are mentioned in the last paragraph; but as the process of solidification goes further dulness becomes more obvious, and the respiratory murmur acquires the bronchial character. At the same time the voice is transmitted by the stethoscope with increased loudness, constituting bronchophony. These signs are most important; but it is essential to bear in mind what was stated at pp. 934 and 938 of the normal presence of bronchial breathing in certain regions of the chest. Moreover, throughout the right suprascapular, supraclavicular, and subclavicular regions the

voice may in healthy persons be heard more loudly than in the corresponding left regions.

Moist sounds may or may not accompany the bronchial breathing of phthisical consolidation. When present they usually have a consonating character. A very common combination is for the inspiration to be attended with râles, so that no bronchial breathing is noticeable, whereas immediately afterwards a blowing expiratory sound is heard, but no râles.

From an early period of the disease the regions above and below the clavicle on the affected side are commonly found to be flattened or slightly hollowed. This is partly due to wasting of the pectoral muscles, and afterwards to shrinking of the apices.

(3) *Excavation.*—The quality of the bronchial breathing in a case of phthisis may become more pure, more blowing, more tubular, without there being any further change in the affected part of the lung than increasing consolidation; and, conversely, excavation may take place to a considerable extent without the bronchial breathing changing in quality. But one is scarcely ever wrong in diagnosing the presence of a vomica where we hear well-marked amphoric breathing.

It is not infrequent for a phthisical cavity to become so large that the recognition of its physical signs is the key to the diagnosis of the case. One might perhaps have anticipated that the percussion-sound over a large empty cavity would become resonant again, or even tympanitic. It has, however, long been known that this is not so; the thick adherent pleura and the condensed lung-tissue round the wall of a vomica serve effectually to check the vibrations of the thoracic parietes, so that a toneless noise always forms a large part of the sound which is elicited on percussion; but mixed with this are tones of varying quality, due to the vibration of the air within the vomica itself, and thus any of the modifications enumerated at p. 930 may result.

A frequent peculiarity of the percussion-sound over a large vomica is that it resembles the noise produced by striking coins together, or by striking over one's knees the hands loosely clasped. Laennec called such a percussion-sound the *bruit de pot fêlé*, or, in English, "cracked-jar sound" (p. 930). For its production in a perfect form the walls of the cavity itself and the thoracic parietes must be elastic and yielding, the percussion-stroke must be somewhat heavy, and the cavity must communicate freely with the bronchial tubes, and these again with the external air through the open mouth. The reason is that the *bruit de pot fêlé* depends upon the expulsion of air from the cavity, just as in striking the hands over the knee one drives air out through a chink between them.* There are, however, other diseased conditions in which this curious percussion-sound may occur. Thus, according to Dr Gee, it is sometimes obtained over the upper part of the front of the chest in cases of pleuritic effusion, sometimes over islets of unsolidified lung embedded in tissue hepatised in acute pneumonia, sometimes in cases of malignant tumour. In healthy children also it may sometimes occur from a sudden percussion-stroke driving in the yielding parietes of the chest. But, along with other signs of phthisis, it is almost a certain proof of a large empty cavity in the lung.

Precisely similar in its mode of origin to the *bruit de pot fêlé* is a pheno-

* The cracked-pot sound was exquisitely marked in a patient who had, outside the thorax, beneath the pectoral muscles, an abscess-cavity which contained air, and which communicated with a pneumothorax by a narrow hole through the intercostal muscles.—C. H. F.

menon which sometimes attracts the notice of the patient himself as well as of other persons, namely, the transmission of the heart-sounds outwards, so that they can be heard, like the ticking of a watch, at a distance of several feet.* This singular phenomenon would doubtless be much less rare than it is, were it not that a cavity of sufficient size to have a quantity of air driven out of it by each pulsation of the heart very seldom exists, except in the upper lobe.

On auscultation over a large vomica one may obtain any modification of bronchial breathing up to the amphoric. Another modification, which appears to be heard only when a cavity has been formed, has recently received from Seitz the name of *metamorphosing murmur*.† It is probably not very different from what Laennec long ago described somewhat vaguely as the *souffle voilé*. It is said to be characterised at the commencement of inspiration by an unusually harsh sound, which lasts only during one third of the inspiratory period, and gives place during the remaining two thirds to bronchial breathing accompanied by a metallic echo, or to ordinary râles. The moist sounds are often "large" enough to be called gurgling.

All kinds of *metallic phenomena* may present themselves in a very large vomica, exactly as when there is pneumothorax (cf. p. 1048).

Vocal resonance often amounts to pectoriloquy. On the other hand, Dr Walshe insists on the fact that over a large cavity, at least at its upper part, there *may be dead silence*, both respiratory and vocal.

One very rare effect of excavation of the lung is the production of subcutaneous emphysema. A case in point occurred at Guy's Hospital in 1882. The patient had been slowly sinking for weeks, and shortly before his death there was a slight crackling below the clavicle and at the root of the neck. Fräntzel refers to similar instances. As pneumothorax is not present, it must be assumed that ulceration extends through both layers of the pleura, the space between having been previously closed by adhesions.

Involution.—The physical signs of quiescent or retrogressive phthisis vary widely in different cases. Shrinking of the upper part of the chest may go on until the clavicle and shoulder are obviously drawn down. The cyrtometer will show the diseased side to be much smaller than the sound. The percussion-sound is usually very dull; indeed, as Rühle remarks, extreme dulness in phthisis is usually a favourable sign, for it means that dense fibrous cicatricial tissue has taken the place of patches of tubercular pneumonia.

The heart becomes uncovered by retraction of the lung, especially if the left is the one affected. Its impulse may be seen and felt over a much more extensive area than is naturally the case, even as high as the third or the second intercostal space. The stomach also may be drawn upwards to the level of the sixth or the fifth rib. On the other hand, if the right lung is diseased, the heart's apex may be displaced to the right side of the sternum, and the liver may be dragged up as high as the fourth rib.

Extension.—In all cases of phthisis it is important that, while watching the changes that take place in the part first affected, we should also be on the look-out for signs of extension to other parts, or to the opposite lung.

* Many years ago my father showed me a case of this kind which had come under his observation. The sounds were sometimes audible across a good-sized room, but I found that when the patient, a young woman, was made to close her mouth, I could instantly stop them by pressing together her nostrils. Just such a case was brought under the notice of the Clinical Society in 1880, by Dr Frederick Taylor.—C. H. F.

† See von Niemeyer's essay on 'Phthisis' (New Syd. Soc. transl.), p. 54, note.

The frequency of excavation in the apex of the lower lobe makes it advisable to listen carefully on a level with the shoulder-blade, after it has been drawn outwards by the patient crossing his arms.

In advanced cases the question is often not what parts of the lungs are diseased, but what parts remain capable of carrying on respiration. It is surprising to how small an area, at the extreme base of one lung, one may find the presence of a vesicular murmur restricted. Here it will probably be exceedingly loud, an example of compensatory "puerile" breathing.

One must not over-estimate the significance of crepitations and râles when heard over the whole of the back of a lung, for they do not prove that the corresponding lung substance contains more than scattered or clustered tubercles. In cases of acute and general miliary tuberculosis it has often appeared during life that large tracts of the pulmonary tissue were breaking up, and yet after death the pulmonary tissue between the tubercles has been found still crepitant.

But in the majority of cases of phthisis the discrepancy between physical signs and *post-mortem* appearances is in the opposite direction. Clinically disease is perhaps discovered in the upper lobe of one lung; the autopsy shows that nearly the whole of that lung is affected, and also the upper lobe of the other lung. This cannot always be explained by the extension of the mischief in the interval that may have elapsed. We must frankly recognise the fact that the presence of well-marked disease in one apex adds greatly to the difficulty of detecting early mischief in the other apex. The reason obviously is that one has lost the standard of comparison on which one is accustomed to rely.

On the whole, where there are well-marked signs of advanced phthisis in one lung we may be pretty sure that there is early phthisis in the other. It is extremely rare to find vomicæ on one side and no tubercles on the other. This was, however, the case in a young mulatto, who died under the writer's care after an unusually rapid course of pneumonic phthisis. One lung was full of tubercles, the other was only œdematous.

Diagnosis.—The recognition of phthisis, if based as it should be upon symptoms as well as signs, is often very easy. But there are cases in which there is the greatest difficulty in arriving at a right conclusion, and in which, indeed, the only safe course is to reserve one's opinion for a time. The diagnosis between phthisis and other pulmonary affections, particularly chronic pneumonia, has been already referred to (cf. pp. 953, 1010, 1013). In practice the doubtful cases are generally those in which physical signs are either wanting or at least slight and obscure, so that one hesitates as to whether the disease is pulmonary, or whether there is not rather some deeply seated new growth, or some lesion of the internal lymph-glands, or of the thoracic duct, or of the adrenals, by which the patient is wasted and worn down. Often, although the nature of the affection cannot be determined, it is clear that he is stricken by fatal disease of some kind.

But more often the prognosis absolutely depends upon the opinion one may form. The doubt is generally whether the patient, if a man, is suffering from the syphilitic cachexia, or is the victim of hypochondriasis and aggravated dyspepsia; if a woman, whether she is only hysterical. In all cases of this kind the thermometer is of the greatest value. One hysterical affection, which has often been mistaken for phthisis—the "anorexia nervosa" of Sir William Gull—has been already described (p. 822).

In other cases the suspicion of pulmonary disease is based mainly upon the fact that the girl, as is said, "spits blood." A glance at the sputum is sometimes sufficient to remove all uneasiness about this. What is expectorated may be found to be a rather slimy liquid, uniformly tinged of a pink or purple colour, so that it looks like plum juice. It is, in fact, saliva or mucus from the mouth, and the blood comes from the vessels of the mucous membrane. Rühle remarks that this sort of hæmorrhage often occurs in the night, from the patient making sucking movements of the lips and cheeks during sleep; and thus the pillow may show stains of blood, the origin of which seems at first to be inexplicable.

Another variety of sanguineous expectoration is due to the rupture of small vessels at the back of the fauces during violent coughing, or "hawking up" of phlegm. Varicose veins in the pharynx sometimes reveal the source of hæmorrhage.

Hæmoptysis sometimes occurs in elderly persons, not from phthisis or aneurysm, but from degenerated vessels (whether bronchial or pulmonary is not ascertained). This occurred in the writer's knowledge a few years ago, and almost at the same time in two eminent physicians; and it was the subject of a lecture before the Medical Society by Sir Andrew Clark. The condition is clinically allied to that which leads to cerebral hæmorrhage, epistaxis, and hæmaturia; but it is not known whether the vessels are atheromatous or the seat of arterio-capillary degeneration.

We must first make sure that hæmoptysis is really blood from the lungs. Hysterical girls and more responsible persons will sometimes simulate this symptom, and as their own blood is most convenient for the purpose, the microscope will not detect the fraud.

Blood may be spit out mixed with mucus, which has come from the gums, the tongue, the tonsils, or pharynx, or from the larynx and trachea.

True hæmoptysis may occur as a symptom of cardiac disease, of purpura, or any of the severe forms of anæmia; and also as the result not only of fractured ribs and penetrating wounds of the chest, but also of compression of the ribs without their being broken.

Lastly, hæmoptysis, like so many other hæmorrhages, has been supposed by some observers to be frequently *vicarious* of the catamenial function. Sir Thomas Watson, for example, says that this is not at all uncommon, and that it is not usually attended with any peril to life. He cites a case which was observed by Pinel at the Salpêtrière, that home of all that is marvellous in disease, in which a woman was said to have menstruated through her lungs from the age of sixteen to that of fifty-eight, often to the extent of two quarts of blood during a period of two days, while she nevertheless remained plump and healthy. A very different view of this question is taken by Rühle, who will only admit that in patients who already have lung disease suppression of the catamenia (or of a hæmorrhoidal flux) may be followed by vicarious hæmoptysis. He speaks of having seen cases in which this recurred at intervals of from four to six weeks, until a few leeches were applied to the anus with a corresponding regularity. Vicarious hæmoptysis, if it occurs at all, is so rare that we should look with great suspicion on a supposed case of this condition.

Dr Walshe, insisting on the difficulty of diagnosis in early cases of phthisis, advises little weight to be laid on differences in vocal resonance, particularly at the right apex in women, or on harsh or jerking inspiration

at the same spot; and urges the propriety of suspending judgment until a second examination of the chest has been made.

Cases in which in the writer's experience phthisis has been wrongly diagnosed have been apical pneumonia, pulmonary syphilis, and empyema in children. Those in which it has been overlooked have been mistaken for enteric fever of unusually protracted course, bronchitis in elderly people, tubercular meningitis, or idiopathic anæmia.

Cardiac disease in children, gastric ulceration, and chlorosis often simulate phthisis in aspect, but can be distinguished by careful and repeated physical examination.

Course and events.—Phthisis varies greatly in the rapidity of its progress, but its duration is almost always for many months, and sometimes for many years. Trousseau used to say that the only *phthisie galopante* is miliary tuberculosis of the lungs.

The most rapid case of true phthisis on record is probably one related by Traube in the 'Berliner klin. Wochenschrift' for 1867. A man, aged twenty-eight, died, after only thirteen days' illness, of "acute tubercular (caseous) pneumonia." The attack began with rigors and pyrexia; a few days later hæmoptysis set in and continued. At the autopsy all parts of the left lung presented patches of lobular hepatisation, the centres of which were caseating, especially in the upper lobe; a similar affection in an earlier stage existed in the right lung, and both apices showed traces of old lesions. Eight cases have occurred in Guy's Hospital, in each of which there was a definite history that the duration of the patient's illness, from its commencement to its fatal termination, was only from five to twelve weeks. In two instances the attack was attributed definitely to a chill: one man said that he got wet through while working in a potato-field, after which he shivered and became hot, and was never well again; the other, that on a particular occasion he slept with his window open. In almost every one of these cases vomicæ had formed before death, especially in the upper lobes, in the centres of the cheesy masses, which formed the most conspicuous lesions observed at the autopsy.*

The sudden commencement of some of the rapidly fatal cases of phthisis is of great importance in regard to their diagnosis from cases of acute pneumonia of the apex; for the most serious errors of diagnosis have been made between the two diseases. The mode of onset usually affords a means of arriving at a right judgment, but it is evident that this is not always the case; and the only point on which one can fall back seems to be one to which Traube has drawn attention, namely, that in acute phthisis bronchial breathing is not discoverable until much later than in pneumonia of the upper lobe—not until the end of the second week, or even for a longer time still.

If one finds very extensive consolidation in a case of phthisis when it first comes under one's observation, one should always think of the possibility that it may, in part at least, be the result of intercurrent pneumonia, and therefore that the prognosis may be far less grave than it otherwise would have been. For it is a striking fact that when fibrinous pneumonia

* It must, however, be borne in mind that the distinction from miliary tuberculosis may, sometimes at least, be not very apparent. The dissemination of the tubercular bacilli by the blood-current may, if the tubercles to which it gives rise in the lungs are not very numerous, have no apparent effect until they in their turn become starting-points of a local infection, when a disease exactly like ordinary phthisis may arise.

occurs in a person who already has phthisis it often seems to run as favourable a course as if it had arisen in one who was healthy.

But even acute phthisis—"phthisis florida," as German writers term it—may, instead of going straight on to a fatal termination, become arrested, and afterwards run a chronic course. Rühle relates a case in a girl, who seemed to have but a short time to live when she was transferred to his charge from that of Niemeyer, his predecessor at Greifswald. Yet her symptoms subsided, she was discharged from the hospital with signs of a cavity in the left upper lobe, and did not die until the following year, having in the meantime given birth to a child. Pregnancy possibly checked the progress of the disease.

The course of ordinary chronic cases is, almost without exception, interrupted by intervals during which the patient may seem to regain his health. Cough may almost disappear; even the evening temperature becomes normal from day to day, the appetite returns, the face is no longer pale, and the ordinary weight is regained. It is of course true that this favourable change commonly takes place under medical advice, and we shall presently see how important it is that the advice should be well carried out. But sometimes it occurs even in those persons who have gone on working in spite of their illness.*

Duration.—Different observers have made widely different estimates as to the duration of phthisis. Laennec, Andral, Bayle, and Louis each put the "mean duration" at about two years. Dr Austin Flint, in America, found that, excluding acute tuberculosis, the average duration of phthisis was thirty-three months. These estimates apply to the first thirty years of the century. Dr Pollock, analysing 3566 cases observed by him at the Brompton Hospital, found that the "average duration" of these cases while under observation was more than two years and a half, and in the course of that time only 127 ended fatally. What was the real average length of the disease among the whole number of cases he could not tell, but it must clearly have been much longer. It is, however, very difficult to believe that Dr Pollock's cases fairly represent the ordinary course of the disease. There must have been an undue proportion of exceedingly chronic cases, and cases running a rapid course must in some way have been excluded. Still more extraordinary are the statements made in vol. liv of the 'Med.-Chir. Transactions' with regard to the duration of life among 1000 cases of phthisis seen by Dr C. J. B. Williams in private practice between 1842 and 1864. Of the patients in question 198 were known to have died; in them the average duration of the disease was nearly seven years and three quarters. In the remaining 802 patients who were alive when last heard of, its average duration had already been more than eight years. Among these cases, however, none were included which had not been at least one year under observation; and this restriction, besides keeping out of the list

* In January, 1874, a hatter, aged thirty-seven, who said that he had been ailing for six months, came to me with signs of phthisis at both apices. His morning temperature was 101·4°. His father had died of consumption. Notwithstanding my urgent advice to give up work, he did not rest for a single day. The only difference he made was that instead of living away from his workshop in the Borough, so that he was exposed to changes of temperature in going backwards and forwards, he now slept in the same building. For a week or two the physical signs increased, moist sounds becoming audible all over the left lung. But his symptoms quickly improved, and by the end of May he was as stout as ever, and said that he felt nearly well. The signs at the apices, however, still remained. In the following year I heard that he was in good health, with only a little occasional cough. But in 1879 his symptoms returned, and he died in September, 1881.—C. H. F.

all rapidly fatal cases, doubtless weeded it of most who failed to improve for a time under the treatment recommended, and therefore ceased to attend.

That life is sometimes maintained for a great length of time after phthisis has developed itself has long been well known. Sir Thomas Watson alludes to a patient of Dr Gregory's who was at least seventy-two years old when he died, and who from the age of eighteen had never been free from symptoms, "being often hectic, and frequently spitting blood."

The writer has a patient who furnishes almost a parallel history. He comes of a very phthisical family, and developed signs of the disease when under twenty years of age. He then went a voyage to Australia for his health. After repeated hæmoptysis, he recovered enough to marry at the age of twenty-five, and in 1875 came under the writer's care. There were the signs of consolidation of one apex, and of less advanced disease of the other lung. He has again and again spent the winters at Bournemouth, at Algiers, and in Egypt, again and again has had attacks of pyrexia, bronchitis, or hæmoptysis, but he has survived them all, has become stout, and now at fifty is a grandfather, and considers himself out of the physician's hands.

Immediate causes of death.—The fatal termination of phthisis is sometimes sudden and unexpected. In 1866 a gentleman aged twenty-six, who had long been ill, went up to London from Brighton one day to transact some business. At the London Bridge station he was seized with alarming symptoms, and was taken down to Guy's Hospital, where he died within a quarter of an hour from the beginning of the attack. In 1868 a labouring man aged twenty-three, who had been indisposed for some time, was at his usual work in the Borough, when about 2 p.m. he began to suffer from dyspnœa; this rapidly got worse, and he was carried to the hospital and died in two hours. In neither case did the autopsy show why death should have occurred at that particular time.

Pneumothorax often brings more or less immediate danger to life. Nevertheless, when the immediate effects are got over, the consequent collapse of the lung appears to be unfavourable to rapid progress of the disease; so that if the other lung is but slightly affected the patient's condition may, at least for a time, improve. Some practitioners, acting on this hint, have ventured to puncture the pleura and thus produce pneumothorax, with the hope of checking severe hæmorrhage.

An accident, happily rare, that may bring phthisis to a sudden close is pulmonary *embolism*, resulting from thrombosis of the femoral vein.

Syncope is sometimes the cause of death; or it may arise from sudden exhaustion of the respiratory centre. Indeed, it is not very uncommon for consumptive patients to be found unexpectedly dead in bed.

In other instances phthisis ends fatally by the supervention of tubercular disease elsewhere than in the lungs, by *tubercular meningitis* or peritonitis, by tubercular pyelitis, or by solitary tubercle of the brain.

It is a remarkable fact that *hæmoptysis* is very rarely the immediate cause of death in phthisis. Considering that it is present in more than four fifths of consumptive cases, it is exceptional to find a patient "choked in his own blood," although every practitioner has seen that event.

The most serious symptom towards the last may be *diarrhœa* resulting from tuberculous ulceration of the intestine, or the *dysphagia* and the other distressing symptoms produced by a like affection of the larynx.

The following non-tuberculous affections are not infrequently fatal

complications of phthisis: parenchymatous nephritis, typhlitis with peritonitis, and abscess of the brain.

The coincidence of *fistula in ano* with phthisis is one which requires brief mention. Dr Pollock points out that it occurs far more often in males than in females, and most commonly in persons who are no longer young, the most frequent age for it being from thirty-five to forty-five. The disease of the lungs has very generally advanced to the formation of vomicæ before the fistula appears. Many observers, including Dr Pollock, are of opinion that in such cases no operation should be attempted; for, when it is successful, the phthisis is very apt to assume increased activity two or three months later. But there seems to be no evidence that the cure of a fistula in a person not already consumptive renders him more liable than before to the supervention of pulmonary disease. In such cases the fistula is probably not tubercular in origin.

Finally, *lardaceous degeneration* plays a very prominent part in bringing to a close many cases of phthisis. If the intestines be involved, an intractable diarrhœa may result, which cannot be distinguished during life from that of tuberculous ulceration. But it is chiefly by affecting the kidneys that this kind of degeneration acquires its clinical importance. General dropsy sets in, and the patient acquires more or less of the appearance usual with those who are affected with Bright's disease. Indeed, tubal nephritis sometimes comes on in phthisis without there being any lardaceous change discoverable in the renal glomeruli or vessels even with the microscope. It is, therefore, not safe to diagnose a lardaceous affection of the kidneys from the mere fact that the patient has albuminuria. According to observations made by Dr Theodore Williams, recorded in a paper read before the Royal Medical and Chirurgical Society in 1882, the occurrence of albuminuria in phthisis has the effect of masking the other symptoms, and especially of making the range of temperature lower.

Cases of recovery.—Phthisis was until lately regarded as certainly fatal; we now know that this is not the case, partly through recognising by help of the stethoscope its existence and early stages, partly through the knowledge derived from morbid anatomy.

Again and again we find in the deadhouse traces of obsolete phthisis in adhesions, cicatrices, and calcareous deposits which occupy the apices of one or both lungs in the bodies of those who have died of some acute and independent injury or disease (cf. p. 1061). Such persons must have suffered from phthisical symptoms at some time of their lives.

Hospital practice gives too unfavourable an estimate of the prospects of phthisical patients; for the poor seldom apply for either indoor or outdoor relief until the early stages are past and the disease has gained a firm hold on one or both lungs. In private practice we see cases earlier, and the desirable treatment can be better followed out. But here many of the cases which recover come to us in what used to be called the "pretubercular" stage, when we rather forebode than diagnose the fatal disease. They improve under treatment, and we do not see them again.

With all these drawbacks few physicians have not been fortunate enough to see unmistakably phthisical patients improve, recover health, and continue many years without return of their symptoms.

The following are some of the cases of "cured phthisis" which have come under the writer's notice.

A lad of seventeen, tall, pale, and delicate in appearance, with cough and physical signs of catarrh at one apex. Was sent to Algiers, and returned two years later stout and well, with no symptoms and no physical signs of the disease.

The patient above mentioned (p. 1079), in good health after more than thirty years of phthisical symptoms.

A young man about nineteen whose mother died of phthisis, who was of typically phthisical aspect, and who developed catarrh with anæmia and loss of flesh. He was sent into the country to live in the open air for several months, and recovered. He had a "break-down" with suspicious symptoms when about thirty-five, but quickly recovered, and is now free from all phthisical symptoms at the age of fifty-three.

A young theological student was attacked by hæmoptysis and other signs of consumption. He was sent to Montpellier, where he spent more than a year and finally recovered his health. He has lived a most useful and laborious life, and is still preaching at the age of seventy-six.

A young physician of great distinction and greater promise was attacked with pleurisy and other symptoms of unmistakable tubercular character: there had also been several cases of phthisis in the family. He went out to an elevated city in South America, and has continued well and able to practise for now twelve years.

A medical student with decided symptoms of hereditary phthisis, after careful treatment for seven years in this country, settled at a watering-place in the South of Europe, and has pursued his profession there for the last eleven years.

Prognosis.—From what has been stated in the preceding paragraphs it may easily be imagined that to give a correct prognosis in phthisis is no light matter. And in point of fact those physicians who have the largest experience are precisely those who most strictly abstain from attempting to predict the duration of life among their patients.*

We have followed usage in dividing the *anatomical* course of phthisis into three stages: the *first* stage of the "formation," deposition, or growth of tubercles; the *second* of their "softening;" the *third* of their "elimination" by the process of excavation. But, as already pointed out at p. 1074, the moist sounds which are supposed to indicate "softening" are very apt to be fallacious. These so-called stages have reference only to the local process in certain parts of the lungs, and not at all to the disease as a whole; for while vomicæ exist in one or both apices, tubercles are commonly being formed lower down. To speak of these as *clinical stages* of phthisis leads to a misconception of their significance in prognosis. To every patient, as well as to his friends, it cannot but appear to be a matter of course that in a malady like phthisis the third stage must be the worst. And yet it is no paradox to say that the contrary would often be nearer the truth.

A factor which, more than any other, requires to be taken into account in attempting to determine the probable course of the disease is its greater or less tendency to advance rapidly in that particular patient. In different cases the differences in this respect are enormous; and it does not appear that any explanation of them can be given, except that, as a rule, the progress is quickest in those who have a strong inherited tendency to con-

* See on this difficult question the sections on prognosis in the works of Dr Walshe and of Dr Williams, and the monograph of Dr J. E. Pollock.

sumption. The formation of a large cavity takes a long time, especially if its walls are to acquire a smooth lining. Hence, whenever the morbid process spreads with much rapidity through one or both of the lungs, the opportunity for such cavities to develop themselves is wanting. In other words, the fact that a case presents the physical signs of the third stage is proof that its course has been such as generally warrants a comparatively favourable prognosis. As a matter of experience, patients with large vomicæ often go on year after year with but little change in their condition, and even with enjoyment of life. Dr Walshe speaks of two singers—a distinguished contralto and an excellent soprano—as having within his knowledge continued to perform at the Opera "while the excavating process advanced in their lungs."

Again, one is compelled to speak very guardedly of the probable duration of phthisis, if physical signs indicate that the morbid process is still actively going on in any part of the lungs, whatever may be the stage to which it has reached in the apices. One must not forget, too, that although in the lung first affected its progress may have been slow, it may hurry on to a fatal termination when it passes to the other lung.

The degree of severity of the general symptoms is very important in regard to prognosis, especially the rate of the pulse and the height of the temperature. But it must be remembered that other causes may render the pulse rapid as well as activity of local mischief. And the existence of pyrexia, as has been shown by Dr Theodore Williams, is not incompatible with gain of weight—nor even, we may add, with the subsidence of many of the other symptoms of the disease—provided that the patient eats and digests well.

The majority of cases of acute or "pneumonic" phthisis occur in young subjects, whereas "fibroid" phthisis, the most chronic form of the disease, is more frequent in those who are advanced in years. This seems to have led to the idea that the prognosis is more favourable in proportion as the patient is older. Dr Walshe, however, says that his observations at the Brompton Hospital failed to confirm such an opinion; and Lebert (in vol. xi of the 'Deutsches Archiv') observed that age seems to have little influence on the intensity of the pyrexia, which we have seen to be one of the most important factors in determining the rate of progress of the disease. It can hardly be doubted that in this disease, as in almost every other, the patient's power of resistance and his capacity for repair must alike diminish as he grows older.

In women the duration of phthisis is, on the average, shorter than in men. In children the disease is much less common than general tuberculosis; when present it runs a rapid and febrile course.

Hæmoptysis is not in itself a dangerous symptom, and cases of recurrent severe hæmorrhage belong, as a rule, to the chronic forms of the disease.

Hereditary cases are the worst, particularly when the hereditary tendency shows itself early.

On the whole, good appetite and increasing weight are more important as favourable tokens than absence of well-marked physical signs.

Ætiology.—The discovery of a specific bacillus in all tuberculous lesions has greatly disturbed the question of the origin of phthisis. And yet the constant presence of a microbe is far from deciding the whole of ætiology. In the most exclusively and directly contagious diseases,

syphilis, typhus, smallpox, none has as yet been certainly discovered. Erysipelas and cholera, in each of which one is present, are not in the ordinary sense infectious.

We have already considered the evidence that tuberculosis generally is a specific disease, with the constant presence of a definite bacillus, and found that it fulfils the criteria laid down (*supra*, p. 312 and p. 13). We have also in this chapter endeavoured to show that pulmonary phthisis is pathologically one and the same disease—tubercular inflammation of the lungs. It follows that phthisis depends on the presence of Koch's bacillus, and the conclusion is corroborated by its being so constant in the sputa that its presence there has become a valuable means of checking our diagnosis in doubtful cases. Moreover, it has been detected by Dr Vincent Harris in old museum specimens of phthisical lungs ('St Barth. Hosp. Rep.,' vol. xxi).

Admitting, however, the constant presence of the bacillus, we have, as in other specific diseases, to determine the power of resistance of the tissues to its invasion. This varies with different contagia. That of smallpox and of syphilis is so efficient, that once taken into the lymph-channels or the blood it probably produces the disease in every case. But even here what is so certain a poison in man fails with most at least of the lower animals; and the most efficient contagia may fail in protected organisms. Probably most persons are partially protected against the invasion of enteric fever or erysipelas.

It seems likely that the microbes of tubercle are so widely diffused that all dwellers in cities, at least, must again and again be exposed to their action; yet how comparatively few become tuberculous! Hence we must, in addition to the necessary "exciting" cause, the bacillus, recognise "predisposing" causes in whatever weakens the resistance of the organism generally or of the lungs in particular, and so allows of the "efficient" cause of all the symptoms of phthisis—*i. e.* the inflammatory lesions which follow the permanent lodgment and multiplication of the bacillus.

Of these predisposing causes the most effectual is the fact of parents having suffered from the same disease, which we state abstractly as hereditary *disposition*, or, to use the Greek form of the same word, *diathesis*. The others are either general, or consist in local pulmonary lesions.

i. *Direct contagion.*—In discussing the subject of tubercle in general (p. 317) we saw that clinical observation is altogether opposed to the idea that direct *infection from* another patient is at all common in the ætiology of tubercular diseases.

Of the fact that consumption is not communicated as a rule directly from one person to another no better illustration could be given than a statement published in 1867 by Mr Vertue Edwards, who had then for seventeen years been resident medical officer at the Brompton Hospital. In that period he remembered personally fifty-nine resident medical assistants, whose duration of office averaged quite six months. Of these he believed all but two to be alive; one had died of aneurysm, one of some cause unknown; three, still living, were said to be consumptive. Very many nurses had been in residence from periods varying up to eight, twelve, or even twenty-four years. Of the head nurses, who slept each in a ward of fifty patients, only two were known to have died—one of apoplexy; the other, after an unhappy marriage, of phthisis. No under nurse, so far as he was aware, had died of phthisis. The matron and her two predecessors,

as well as the chaplain and his two predecessors, were all alive. Of the physicians, whether for in-patients or out-patients, all were living except two; one had died of causes unconnected with disease of the lungs, the other from some disease of unknown nature, after twelve years' absence from the hospital. Mr Edwards himself happily survives after forty years.

The fact that phthisis does not commonly spread from a patient who remains in his home to brothers, or sisters, or other relatives, is the more striking because they must be supposed to have inherited more or less predisposition to the disease.

In Dr Weber's cases, recorded in the Clinical Society's 'Transactions' for 1874, the disease seemed to pass immediately from husbands to their wives. The husbands, all of whom were affected before marriage with pulmonary tubercle, were nine in number; but the deaths from phthisis among their wives were as many as eighteen: one lost four wives in succession, one lost three, four lost two each, three lost one each. In seven out of the nine husbands there was a decided family taint; the wives were with one exception free from any such taint, and they were all healthy at the time of marriage. The disease ran a very rapid course in all the wives, terminating in several instances within twelve months, and being never prolonged beyond eighteen months. It was not caused by anxiety or fatigue in nursing the husbands, for the husbands were all apparently well, and none of them succumbed to phthisis until long after their wives. Almost all the wives bore children to their husbands, so that it is perhaps possible that infection took place through the fœtus; but about the state of health of the children nothing is said.*

Judging by the analogy of syphilis, we should say that either infection took place through the ovum or else by direct transference of contagion from local tubercular lesions of the epididymis, but there was no evidence of either; and it seems more probable that the infection was by the breath independently of sexual intercourse.

Dr Weber was acquainted with only thirty other consumptive husbands whose wives escaped phthisis. But in all likelihood this was an accidentally small number; and among twenty-nine consumptive wives who married healthy husbands, only one lost a husband from phthisis.†

The possible infection of phthisis is no new doctrine. Morton wrote in 1697, "I have often found by experience that an infected person may poison a bedfellow by a kind of miasm like that of a malignant fever." In

* Dr Weber seems to have been disposed to think that the seminal fluid was the medium of transference of the disease. But even if this were the case, it does not follow that there must have been conveyance of a specific contagion. May not such cases merely afford another instance of that inexplicable influence of impregnation which stamps on the female organism the characters of the male, so that they can be transmitted long afterwards to offspring by a different male? Cases of this kind among the lower animals are well known to be frequent; and it is said that similar cases occur as the result of sexual intercourse between human beings belonging to different races. The following case, if not merely accidental, was probably an instance. A candidate for life insurance, whose mother had had two husbands, said that the first husband and several of his children had died of phthisis; the second husband was free from all tubercular tendency, but the eldest of the offspring of this marriage nevertheless became affected with the disease.—C. H. F.

† On this subject see the facts recorded in the 'Collective Investigation Record,' "Report on the Communicability of Phthisis;" and also a paper by Dr Burney Yeo, in which he skilfully states the case for regarding phthisis as a contagious disease ('Brit. Med. Journ.,' April 18th, 1885). Dr Longstaff's statistical correction of the figures adduced shows, as Dr Ransome remarks, that the proportion is not greater than would result from merely accidental coincidence.

Italy consumption has always been regarded as a contagious disease. The demonstration of the presence of the bacillus of tubercle in cases of phthisis affords a ready explanation of its being communicable. That it is not readily communicated is certain, but it does seem to be so under certain favourable conditions, as in the case of husband and wife and other persons living in close and habitual contact. The dried sputum of phthisical persons preserves the bacillus for a long time, and in crowded towns there must be abundant opportunities of infection from so common a disease.

There is also reason to believe that the tubercular contagion may be conveyed by means of milk from diseased cows, and may thus gain entrance directly to the intestine and indirectly to the lungs (cf. *supra*, p. 316). The same property has been ascribed to imperfectly cooked meat from tuberculous cattle, but with less evidence at present. A Royal Commission on this important subject is about to begin its inquiries (July, 1890).

ii. *Habitual breathing of air rendered impure by overcrowding or by defective ventilation* may, and probably does, act in two ways: first, indirectly by weakening the resistance of the tissues; and secondly, directly, by increasing the chance of infection. The organic matter exhaled during respiration appears to be directly poisonous. Dr Parkes cites some experiments made by Gavarret and by Hammond with expired air from which the carbonic acid and water had been removed, so as to leave only the organic matter; a mouse placed in it died in forty-five minutes. Dr Parkes says that he has known instances in which breathing for three or four hours air contaminated by having been previously used in respiration, caused headache and febrile symptoms which lasted one or two days. It is important to remember that such organic substances probably differ from gases like carbonic acid in having far less tendency to rapid diffusion through the atmosphere; they readily adhere to textile fabrics, especially to those which are dark-coloured, and cling to them obstinately. Every physician is familiar with the peculiar odour belonging to the clothes of the women and children of the poorer classes; one perceives it as soon as they enter the out-patient room. Precisely the same smell is constantly to be perceived in the rooms in which these people live. The organic matters which cause it cannot be removed by merely causing a current of air to blow through a room for a few minutes in the day; still less will they escape through a door towards which there is no active draught. In all probability the only way of getting rid of them is by oxidation; and possibly the agency of ozone is likely to destroy them more rapidly in full sunlight.

Strictly speaking, overcrowding and defective ventilation are not convertible terms; but in practice we scarcely ever meet with one of them apart from the other. It is possible no doubt for one person occupying a room of good size so to close up all the openings as to render the air impure. But there is never overcrowding without bad ventilation, because when many persons are huddled together in a small space the needful admission of fresh air exposes some of them to cold draughts, and is sure to lead to one aperture after another being shut up.

The proof that impure air is a cause of phthisis rests mainly upon the evidence of statistics as to the frequency of the disease among soldiers, artisans, and inmates of prisons. As regards soldiers, a Royal Commission upon the Sanitary Condition of the Army, which reported in 1858, brought to light the fact that the death-rate from consumption in all branches of the service was in excess of that of the civil population of large towns, and

(what was most remarkable) that among the Foot Guards it was more than twice as great as that of the civil population. The only explanation that could be offered was that it came from defective barrack accommodation, since neither the clothing of the soldier, nor his food, nor the nature of his occupation could be supposed to be the cause of it. There was evidence that in barrack dormitories the cubic space actually given to each man was often not more than one half or two thirds of the amount of 450 feet, which was the minimum allowed by regulation; and it was also shown that the air in these rooms became offensive before morning. The conclusion at which the Commission arrived has since been confirmed by the great fall in the consumptive death-rate, especially among the Foot Guards, which has followed the introduction of sanitary improvements.

Dr Farr long ago stated his belief that the prevalence of phthisis in the armies of Europe is probably due in part to the inhalation of expectorated tubercular matter dried, broken up into dust, and floating in the air of close barracks.

In Dr Ransome's Milroy Lectures it is stated that in Canada, a country comparatively free from phthisis, the death-rate among English soldiers was 23 per 1000, compared with an average throughout England of less than 10 per 1000, and a death-rate in Manchester of only 12·4. After the barracks had been properly drained and ventilated, the mortality had sunk from 23 per 1000 in 1865 to 9·5 in 1872, and 6 in 1874.

As to workmen, we have evidence given by Dr Guy before the Commission of Inquiry into the State of Large Towns, of which the Duke of Buccleugh was president, and which reported in 1844. Dr Guy had most elaborately investigated the relative liability to phthisis of different classes of the population of London. He found that the disease was more fatal to artisans than to tradesmen, and more fatal to tradesmen than to professional men and the upper classes. Even hawkers, standing about in the streets and exposed to all inclemencies of weather, had the advantage over men employed in workshops. Among printers he instituted a very close comparison as to the frequency of symptoms of lung disease, arranging the men in classes according to the amount of air-space in the rooms in which they worked. Of 104 men having less than 500 cubic feet of air to breathe, 13 had suffered from blood-spitting and 13 others from catarrh; of 115 men having from 500 to 600 cubic feet of air, 5 had suffered from blood-spitting, 4 from catarrh; of 101 men having more than 600 cubic feet of air, 4 had suffered from blood-spitting and 2 from catarrh.

With regard to prisoners, there is the contrast between two prisons in Vienna, cited by Dr Parkes in his 'Practical Hygiene.' In the Leopoldstadt prison, which was very badly ventilated, there died in the years 1834—1847, 378 prisoners out of 4280, or 86 per 1000; of whom no fewer than 220, or 51·4 per 1000, died from phthisis. In the well-ventilated House of Correction in the same city there were from 1850 to 1854, 3037 prisoners, of whom 43 died, or 14 per 1000; and of these 24, or 7·9 per 1000, died of phthisis. Diet and mode of life were, it is believed, the same in both establishments. It is a flaw in the case that the average length of the periods during which the prisoners were detained in each prison is not given; but Dr Parkes thinks that no correction on this ground, even if needed, could account for the discrepancy in the death-rate. The great prevalence of phthisis among prisoners in general was long ago pointed out by Dr Baly, as the result of an examination of the 'Records of the Millbank Peni-

tentiary.' But he was unable to determine what part in the ætiology of the disease should be assigned to defective ventilation, and what to other depressing causes.

Indeed, it is obvious that only in very exceptional instances can overcrowding or defective ventilation be so isolated from other injurious conditions, apart from direct contagion, as to be proved the main cause of phthisis. For example, an important investigation was made by Dr Greenhow in 1860 and in 1861 as to the origin of the great mortality from phthisis among the workpeople employed in many of the largest industrial occupations of the country, and he found that the inhalation of dust of various kinds is the main cause of the disease so far as these persons are concerned (p. 1092). But Dr Greenhow himself pointed out that another cause was certainly the "working in ill-ventilated or over-heated factory rooms or workshops, as in those of some of the silk mills of Coventry, in the domestic weaving shops, and in the watchmakers' factories and workshops of the same city, in the button-makers' and various other workshops of Birmingham, in the factory rooms of Blackburn and Nottingham, and in many domestic shops and warehouses." Abundant details in support of this conclusion are to be found in the report which he furnished to Mr Simon.

iii. *Inheritance* is the most important predisposing cause of phthisis.—It is a matter of universal experience that in some families deaths from phthisis occur, generation after generation, with terrible frequency. Parents and their offspring are swept off in turn, so that sometimes there is hardly a survivor to maintain the stock. Actuaries are so impressed with these facts that whenever it can be ascertained, in reference to a candidate for life insurance, that he has lost a parent or more than one brother and sister from consumption, it is held at almost all offices that an addition to the premium is absolutely necessary to cover the increased risk; and if both parents have died of phthisis, or more than two other near relatives, the "life" is generally regarded as almost uninsurable on reasonable terms. It might at first sight appear strange that an augmented liability to what is (after all) only one among a great many other possible causes of death should be taken as diminishing to so great an extent the general "expectancy" of the candidate, but the requirements of the offices are in practice found to be fair and equitable.

From a scientific point of view, however, the question of the inheritance of consumption requires far more consideration than has generally been given to it. Among persons actually affected with phthisis, the proportion of cases in which the occurrence of a like disease can be traced in their relatives appears from certain investigations made by Dr Theodore Williams, and recorded in the 'Med.-Chir. Trans.' for 1871, to be 48·4 per cent. The patients were seen in private practice, so that the results are probably as little inaccurate in the way of omission as can be expected in such inquiries, although it would be an advantage, for the purpose of comparison, if we knew to what extent a similar family history exists in the population generally. But it is impossible to accept the figures given by Dr Williams, or any similar figures, as indicating in a scientific sense the extent to which consumption is transmitted by inheritance. The difficulty is often brought out very clearly by proposers for life insurance. A candidate has had perhaps two or three brothers who were consumptive, but one, he says, brought on the disease by dissipation and intemperance; another was in the army, and was stationed first in India and then in Canada at a few months'

interval; a third may have got a chill in bathing; and he winds up by declaring that phthisis has not been a "family complaint" after all. Now, among the cases collected by Dr. Williams, 484 in number, in which phthisis was traced among the relatives of patients themselves phthisical, there were 120 in which the disease had existed in one or both of the parents, but 224 in which it affected only brothers or sisters. There can be no doubt that if inquiries had been made as to the existence of definite "exciting causes" of the disease in these cases they would have been found to be very often present. It is, in fact, impossible to draw a line anywhere between what might be called respectively "hereditary" and "accidental" phthisis. Probably there are few families in which the consumptive tendency is so strong that it could not be kept in abeyance by hygienic precautions if they were thoroughly and vigorously carried out; and, on the other hand, there are very few families, if any, in which the disease may not show itself in such members of it as systematically neglect their health, or are exposed year after year to unfavourable circumstances.

It is impossible at present to determine in what proportion of cases the so-called "family predisposition" to consumption implies the actual transmission of a definite tendency to this disease, and in what proportion of cases it is merely the expression of a general delicacy of constitution (or, as the Germans call it, "vulnerability"), which renders those who are derived from certain stocks liable to be attacked by consumption in succession, as they happen to come under conditions suitable to its development. From the point of view of the insurance offices the distinction is not material, for in either case the demand for an enhanced premium is equally justifiable. One fact which tells strongly in favour of the opinion that family predisposition is often a mere vulnerability is that the liability to consumption is believed to be much above the average in those who come from parents already failing in health from any cause, in those begotten by a father advanced in years, in those born of a very young mother, and also in the later offspring of a woman exhausted by frequent and rapid child-bearing. Moreover, there is little evidence of a specially strong tendency to phthisis in the children of parents actually consumptive, one or both of them, at the time of procreation.

The experience of insurance offices as well as of private practice is that a phthisical tendency is more frequently transmitted by the mother than by the father.

iv. *Personal predisposition.*—It is a very old belief that persons of a particular bodily frame and physiognomy are prone to tuberculous diseases; and this has been called a *diathesis.* Little value, however, can be attached to the statements of early writers on the subject, because "scrofula," as it was called, was often confounded with rickets or with congenital syphilis; and, according to Sir Thomas Watson, the numerous signs of the "scrofulous diathesis" varied widely with the "temperament" of the individual, whether "nervous," "sanguine," or "bilious." All this was very confusing; and it seemed a step in advance when Sir William Jenner, in 1860, proposed to distinguish two *diathetic states*—tuberculosis and scrofulosis.

As leading features of *tuberculosis* he gave the following:—"Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organisation generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright, pupils

large; eyelashes long; hair silken; face oval, good-looking; ends of long bones small, shafts thin and rigid; limbs straight. Children the subjects of tuberculosis usually cut their teeth, run alone, and talk, early."

He described *scrofulosis* as follows:—"Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty-looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick."

Among the pathological tendencies of the former morbid conditions he mentioned not only "deposits or formations of tubercles," but "fatty degeneration of liver and kidneys, and inflammation of the serous membranes." To the latter he assigned "inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the bronchial glands on trifling irritation; obstinate diseases of the skin; caries of bone."

Many physicians still believe that Jenner's descriptions correspond with two great types, the recognition of which is really important in practice. But this belief is incompatible with modern views as to the relation between tubercle and caseating affections of lymph-glands; no less incompatible with Buhl's theory of infection than with the more recent demonstration that the glandular affections are themselves tuberculous.

Moreover, though many of those who die of phthisis present the characters, according to Jenner, of scrofula, a much larger proportion have no such indications; while his description of tuberculosis seems to refer to a habit of body which, while not incompatible with symmetrical growth and physical beauty, shows a want of power of resistance to disease in general, rather than to the invasion of tubercle in particular.

Mr Francis Galton and the late Dr Mahomed recorded in the 'Guy's Hospital Reports' for 1881 the results of "An Inquiry into the Physiognomy of Phthisis by the Method of Composite Portraiture." Their conclusions seem to bear out the view just stated. For although they were able to obtain from the photographs of 442 phthisical patients two types of face—the one of narrow ovoid shape, the other a broad face with coarse features,—yet this was only by the careful selection of a few out of the whole number; and they found a larger proportion of narrow ovoid faces among patients who were not phthisical than among those who were.

The fact is that the words *scrofula* and *struma* have been applied in so loose and arbitrary a way, quite independently of chronic swelling of the cervical lymph-glands, or even of evidence of caseous disease in any part of the body, that it is better to avoid their use until we have a collection of facts based on the accurate use of clinical or pathological terms. Moreover, tendencies to scrofula or tubercle which are never carried out can scarcely be the subject of useful discussion, nor can the exploded doctrine of temperaments be revived until we again accept the four Galenical humours and their eucrasia or dyscrasia.

The phthinoid chest.—There still remains the question whether a tendency to phthisis is indicated by any particular *form of chest*. Dr Gee recognises two shapes of chest in persons predisposed to consumption.

One of them is the "alar," or "pterygoid" chest of Galen and Aretæus. This is narrow and shallow, the antero-posterior diameter being especially small, and the angles of the scapulæ projecting like wings; its peculiarities

depend upon drooping or increased obliquity of the ribs, as the result of which the shoulders fall and the length of the thorax from above downwards is increased; the alar or "winged" appearance is caused by the projection of the scapulæ. The throat is often prominent, the neck long, and the head carried unduly forwards.

The other is called the "flat" chest; this, instead of being rounded, is flattened in front, the rib cartilages losing their curve and becoming straight; or the sternum may actually be depressed below the level of the costal ends of the cartilages. Persons who are extremely flat-chested often have broad shoulders, so that, as one stands facing them, one might fancy them to be by no means ill developed. Traube and other German writers lay great stress on the significance of a flat chest as indicating a liability to consumption, and Dr Wilks used to insist on it strongly.

But probably the "flat chest of phthisis," like the "rounded chest of bronchitis," is an acquired condition, the *result of disease*, not the indication of its future advent. Neither the alar nor the flat chest is seen in early phthisis with more frequency than other varieties of ill-shapen chest, which often result from neglect during childhood, and are consequently very common among the poorer classes.

Again, one cannot dissociate congenital from acquired deformities of the thorax in regard to their possible influence on the subsequent occurrence of tubercular disease of the lungs. Freund, in 1859, maintained that what caused a small and contracted chest was often a premature ossification of the cartilage of the first rib, occurring even in early infancy. It is worthy of notice that Dr Hutchinson, in advocating the use of the spirometer, did not suggest that a defective vital capacity of the lungs indicated a tendency to phthisis, but rather that it was a sign of the actual presence of the disease at an early stage. Again, deformity of the chest from lateral curvature seems certainly not to carry with it any increased liability to consumption. It appears to be very doubtful whether the habit of stooping at a desk, or in the work of a tailor or shoemaker, or weaver, although it cannot but be injurious to the health, specially favours the development of phthisis. Nor is there evidence that the foolish practice of compressing the base of the chest by stays is capable of producing such an effect.

iv. *Predisposition from general causes.*—The following deteriorating influences lead only indirectly to phthisis, by weakening the health and the power of resistance of the tissues.

Food insufficient in quantity and bad in quality is assumed to predispose to phthisis, but it would be difficult to adduce any positive proof of this.

The same doubt applies to the presence of *anæmia*. Certainly both emaciation and anæmia may go on to an extreme degree in hospitals where cases of phthisis are present, and yet no tubercle develops.

Child-bearing and suckling seem often to be concerned with phthisis in women. It is a well-known and remarkable fact that during gestation, consumption is scarcely ever fatal. The patient survives until labour is over, after which the symptoms become more urgent than ever, and death may follow rapidly. In many cases the disease appears to begin during lactation, or after profuse puerperal hæmorrhage. Cases associated with child-bearing generally run a particularly acute course.*

* These considerations are so important in regard to life insurance, that it is only prudent to count all deaths of mothers "in childbed" or "after delivery" as due to phthisis, unless there is explicit evidence of previous good health.

Alcoholic intemperance has a doubtful position in the ætiology of phthisis. Clinical experience shows that drunkards die in large numbers of this disease; but it is often difficult to exclude the operation of other causes, such as bad ventilation, and exposure to cold and wet. Moreover, some writers have believed that the moderate and even the excessive use of alcohol is rather prophylactic against than productive of phthisis. Observations in the United States have been cited in favour of this view. Dr Walshe and Dr Wilks do not accept intemperance as a cause of consumption, while Dr Williams has no hesitation in doing so. Lately Dr Owen, of St George's Hospital, found that, among the phthisical patients attending there, 50 per cent. were, on their own showing, excessive drinkers, while only 33·5 per cent. of the other patients confessed to habits of intemperance; or, put in another way, of 100 non-consumptive patients, 41·5 professed to be temperate; of 100 consumptives, only 23.

It must be remembered that drunkenness goes with poverty, exposure, and unhealthy living, which are acknowledged to be predisposing causes of bronchial and alveolar catarrh and so of phthisis. Again, most of the publicans, barmen, and other intemperate classes who consult a specialist will be subjects of the disease for the treatment of which he is esteemed. Is phthisis less common in Italy than in Great Britain, in Constantinople than in Glasgow? Are total abstainers more exempt from consumption than temperate persons? And do no intemperate persons recover from phthisis? These are questions not yet answered.

It has often been supposed that phthisis in drunkards is usually fibroid rather than tuberculous. Huss, of Stockholm, suggested this opinion about 1850, and it has been supposed to derive support from the fact that alcohol causes a growth of interstitial fibrous tissue in the liver, and perhaps also in the kidneys and elsewhere throughout the body. But it is certain that tubercular peritonitis of the most typical kind is often seen in intemperate persons who also have hepatic cirrhosis. We have given reason to believe that "fibroid phthisis" is not essentially distinct from the more purely tubercular form of the disease, but is only the extreme type of what almost every case of phthisis shows in some degree, a power of repair by cicatrisation, adhesions, and fibroid thickening. Moreover, the diagnosis of fibroid phthisis which during the life of the patient had been partially based upon a history of alcoholic intemperance, has been repeatedly falsified by the autopsy.

Diabetes is a frequent cause of a phthisis which is peculiarly pneumonic in character; its relations to tubercle are still disputed. This point will be discussed in the chapter on Diabetes, but here the probability may be stated that the diabetic affection of the lungs is an acute variety of tuberculous phthisis.

Syphilis is sometimes followed by the development of a destructive disease of the lung, which on *post-mortem* examination is found to be typically tuberculous. No fewer than thirteen cases of this kind occurred at Guy's Hospital between the years 1863 and 1873. But one must not forget that among syphilitic patients, especially in hospital practice, many are intemperate, destitute, and every way careless of their health. Consequently it is quite possible that the relation between the phthisis and the syphilis may, in many of the instances just referred to, have been one of mere coincidence. Or it is possible that the cachexia of syphilis may favour the development of tubercle in the same way as it leads to lardaceous disease of the viscera and to tabes dorsalis. The administration of iodide of potassium is occasionally

followed by striking improvement in the physical signs and symptoms of what had appeared to be phthisis in a syphilitic subject, or even by their complete subsidence. It is true that any treatment which improves the general health of a consumptive patient may be followed by an apparent cure of the pulmonary affection; but a more probable way of explanation of the striking action of antisyphilitic remedies in the cases in question is to recognise a syphilitic disease of the lungs capable of simulating phthisis, but pathologically distinct. The characters of such a disease, distinct from true or tubercular phthisis occurring in a syphilitic subject, have been set forth in the chapter on chronic inflammations of the lungs (*supra*, p. 1011).*

The following conditions affect the lungs directly, and act as predisposing causes of phthisis by producing precedent catarrh.

vi. *Inhalation of dust.*—Ramazzini, in his work, 'De Morbis Artificum,' published in 1703, seems to have been the first to point out that certain classes of workmen are liable to have their lungs injuriously affected by dust given off by the materials which they employ. In the present century the question has been studied very thoroughly, especially in England and in Germany; and Zenker has proposed to term the pulmonary diseases due to this cause *Pneumonoconiosis* (κόνις = dust). The nature of the mischief set up in the respiratory organs by breathing dust varies widely in different cases. It may be a mere catarrh of the trachea and bronchial tubes, leading to chronic cough and emphysema, and perhaps proving fatal by dilatation of the right side of the heart and dropsy. But in many instances it consists in consolidation of the lung, which may ultimately lead to the formation of cavities, and spread through the pulmonary tissue from apex to base, as in other cases of phthisis; so that it has been usual to speak of "miners'," "weavers'," and "knifegrinders' consumption." Pathologists, however, almost without exception maintained that the lesions found in such cases were not tuberculous. Whether tubercle was regarded as a deposit from the blood in a special dyscrasia, or as a particular kind of new growth, it seemed equally logical to deny the possibility of its being due to a mere local irritation of the tissues. These views were of course

* There is, of course, no reason why gummata should not develop themselves in the lung as well as in any other organ; but the recorded instances in which such lesions are stated to have been found in the *post-mortem* room seem to me to be generally open to more or less doubt. Take, for example, Dr Goodhart's case, of which there is a coloured plate in vol. xxx of the 'Pathological Transactions.' He himself mentions that Dr Reginald Thompson considered the supposed gummata to be relics of pulmonary hæmorrhage (*loc. cit.*, p. 236), and they certainly do not seem to me to differ at all from the encapsuled cheesy masses which are often found in ordinary cases of phthisis. Moreover, one cannot admit in the present state of pathology that the undoubted grey tubercles which also existed in large numbers in Dr Goodhart's case were merely secondary results of the caseation of gummata, causing infection of the pulmonary tissue, although a few years ago such an interpretation of the facts would to most observers have appeared satisfactory. In vol. xxviii of the 'Pathological Transactions' may be found a detailed discussion of the relations between syphilis and phthisis by Dr Goodhart (pp. 313—329); and there are also cases in which the lungs appeared to contain syphilitic lesions, recorded by Dr Pye-Smith and Dr Green (*ibid.*, pp. 331, 334). But I fail to discover, either there or anywhere else, such examples of extensive lung disease, obviously and unmistakably different from tubercular phthisis, as certainly ought now and then to be met with if the views held by these observers were correct. Dr Moxon, when pathologist at Guy's Hospital, was much impressed with the fact that in several syphilitic cases he found in the lungs indurated patches, the centres of which had sloughed away so as to form cavities in the interior of which shreddy masses were still hanging. And I think that he came to the conclusion that such appearances were almost characteristic of a syphilitic affection. But the difficulty is that they are altogether unlike those which syphilis is known to produce in other organs.—C. H. F. (1880).

adopted by those who accepted Niemeyer's doctrine of the non-tubercular nature of phthisis in general. And the theory of the local development of tubercles, as the result of tissue-infection from lesions themselves inflammatory, seemed to remove all difficulties by explaining the fact that in many cases the more recently affected parts of the lungs show tubercles when none can be discovered in the parts which were earliest diseased.

It might be thought that the reports of autopsies could decide the matter, since they generally affirm that the appearances found in the lungs are altogether different from what are seen in tubercular cases. But several points must be borne in mind. One is that the character of the lesions in the lungs is often greatly obscured by the discoloration from the foreign particles deposited in the pulmonary tissue. Another is that as a rule the destructive diseases of the lungs which are due to inhalation of dust occur at rather a late period of life, and advance slowly to a fatal termination; in both respects they resemble that form of phthisis which is regarded by many pathologists as distinct from the tubercular, and termed fibroid. In descriptions of the diseased lungs of miners, potters, or stoneworkers, the use of the word tubercle is carefully avoided; but mention is often made of "nodules," from the size of a millet-seed to a pea, scattered in the pulmonary tissue, of an opaque, greyish-yellow colour, and perhaps with a small central cavity. The microscope is called in to prove the negative fact that the nodules in question fail to present whatever histological structure may happen to be regarded as characteristic of tubercle at the time when the observation is made; or possibly it is asserted that they are distinguished by their hardness and resistance to pressure, the fact being overlooked that undoubted tubercles, if they fail to caseate, undergo conversion into precisely similar bodies. Anatomically the chronic pneumonia of grinders and potters cannot be separated from chronic tubercular phthisis.

Nor does it appear that the clinical course of the disease due to dust-inhalation differs essentially from that of the more chronic forms of phthisis. Dr Greenhow, who was one of the leading authorities on the subject, insisted (in the 'Pathological Society's Transactions,' vol. xvi, p. 61, *et passim*) on "the coincidence of a cool skin and a quiet pulse with wheezy asthmatic cough and copious muco-purulent expectoration," as rendering "the diagnosis from tubercular phthisis comparatively easy." And elsewhere he says that "shortness of breath almost invariably precedes by some considerable time the appearance of cough, and the patient is often ailing for many years before being disabled from work." But with regard to the suggestion that the early dyspnœa is distinctive, it must be remembered that bronchitis and emphysema are marked effects of the inhalation of dust. Moreover, it is possible, as Seltmann suggested in the 'Deutsches Archiv' for 1867, that when foreign matters are deposited in the lungs in large quantity the respiratory surface may be so much diminished that dyspnœa is a result.

Lastly, recognition of the presence of Koch's bacilli has conclusively established the tuberculous nature of the disease, and confirmed, in several cases of pneumonoconiosis, the justice of the opinions above expressed.*

* Bacilli were found in two cases of grinders' phthisis at Brompton Hospital kindly communicated to the writer by Dr Theodore Williams, and in three cases of potters' phthisis, published by Mr Watson Cheyne in a valuable paper on the "Relation of Micro-organisms to Tuberculosis," in the 'Practitioner' for April, 1883 (p. 294).

The materials which give off dust that may enter the respiratory organs are of various kinds. They may be classified as follows.

(1) *Particles of carbon.*—As far back as 1813 Pearson, in the 'Philosophical Transactions,' threw out the suggestion that the black discoloration of the lungs and of the bronchial glands, which is found in most adults, but not in children, consists of particles of carbon "introduced with the air in breathing," and originally "derived from the combustion of coal, wood, and other inflammable materials." A similar opinion was expressed by Laennec. In 1831 Dr Gregory recorded the case of a labourer in the coal mines of Dalkeith; both lungs were throughout of a uniform coal-black colour, and yielded, when washed, a dark matter, which was found by Dr Christison to resist the action of concentrated nitric acid and of chlorine, and to yield by distillation products just like those which result from the distillation of coal. The conclusion seemed irresistible that the organs had been discoloured by the penetration of coal-dust from without. "Spurious melanosis," therefore, and *Anthracosis*, proposed by Stratton in 1837, were the terms applied to this condition of the lungs. But Hasse in 1841, and Virchow in 1847, gave reasons for doubting this explanation, and it was not until 1860 that a case was recorded by Traube in the 'Deutsche Klinik,' which finally established the reality of anthracosis. The patient was a man who for about twelve years had been engaged in loading and unloading wood-charcoal. He had long been accustomed to expectorate a black substance, and when he died his lungs were found to be almost everywhere of a black colour, yielding to pressure a black frothy fluid which stained the fingers like thin Indian ink. Both in the sputa, and in the pulmonary tissue, there were found minute foreign bodies of irregular form, with pointed processes. That these were fragments of wood-charcoal was evident not only from a comparison with particles of the charcoal brought from the place where the man had worked, but also from the fact that some of them showed the circular discs characteristic of the woody fibres of Coniferæ. A second similar case came before Traube six years later, but even he did not at first admit from these two cases that the finely granular material which gives a black colour to healthy lungs as life advances is also inhaled carbon. This conclusion was greatly helped by Zenker's discovery that the lungs of those who work with red oxide of iron become full of red particles. In 1866 Virchow took an opportunity of recanting his former views, and Rindfleisch in his 'Handbook' ascribes anthracosis to the cause denoted by the word.

Modern histology has removed one of the greatest obstacles in the way of the acceptance of this doctrine, by showing that leucocytes are capable of taking particles of foreign materials into their substance, and that animal membranes are permeable in a way that formerly was unsuspected.

In 1858, in describing in the 'Edinburgh Medical Journal' a specimen of "miner's lung" which had been sent to him from Scotland, Virchow had stated that scarcely any of the black matter was found in the interior of the alveoli; and in their walls it lay not beneath the endothelium, but between the elastic fibres and the connective tissue. It was still more abundant in the interlobular and peribronchial fibrous tracts, and beneath the pulmonary pleura. It may even be present in the costal and diaphragmatic pleura, as well as in the bronchial glands. At Guy's Hospital we once found some of it free in the upper and back part of the pericardial space, separated by the fibrous wall of the sac from an intensely black gland that lay just outside. The distribution of inhaled particles of dust has been studied

experimentally by von Jus, whose observations are recorded in the 'Arch. f. exp. Pathologie' for 1876. He injected cinnabar into the air-passages of dogs, and found that the particles were rapidly taken up by cells which he believed to be altered leucocytes, so that five days later scarcely any pigment was left in the pulmonary alveoli; within six hours after the commencement of the experiment some of it reached the bronchial glands, being first deposited in their cortical layer, but ultimately reaching their medullary structure; much, however, remained in the lungs, being accumulated in the connective tissue, between the lobules, round the vessels and the tubes, and beneath the pleura. In other words, von Jus found that its distribution corresponded precisely with what had been described by Virchow in the case of the miner's lung.

The phthisis associated with anthracosis is attended with one special symptom, which may be conveniently mentioned here. This is the "black spit" which is often ejected in considerable quantities and for a long time, even by miners who have entirely ceased to follow their occupation. There is no doubt whatever that it is often due to the gradual disintegration of the blackened and infiltrated parts of the lungs, or comes from vomicæ such as are sometimes found after death, full of a black liquid. Thus, Dr Greenhow showed to the Pathological Society in 1869 the lungs of a collier who about ten days before his death suddenly spat up matter closely resembling black paint, and continued to expectorate four or five ounces daily until he died; in the right lung there was a large irregular cavity, containing a quantity of black pulpy residue. Nevertheless sputa may be very black without there being any lesion of the pulmonary tissue beyond the anthracosis; hæmoptysis or the detection of elastic tissue or of bacilli in the sputum is needful to prove that ulceration is going on.

(2) *Oxide of iron.*—In 1864, in discussing the subject of anthracosis, Friedreich asked the question why, if the black lungs and bronchial glands of coal-miners were due to inhaled carbon, the workers in red sandstone quarries should not have the corresponding organs reddened by the dust to which they were exposed. Now, it happened that Zenker had at that very time in his possession the lungs of a woman who had, for seven years before her death, been engaged in making the little paper books in which gold-leaf is laid. The paper has to be coloured red with peroxide of iron, and this is rubbed in with a piece of felt. The occupation is a very dusty one; and the woman's lungs were found after death to be throughout of a bright brick-red colour, so that their cut surface looked just as if it had been daubed over with red paint. The microscope showed that granules of oxide of iron were present, beneath the pleura, in the interlobular fibroid septa, in the peribronchial sheaths, in the walls of the alveoli, and even in cells occupying their interior. In the twentieth volume of the 'Pathological Transactions' there is a coloured drawing of a section of this lung, taken from a specimen in the possession of Dr Wilson Fox; the tint is much browner than in Zenker's drawings published in the 'Deutsches Archiv' two years before. Zenker proposed to name the affection Siderosis (σίδηρος = iron). In 1874, Merkel, in 'Ziemssen's Handbuch,' was able to refer to seven other cases, one of which followed the use of red oxide of iron for polishing glass. He had also met with two instances in which the lungs were blackened by the black oxide of iron, and one in which ferric phosphate was present. There was no difficulty in detecting the iron in the sputa by hydrochloric acid and ferrocyanide of potassium.

(3) *Silica and alumina.*—It has long been known that workmen whose occupations expose them to siliceous or argillaceous dust are very prone to die of phthisis. Very full information with regard to the excessive mortality from this cause in certain districts of England is contained in a paper by Dr Greenhow in Mr Simon's third 'Report to the Privy Council,' published in 1861. Merkel proposes to term the resulting disease of the lungs *Chalicosis* (χάλιξ = gravel). The presence of silica in the pulmonary tissue seems to have been first detected chemically by Dr Peacock and by Dr Greenhow. Kussmaul, however, has since shown (in vol. ii of the 'Deutsches Archiv') that this substance is present in greater or less quantity in the lungs of all persons (though not in those of a fœtus), having doubtless been derived from the dust of the streets and roads blown up by the wind. In a railway signalman, stationed in a very sandy district, Meinel found that silica actually formed as much as 18·2 per cent. of the ash of the lungs after incineration; even in a stonemason who died of phthisis, and whose lungs were analysed by Kussmaul, the amount was not greater than 24·7 per cent. of the ash. Under the microscope the particles of silica may be seen as bright bodies of round or angular shape.

Among artisans who suffer from this cause are, of course, *stonemasons*, lithographers, and millstone makers. Still more fatal is the grinding and polishing of steel instruments—from scythes to needles—such as is carried on in Sheffield and Birmingham. Whether the exact nature of the work be needle-pointing or fork-grinding, or the sharpening of fish-hooks, the result differs but little; a large number of the men die prematurely, some between twenty and thirty, and more between thirty and forty: very few survive the age of forty without suffering more or less from pulmonary symptoms. This has been long known at Sheffield as "grinders' rot." What is termed "dry-grinding" is much more injurious than "wet-grinding;" for in wet-grinding the wheel, as it revolves, dips into water and deposits a large part of the dust which would otherwise be carried into the air. But even wet-grinders are greatly exposed to dust in "hacking" their grindstones, which generally has to be done every day.

Potters, again, are exceedingly apt to be attacked with phthisis; "flat-pressers" suffer more than "hollow-pressers;" but the worst off of all are "china-scourers," whose business is to rub off the loose flint powder from the china with sand-paper, after it has been baked. Another dangerous occupation is pearl-shell cutting.

By the use of respirators, and by other preventive means, the terrible mortality in these trades has of late years been happily reduced.

(4) To the occupations which lead to injury from the inhalation of vegetable matters must be added the carding of *cotton*, and the hackling of *flax*. In two men who had worked in a *tobacco factory*, Zenker found "brown spots in the lungs and in the bronchial glands, evidently due to the deposition of powdered tobacco."*

Pathology of these pneumonoconioses.—It is clear, then, that foreign particles of various kinds may find their way into the lungs, may be deposited in the pulmonary tissue, and may either remain there or be ultimately transported to the bronchial glands. But it by no means follows, as a matter of course, that their presence must be injurious. Indeed, since the true nature of the

* According to Hirt's tables of mortality from phthisis in various trades in Germany (quoted by Dr Ransome) the highest death-rate is among flintworkers and filecutters, then come grinders and stonecutters, next brush-makers, and then cigar-makers and glasscutters.

black material found in the lungs, whether in health or in disease, has of late years become established, increasing doubts have been expressed as to whether it can fairly be regarded as the cause of inflammatory or destructive changes. In Traube's first case, referred to above, there was not the slightest trace of any newly formed connective tissue nor of any induration of the substance of the lungs; the patient's symptoms were probably due to chronic pericarditis, accompanied by double pleurisy. Seltmann, of Zankeroda, near Dresden, asserts positively in the second volume of the 'Deutsches Archiv' that among the coal-miners of that district there is generally no overgrowth of connective tissue whatever, even in lungs full of black deposits, and that the formation of cavities is very rare. As for tuberculosis, he thinks that the inhalation of carbon is actually antagonistic to its development—an opinion which Merkel appears to share.

If, therefore, English miners are peculiarly liable to destructive disease of the lungs, the explanation may be that the galleries in which they work are so often badly ventilated; the real cause of their being attacked with phthisis is not that the air which they breathe contains coal-dust or smoke, but that it is rendered impure as in crowded workshops or sleeping-rooms. Indeed, in his fourth report, Mr Simon drew special attention to the fact that the colliery miners of Durham and Northumberland differ from other miners in not suffering from any important excess of pulmonary disease, and argued that the reason for this is the good ventilation of the mines in which they work; but he was still disposed to think that this operated mainly by removing the coal-dust and powder-smoke. The great heat to which miners are exposed is probably another factor in the ætiology of the pulmonary diseases to which they are liable.

On the other hand, there is no doubt that destruction of the pulmonary tissue is the result of the entrance of other kinds of foreign particles into the lungs. Merkel says that in his cases of siderosis fibroid changes were never wanting, even when the patient had died of some independent disease. And among needle-grinders, potters, and other classes of workmen exposed to the inhalation of flint-dust or finely-powdered clay the prevalence of phthisis is far too great to be accounted for in any other way. The only opportunity which Merkel ever had of examining a grinder's lung was in the case of a boy aged sixteen, who was accidentally killed after having worked at the trade four and a half years, when apparently in good health. His lungs already contained small, tough, black nodules, of the size of a pin's head, as well as minute particles of sandstone and of iron.

Whatever may be the nature of the irritant which sets up destructive changes in the lungs, the resulting affection appears to have exactly the same characters. This was strongly insisted on by Dr Greenhow, when in 1865–70 he successively showed at the Pathological Society's meetings the lungs of a collier, a copper-miner, a razor-grinder, a stone-worker, a potter, a flax-dresser, and a pearl-shell cutter. It is remarkable that the lungs may be almost if not quite as black in those patients in whom the affection was set up by sand or clay as in the miners themselves. The explanation is that bronchitis, by interfering with the natural ciliary action of the mucous membrane, causes the particles of carbon in dust and smoke, which in greater or less amount are inhaled by everyone, to become deposited in the pulmonary tissue, instead of being swept back into the trachea. But, further, when any destructive process is set up in a lung the affected parts are very apt to become more deeply blackened than the rest of the

organ. One of the points on which Virchow insisted when he upheld the view that the discoloration was due to a pigment derived from altered hæmatin, was that even in children the development of phthisis was sometimes attended with a blackening of the tissue which at their age could only be regarded as exceptional. The experiments of von Jus, referred to above (p. 1095), enable us to understand why the foreign material should accumulate in newly-formed fibroid tissue, and even in fibroid tubercles of a diseased lung, just as it does in the connective-tissue tracts of the healthy organ rather than in its alveoli.

The complete identity of the pulmonary affections to which so many different classes of operatives are liable is in itself sufficient to show that a common pathological process is concerned in producing them. Since the irritants which excite these several diseases act upon all parts of the lungs alike, the resulting lesions might be expected to be uniformly diffused. But, as a matter of fact, it is found that in most cases they attack one lung before the other; they almost invariably begin in the upper lobe, and spread gradually downwards through the organ towards its base—that is, they follow the characteristic distribution of phthisis.

vii. *Cold and wet.*—No idea is more firmly rooted in the public mind than that consumption is often the result of accidental causes, such as getting chilled by remaining in wet clothes, or by exposure to a draught when heated in dancing, or by sleeping in damp sheets; and generally that there is danger, especially in those who are hereditarily predisposed to the disease, of its supervening upon a cold, or a succession of colds. But the weight of medical opinion has in our day tended against such notions.*

The considerations which led pathologists to reject the idea that phthisis could arise out of a common catarrh were probably in the main identical with those which induced them to regard as necessarily non-tubercular the cases due to dust-inhalation. And such theoretical opinions were greatly strengthened by the practical observation that many persons, even of delicate aspect, suffer for years from an extreme liability to bronchial attacks without ever becoming consumptive. Indeed, Rokitansky believed that pulmonary emphysema and dilatation of the bronchial tubes, if carried far enough to cause venosity of the blood and cyanosis, afford exemption from the liability to pulmonary tuberculosis (cf. p. 1106).

But, apart from the crucial instance of congenital disease of the heart, there are striking exceptions to Rokitansky's rule. In 1864, for example, a girl of seventeen was admitted into Guy's Hospital with extreme dyspnœa and dropsy, and with clubbing of the fingers and toes. The bronchial tubes were found widened out into great sinous passages, so that the cut surfaces of the lungs showed hollow spaces as extensive as the remains of the pulmonary tissue. Yet there were scattered yellow tubercles, especially in the left lung, spreading from the apex downwards. In 1874 a woman, aged thirty, died, who had long been more or less subject to cough, which for nine months before had become continuous. There was extreme emphysema of the bases and anterior parts of the lungs, and the tubes contained a large

* The only statistical facts which are in favour of the popular view are those given by Dr Theodore Williams in vol. liv of the 'Med.-Chir. Transactions.' Out of 1000 cases of phthisis he found that no fewer than "149 had originated in, or been closely preceded by, pleurisy and pleuro-pneumonia, and 118 by bronchitis;" but probably this tabulation must have generally rested on the unsupported assertions of the patients themselves, who doubtless consulted him or his father, Dr C. J. B. Williams, at variable periods after their illness had begun, and often when a considerable time had elapsed.

quantity of pus; but both lungs also showed scattered grey tubercles and patches of translucent grey consolidation, with points of caseation breaking down here and there into cavities. There would be no difficulty in citing many similar cases from our *post-mortem* records were it needful.

Another fact which indicates that catarrh is apt to lead to phthisis is the frequency with which, in children, pulmonary tuberculosis follows whooping-cough or measles.

We certainly have no proof of the occurrence of what is so often spoken of—a primary catarrh of one or both of the apices. But if tuberculosis is a modification of the inflammatory process due to the presence of bacilli which are especially apt to attack the apices of the lungs, there is nothing improbable in the supposition that irritation of the whole of the pulmonary tissue by cold may set up phthisis there, while failing to do so elsewhere. More than one instance has come before the author in which consumption has appeared to be distinctly traceable to moving into a damp, newly built house, the patient having been quite well before.

Nevertheless, caution is required in accepting the statements of patients as to the origin of their illness. In 1869 a young man died of phthisis in Guy's Hospital, who attributed the disease to his having slept with his window open one night five weeks before. He admitted, however, that nine months previously he had been ill for a week with a cough. At the autopsy, beside very acute pneumonic phthisis, there were found at the left apex old clustered grey and black tubercles and cavities. This, therefore, could not be cited as a case caused by exposure to cold.

viii. *Damp soil.*—It is most probably by the increased liability to attacks of slight bronchial and pulmonary catarrh which must necessarily follow residence in damp situations, that we can account for certain remarkable facts with regard to the influence of damp and ill-drained localities upon the frequency of phthisis. In 1862 Dr H. I. Bowditch, of Boston, took occasion, in addressing the Massachusetts Medical Society, to bring forward a mass of evidence which led him to believe that, in that State, consumption, instead of being equally distributed, prevails especially in such places as are situated upon a damp soil, and seldom occurs when the soil is dry. This evidence consisted chiefly of an analysis of the replies of medical men living in 183 townships to inquiries as to the frequency of the disease in their practice, and as to the moisture or dryness of the localities. It also included some striking instances in which phthisis had carried off in succession a number of persons living in certain houses surrounded by wet meadows, or placed by the side of a millpond, or shut in by trees.

Far more conclusive, because resting upon an accurate statistical basis, is a body of facts which were collected by Dr Buchanan during the years 1865 and 1867 in England, and published in Mr Simon's ninth and tenth reports to the Privy Council. The inquiry began in a tour of inspection made for the purpose of ascertaining the results of sanitary works that had been carried out in twenty-five towns, containing an aggregate population of 606,186 persons. It was found that in several places there had been a great diminution in the general death-rate, and that the prevalence of enteric fever had become much less, especially where a good water supply had been substituted for a bad one, and where drainage works had displaced cesspools or middens. But in other towns it was by a decrease in the number of cases of phthisis that the good effects of sanitary improvements appeared to be manifested; and the particular change which coin-

cided with this result was found to have been drying the ground by drainage of the subsoil. The following table shows the amount of change in the death-rate from phthisis in twenty-four of the towns visited by Dr Buchanan:

Town.	Previous death-rate per 10,000 from phthisis.	Degree of change in death rate from phthisis.		Influence of sewage works on subsoil.
		In total population.	In females between 15 and 55.	
Salisbury	44⅛	−49 p. c.	?	Much drying.
Ely	32	−47 p. c.	?	Much drying.
Rugby	28½	−43 p. c.	−48 p. c.	Some drying.
Banbury	26⅜	−41 p. c.	−36 p. c.	Much drying.
Worthing	30½	−36 p. c.	−41 p. c.	Some drying.
Macclesfield	51½	−31 p. c.	−22 p. c.	Much drying.
Leicester	43½	−32 p. c.	−16 p. c.	Drying.
Newport	37	−32 p. c.	−13 p. c.	Local drying.
Cheltenham	28⅜	−26 p. c.	−25 p. c.	Some drying.
Bristol	33½	−22 p. c.	−18 p. c.	Some drying.
Dover	26½	−20 p. c.	−18 p. c.	Local drying.
Warwick	40	−19 p. c.	−10 p. c.	Some drying.
Croydon	*	−17 p. c.	?	Much drying.
Cardiff	34⅜	−17 p. c.	?	Much drying.
Merthyr	38½	−11 p. c.	−12 p. c.	Some recent drying.
Stratford	26⅜	− 1 p. c.	− 4 p. c.	Some local drying.
Penzance	30⅜	− 5 p. c.	0	No change.
Brynmawr	28½	+ 6 p. c.	− 8 p. c.	No notable change.
Morpeth	30½	− 8 p. c.	+12 p. c.	No change.
Chelmsford	32½	0	+11 p. c.	Slight drying.
Penrith	39½	− 5 p. c.	+27 p. c.	No change.
Ashby	25½	+19 p. c.	−10 p. c.	Some drying.
Carlisle	32	+10 p. c.	+11 p. c.	Drying (with local defects).
Alnwick	28½	+20 p. c.	+36 p. c.	No drying.

It is perhaps worth while to give some details as to one or two of these towns, since the full significance of the change that has been effected in them by drainage works can hardly be appreciated otherwise.

In 1851 Mr Rammell had reported of Salisbury as follows: "Numerous streams of water, supplied by the Avon, run through most of the streets. . . The soil is a porous gravel, containing everywhere a great deal of water, which rises to within a short distance of the surface. There have been several instances of the cathedral being flooded by the water of the subsoil. The foundations of the houses are almost without exception damp." The water supply is from wells "dug about eight or ten feet deep, the water rising to within three or four feet of the surface." Mr Middleton had drawn public attention to the same point in his address to the British Association in 1864. Apart from the bad system of drainage which the canals of Salisbury were made to serve, he clearly showed that they were also injurious by keeping the subsoil constantly damp.† In 1853 efficient drainage works were begun in Salisbury, and they were completed in 1855. In 1865 Dr Buchanan writes as follows: "The subsoil is now dry, and cellars of considerable depth can now be made in different parts of the town which do not become flooded at any time. On an average the sub-

* "Phthisis and other lung-diseases" together were previously 59⅛ p. c. Reduction of this rate is what is above given for Croydon. (Ninth Report, 1866, p. 48.)

† "Benefits of Sanitary Reform at Salisbury," 1865.

soil water has been lowered four or five feet all over the city. The cathedral has never been flooded since the drainage works. As is shown in the table, the annual death-rate from phthisis fell in Salisbury from 44½ per 10,000 in 1844–52, to 22⅔ per 10,000 in 1857–64."

Of another town, Banbury, Mr Rammell had reported in 1850 in the following terms :—"The drains are not all at a sufficient depth to drain the cellars of the houses. In the principal streets of the town water is raised from the cellars into the drain by buckets, and creates a nuisance." Sanitary operations were begun there in 1854. "At present," says Dr Buchanan, writing in 1865, "the sewers and drains all act efficiently. . . . Many of the wells of the town have been dried by the sewers." As appears from the table, the phthisis death-rate for 10,000 has declined from 26⅔ in 1845–53 to 15⅗ in 1857–64.

It must of course be understood that drying of the subsoil is not the only improvement that has been made in these towns. Excreta have at the same time been carried off from the houses, a good water supply has often been provided, and overcrowding has been diminished. Now, as regards removal of filth it does not seem that this has acted in reducing the death-rate from phthisis. Dr Buchanan placed the several towns in another list, according to amount of decrease in the mortality from enteric fever, and the order in which they stand in the phthisis-list is by no means the same as that in the fever-list, which appears to be more affected by removal of filth than by anything else. Many of the towns lower down the phthisis-list, such as Alnwick and Brynmawr, had made very good arrangements for carrying away excreta. And, on the other hand, Worthing and Rugby, both of which stand well in the phthisis-list, are very low in the fever-list. The cases in which "sanitary works" have failed to reduce the death-rate from consumption are chiefly those in which the soil previously contained little water (as at Penzance and Brynmawr), and those in which the deep drainage was effected by impervious pipes laid down in compact channels (as at Penrith and Alnwick) so that no extensive soil-drainage could occur either through or beside them.

The importance of these observations appeared to be so great that, in 1867, Dr Buchanan was directed by the Privy Council to make a special investigation in the three south-eastern counties, Surrey, Kent, and Sussex, for the purpose of determining whether any relation could be traced between the prevalence of consumption and the state of the soil as regards moisture. These three counties were chosen because in them only had the Geological Survey then minutely mapped out the surface ; but no part of England could have been better adapted to the purpose, on account of the great varieties of soil found there and the comparative absence of other differences between the several districts.

The first point was to ascertain the extent to which phthisis prevailed in different parts of the three counties. The basis for this part of the inquiry was afforded by the Registrar-General's Returns. Of course they cannot pretend to exact pathological accuracy. But seeing that in each registration district the certificates are furnished by several medical men, it is not likely that any serious error can result from their being used for the purpose of comparing the death-rate from so common a disease as consumption in one district with that in another. Moreover, if cases of phthisis are wrongly returned under any other head, it must be generally under that of bronchitis or of some other lung disease. Dr Buchanan, therefore, took pains to con-

sider the mortality from lung diseases in general, as well as that from phthisis, before he drew his conclusions.

Corrections had to be made in the returns for certain districts on account of the presence of camps or dockyards, causing the population to have a large excess of men in the prime of life; in others the returns were vitiated by their containing hospitals or asylums; in others, again, by their being resorts for invalid visitors; eight districts were set aside as being subject to one or other disturbing influence, which rendered their true consumptive death-rate uncertain. There were left fifty other districts which it was believed could be fairly compared with one another.

In instituting this comparison, Dr Buchanan first classified the several districts as having mainly soils *permeable* by moisture, or soils of such a character that water is unable to escape from them, so that they might be called *retentive*. He then massed together the fifty districts into five groups of ten each, according to the greater or less prevalence of phthisis in them, and in this way he obtained the following table.

Groups of districts.	Proportion of population (per 1000) residing on	
	Permeable soils.	Retentive soils.
A. With least phthisis	909	91
B. With next least phthisis . . .	877	123
C. Middle as to phthisis . . .	795	205
D. With more phthisis	792	208
E. With most phthisis	642	358

He pointed out that this tabulation, apart from arithmetical objections, must be corrected by certain geological considerations. Where the soil is pervious, its being moist or dry must depend entirely upon whether the water which reaches and sinks into it can escape through or from beneath it. It is no advantage for a place to be situated on gravel if the subsoil water cannot get away. Roughly, one may say that such a district will be *dry* in proportion as it *lies high* in relation to the places round it, *damp* in proportion as it *lies low*. On the other hand, among impervious soils, the question of dryness or moisture is almost entirely one of the *inclination of the surface*. Even among clays there is a great difference as regards dampness, according to the flat or sloping character of the ground.

A more exact comparison between retentive and pervious soils in regard to the prevalence of phthisis is afforded by a limited area, the Wealden, which in part is formed by the Weald clay, in part by the Hastings beds of alternate sands and clays. There are, indeed, no districts wholly of sand to contrast with others wholly of clay; but there are great differences in the proportion of the two soils in different districts. How closely these differences correspond with differences in the consumptive death-rate appears from the table on the opposite page.

The districts are arranged in order of the death-rate from phthisis, those being placed highest in which it is least. Where there are gravels over the Weald clay the figure is divided between the last two columns, it being presumed that they occupy an intermediate position.

Still more striking perhaps are certain comparisons between particular sets of districts which differ—if *pervious*, in being *high-lying* or *low-lying* respectively; if impervious, in being *sloping* or *flat*.

District (in order of phthisis death-rate).	Percentage of population resident on						Total on	
	Higher beds, mostly Lower Greensand.		Weald clays.		Hastings beds.		Sands + half gravels over Weald clay.	Clays + half gravels over Weald clay.
	Sands.	Clays.	With gravel.	Without gravel.	Sands.	Clays.		
Hastings	...	...	...	...	95	5	95	5
Cranbrook	...	...	1	6	84	9	84	16
East Grinstead	...	...	...	12	82	6	82	18
Tunbridge	...	1	24	7	64	4	76	24
Hambledon	49	...	20	31	...	...	59	41
Battle	...	...	...	...	80	20	80	20
Rye	...	4	...	...	79	17	79	21
Maidstone	43	1	45	11	...	...	66	24
Cuckfield	21	1	...	25	48	5	69	31
Uckfield	...	...	...	1	82	17	82	18
Hailsham	...	...	...	34	61	4	61	38
Ticehurst	...	...	...	...	67	33	67	33
Tenterden	...	...	...	29	42	29	42	58
Horsham	...	...	...	56	44	...	44	56
Petworth	30	...	...	70	...	...	30	70

(1) As between *high-lying* and *low-lying pervious* soils, a contrast is afforded by districts formed by the chalk. No soil is drier than chalk when it has a good elevation; at its higher parts there are no streams, water cannot be reached by ordinary wells, and the people can only obtain a water supply from less elevated ground. In many districts, however, the bulk of the population who live on chalk occupy valleys with the water-line in the chalk not very far below their houses; and in the south of Sussex a still greater degree of wetness is reached, for a large part of this area reckoned as chalk is a flat plain on the sea level, covered by gravel, with the dip of the chalk here and there inland. Accordingly, we find the phthisis death-rate for North Aylesford and Dover (both of which lie high) to be 289 and 296 respectively; while those for Worthing, Lewes, and Westbourne (all of which lie low) are 419, 426, and 498. In general, Dr Buchanan adds, the connection between a low death-rate from phthisis and elevation of the chalk area is unmistakable.

So, again, with regard to the population living mainly on the Lower Greensand, there is a great contrast between a southern tract of this formation which lies low and the hills which are made up of it elsewhere. And a corresponding difference exists between the phthisis death-rates of Thakeham, Midhurst, and West Ashford (454, 455, 421 respectively) and those of Reigate and Godstone (337 and 282).

(2) As between *sloping* and *flat impervious* soils, a capital contrast is presented by two widely distributed tracts of clay, the London clay and the Weald clay. The former in its main extent throughout the three counties is disposed in long slopes or hills; the latter constitutes sometimes gently undulating, but more often flat and level ground. The former is covered over large areas by gravel reaching to many feet in thickness; the latter has only very level gravels, occupying its undulations, and these are seldom thick. The former generally has the direction of drainage from other beds away from it; the latter is always bounded immediately to the north and to the south by higher grounds, so that other beds drain more or less into it.

All these considerations show that the London clay is commonly much less wet than the Weald clay. The difference between the two formations in respect of their phthisis death-rate is, Dr Buchanan says, unmistakable. All districts that have even a third of their population on Weald clay have a high mortality from consumption, whereas there may be in a district a notable proportion of uncovered London clay without any like result.*

ix. *Direct injury.*—Whether this can be enumerated among the causes of phthisis is very doubtful. In 1880 there died at Guy's Hospital a man, aged thirty-eight, a patient of Dr Moxon. He had been a cab-driver, but was said to have been very moderate in his habits; there was no history of consumption in his family. Four weeks before his admission his cab had been upset and turned over upon him. Afterwards he spat a little blood, and three days later he brought up half a pint of blood. Of course he regarded his illness as the direct result of the accident, but the physical signs were exactly like those of phthisis affecting the left lung. When he had been in the hospital two months and a half he died. The left lung was found to be destroyed by a phthisical affection of "pneumonic" character. The upper lobe and the upper part of the lower lobe were hollowed out into a number of cavities; the rest of the lower lobe was consolidated by a pinkish-grey infiltration, scattered in which were many yellow tubercles and caseating patches with sinuous edges. But the right lung was affected with a more chronic form of the disease; in the upper lobe was scattered much pigmented grey tubercle; there were also some caseous tubercles, and one or two small vomicæ. The tubercular nature of the disease was confirmed by the fact that the small intestine contained ulcers which showed yellow submucous tubercles in the floor. In the larynx, too, there was a deep ulcer over the left arytænoid cartilage. Evidently the accident can only have accelerated a morbid process already in existence.

In four other cases phthisis followed an accident; in one there was fracture of ribs, in another fracture of the collar-bone, in the other two injuries of an undetermined kind from a fall into the hold of a ship and from a railway accident respectively. But in none of these cases is there any proof that the lungs were previously healthy; nor, indeed, is it certain that there was any closer connection between the accidents and the pulmonary disease than mere coincidence (*cf. supra*, foot-note, p. 1068).

The analogy of some other tubercular affections, as those of the kidney and the joints, would lead one to admit that an injury to the chest might set

* See on this subject and the ætiology of phthisis generally the valuable Milroy Lectures before the College of Physicians by Dr Ransome, of Manchester, in March, 1890.

It is right to mention that Dr Kelly, the Medical Officer of Health for East Sussex, has expressed doubts of there being any intimate relation between dampness of the soil and phthisis. He finds that in the years 1861–70 the order in which the several districts have to be placed in regard to their death-rates from phthisis is different from that given by Dr Buchanan for 1851–60. He points out that most of the impervious beds are to the north of the South Downs, and that consumption seems most common in places which are bleak and exposed as well as damp. He insists on the fact that in West Sussex (as indeed throughout England and Wales) there has of late years been a great decrease in the mortality from consumption, although there has been no change in the drainage of Sussex. Dr Kelly is inclined to attribute it mainly to the progress which has taken place in the social state of the rural population. Hirsch doubts the direct effect of good draining, and quotes the experience of Berlin, Dantzig, and Brunswick, of improved drainage being followed by increased death-rate from phthisis.

It is much to be desired that by a Royal Commission, or any better means, an extensive and searching official investigation over long periods of time should be undertaken, which might at least settle the facts upon which must rest the significance of damp soil and its share in the ætiology of phthisis.

up phthisis. But analogy is an uncertain guide, and considering how common are injuries to the chest, and how common a disease is phthisis, instances of a connection between them ought to be more frequent if it existed.

Opposing pathological conditions.—To complete our survey of the ætiological relations of phthisis, it is necessary that brief allusion should be made to certain conditions which have been, or still are, supposed to be *antagonistic* to its development.

One of these is habitual exposure to *malaria*. This opinion is now generally discredited in England, but Walshe brings considerable evidence in its favour.

The simultaneous occurrence of *carcinoma* and of tuberculosis is rare, but probably only due to the facts that each of these diseases is usually fatal, and that one of them chiefly attacks older, the other younger persons. Nine or ten cases occurred at Guy's Hospital between 1855 and 1874 in which more or less active phthisis had been present in patients who had also had cancer of the stomach, or womb, or œsophagus, or some other organ. One was a woman aged twenty-two, another a man aged twenty-four, a third a man aged thirty; the rest were older, and one had reached sixty-seven.

Antagonism between phthisis and *gout* has also been generally accepted, and probably not without reason. Both diseases are common in men between twenty-five and forty-five, yet cases of their concurrence in the same patient are rare. It would be interesting to know how often deposits of lithate of soda in the great toe joint are found in bodies which also show evidence of obsolete or recent disease of the apices of the lungs. That gout and active phthisis may coexist is proved by three cases observed during life and recorded in the 'Guy's Hospital Reports' for 1873 (vol. xix, p. 338), while in a fourth patient with gouty deposits in the joints signs of old phthisis and some recent clusters of tubercles were discovered in both lungs.

On the other hand, it appears to be indisputable that at least one kind of valvular *disease of the heart* is an almost complete bar to the development of phthisis. Mitral stenosis is exceedingly common in young persons, and it often fails for several years to affect the general health to any marked extent. That this lesion should be scarcely ever found in those who die of consumption is therefore a remarkable fact. Traube could not remember to have met with an instance. In our records of *post-mortem* examinations at Guy's Hospital, from 1854 onwards, only four examples occur. One was in a man, aged forty-two, whose mitral orifice was so narrowed, as the result of rheumatism, that it would only admit two fingers.* Traube speaks of having seen several cases in which persons with regurgitant disease of the aortic valves became affected with phthisis.

Whatever may be the explanation of the rarity of consumption in cases of mitral stenosis, it can hardly depend upon the venosity of the blood, as Rokitansky thought. For it is now well known that those who have congenital narrowing of the right sigmoid orifice are very apt to die of tubercular disease of the lungs. Traube says that he has seen two examples of this; and two occurred in the author's practice at Guy's Hospital.

Traube's view was that mitral stenosis, by causing serum to exude into the pulmonary tissue, prevents the occurrence of caseation; and Dr Hamilton, in the 'Practitioner' for 1880, throws out a similar suggestion.

* One of Dr Moxon's beautiful drawings in our pathological theatre shows how ill tubercular inflammation of the lungs thrives in cases of mitral disease.

It has been generally believed that pulmonary *emphysema* is unfavourable to the development of tubercle, and there is reason to believe that this is the fact. Even when emphysema supervenes upon primary phthisis the latter disease seems to be checked in the part so affected.

The supposed immunity of humpbacks from phthisis is not borne out by observation. Angular curvature of the spine usually depends on tubercular caries of the vertebræ, and is frequently followed by tubercular disease of the lungs (see a paper by the author in the 'Guy's Reports' for 1874).

Age and sex.—Phthisis may occur at all periods of life. The idea that it is a disease confined to young adults is inaccurate. At Guy's Hospital there has been little if any diminution in the number of fatal cases for each quinquennial period up to the age of forty-five. Several cases have occurred in persons between sixty and seventy, and two at the age of seventy-two. Probably these patients had phthisis in youth. At Copenhagen the proportion of deaths from phthisis to those from other causes increases with advancing years, and the same statistical result is reported from Würzburg and other parts of Germany. Statistics of Dr C. J. B. Williams's patients appear to show that, other things being equal, the *duration* of phthisis increases with age, and it is a matter of general observation that the disease in children runs an acute course, while senile phthisis is usually chronic. This would bring the average age at which it begins earlier than the statement of ages at death would indicate.

The period at which appeared the first decided symptoms of what afterwards developed into phthisis was found by Dr Williams to be distributed as follows among 1000 private patients:—195 cases at an age below twenty (of these only 13 under ten), 667 between twenty and forty (and of these 418 below thirty), 94 between forty and fifty, 30 between fifty and sixty, and in 14 cases symptoms only began at an age above sixty. The average period was later in men than in women.

More men than women die of consumption in the hospitals of London, although the Registrar-General puts the rates of males to females in the population generally as 3·77 to 4·13. Of Dr Williams's 1000 cases 625 were males and 375 females. Among the out-patients at the Brompton Hospital, Dr Pollock found 60·7 per cent. male and 39·3 per cent. female.

It must not be assumed that sex or age constitutes a predisposing cause of the disease in a strict sense of that term. The question may be of the more frequent operation, at different periods of life and in one sex rather than the other, of the various causes that have already been enumerated, particularly exposure and catarrh.

In women consumption shows itself, as a rule, earlier than in men, and more frequently runs a rapid course.

Geographical distribution.—Phthisis is, on the whole, more prevalent in temperate regions than in those which are very hot or very cold. It is endemic throughout Europe, and is most abundant in *large cities*, particularly in Paris and Vienna; but also in New York and Philadelphia, in Alexandria, and in Melbourne. It is probably as common in the United States, in China, and in Australia as in England; but is less so in the East Indies and within the tropics generally, although imported cases from Europe are said to be very unfavourably influenced by a hot climate. It is extremely rare in Iceland, in the highlands of the Andes, in New Zealand

and in some oceanic islands, in Syria, and in Upper Egypt. It is rare in swampy and malarious regions, as was first pointed out in 1812 by Dr Wells (the author of the essay on 'Dew'), and since confirmed by Boudin and many other observers. No quarter of the globe and no race of men is known to be completely exempt from consumption.*

Phthisis is well known among the lower animals, particularly the Quadrumana and the Ruminants (see, however, Mr J. B. Sutton's records, 'Path. Trans.,' vol. xxxvi, p. 546, and Dr Creighton's monograph on 'Bovine Tuberculosis in Man').

Prophylaxis.—It is doubtful how far it is possible to guard directly against the invasion of bacilli. Suspected milk may be boiled, crowded rooms may be ventilated, and the sputa of phthisical patients may be disinfected, but at present there is want of proof that "consumption hospitals" are dangerous, or that the health resorts for "poitrinaires" like San Remo and Davos, are more unhealthy than other places. Further investigation may show that though "infective" phthisis is not "contagious" (*i. e.* easily caught) except under special circumstances—as, for example, is the case with two well-known infectious diseases, syphilis and enteric fever.

The hereditary transmission of consumption is not altogether beyond the scope of prophylaxis. The physician's advice and the enlightened general opinion which he in time can produce may do much to prevent the intermarriage of cousins belonging to a phthisical family, and to dissuade from marriage those in whom symptoms of the disease have already appeared.

If a mother is known to be already phthisical, or even disposed thereto, it is a grave question whether she should be allowed to suckle the children.

Care should be taken that the residence of those who have tubercular tendencies should be on a dry soil. The rooms in which they live or sleep or work should be airy, well ventilated, and so situated as to be exposed to sunlight. Their food should be nutritious and fattening; and in feeding children it would probably be desirable to have all the milk boiled. They should have plenty of exercise in the open air, and they should be accustomed to exposure to the weather, but within reasonable limits. Cold bathing is advisable, provided that there is always a good reaction after the bath.

Special care should be taken during convalescence from whooping-cough and measles; and the recurrence of attacks of bronchial catarrh should be sedulously avoided. The habit of breathing with the mouth shut is an effectual preservative from most of the chances of catarrh, and possibly may make bacillary infection more difficult. Study, whether in preparing for examinations or in the pursuit of professional eminence, should be kept within bounds. Indeed, as adult life is approached, the necessity for moderation in all things should be impressed on everyone who would avoid the risk of phthisis. Temptations to intemperance and to dissipation must be resisted; but often the tendency of those who have a phthisical predisposition is rather to asceticism than self-indulgence, and this may be scarcely less physically harmful.

The son of a phthisical miner, or potter, or weaver should avoid such kinds of work, and country life should be preferred to the close and sedentary life of towns.

* See the diagrams in Dr Ransome's second lecture ('Brit. Med. Journ.,' March 8, 1890).

The diminution of the mortality from phthisis which has been observed in England (from 2567 per million in 1858–60 to 1541 per million in 1888), and also in New England (from nearly 4 pro mille in 1857 to about 3 pro mille in 1883), depends probably upon better drainage and better lodging; and, if so, is a great encouragement to prophylaxis in these directions.

Treatment.—This naturally falls under three heads:

(1) We must seek to check the early symptoms of phthisis, and to bring the disease to a state of obsolescence.

(2) A strenuous effort must, if possible, be made to arrest further progress or to prevent a relapse.

(3) If this fails, we must aim at retarding the fatal issue to the farthest attainable limit, and at relieving the most distressing symptoms.

(1) When phthisis sets in with inflammatory symptoms, with or without hæmoptysis, the patient should be put to bed and kept absolutely at rest. He should be limited for a time to a very light diet, consisting mainly of milk, without wine or other stimulant. Whether hæmorrhage has occurred or not, a very good prescription, if there is much pyrexia, is Niemeyer's pill of quinine (gr. j), digitalis (gr. $\frac{1}{2}$), and opium (gr. $\frac{1}{4}$), to be taken every four or six hours. Tincture of iodine should be applied to the affected part of the chest, or even a blister, or the croton-oil liniment. It is almost an advantage to the patient, if he is to have phthisis at all, for it to set in with hæmoptysis; because then the real gravity of his condition is appreciated, and there is no hesitation in carrying out the measures which are necessary. "Catarrhal phthisis," in which one apex becomes quickly consolidated, has been held distinct from the "tubercular" form of the disease, because it yields so readily to treatment; and probably cases in which rapid consolidation occurs may run a more favourable course than others; but this may be because they are taken in hand more carefully and more energetically.

When the acute symptoms have passed off, the patient may go to the sea-side for a few weeks, or to some dry and healthy place inland, such as Tunbridge Wells or Malvern, or Ben Rhydding if the season be suitable. On the Continent a favourite plan is to send him to Lippspringe, near Paderborn, to drink the water of the lime spring there, or to Soden and other health resorts in the Taunus.

Sending a youth brought up in town to live in the country for several months, with strict attention to early hours, nourishing diet, and sedulous avoidance of all that is exciting, enervating, and exhausting, has often saved him from threatened consumption.

(2) It is impossible to insist too strongly on the importance of not letting slip the opportunity, which occurs in phthisis only at an early stage, of arresting its further progress, and of preventing a relapse. The measures by which this—the virtual *cure* of the disease—can be effected, involve, as a rule, a prolonged change of climate.

A long sea voyage, either to the Cape of Good Hope (or the highlands of Natal), or else to Australia by the Cape, or round the world. About twenty-three days are taken in going to the Cape, twenty-eight days to Natal; the voyage to Australia made in a steamer is about forty days, in a sailing ship, about three months. With regard to details, a knowledge of which is absolutely necessary to the invalid for whom a long voyage is recommended, information must be sought either in a little book by Dr Wilson, 'The Ocean as a Health Resort,' or in a series of papers by

Dr Faber, published in the 'Practitioner' during 1876–7. Sailing ships are preferable to steamers, one advantage being the greater length of the voyage, which renders the changes of climate less sudden and trying; while the greater cleanliness and quiet, together with the easier motion, are also strong recommendations. But, unfortunately, suitable sailing vessels are now seldom to be found. There is of course considerable heat in crossing the equator, and, on the other hand, during the latter half of the journey to Australia the weather is often cold and stormy. As large a part of the day as possible should be passed in the open air, and exercise on deck should be systematically taken. It seems to be important that the excessive appetite which generally arises should not be too freely indulged. A stay in Australia of from six weeks to three months should be made, at the end of which time the voyage home should, if possible, be made by the Cape, this taking generally three and a half or even four months. Dr Faber insists that no patient should be sent to the Antipodes who is not quite free from pyrexia in the evening; the climate of the tropics is very apt to cause increase of fever, and to render it continuous instead of hectic. It is also apt to bring on hæmorrhage, so that a marked disposition to hæmoptysis, or the presence of degenerated arteries are strong objections to a long sea voyage. The extent to which a patient is likely to suffer from sea-sickness cannot be foretold unless it has been proved by former experience; the result of a short trip across the Channel decides nothing.

Residence in an elevated mountain region, or at least in a dry bracing climate. It is impossible in the limits of this work to enter into details with regard to the climatic treatment of phthisis. The reader must consult the fourth edition of Dr Walshe's 'Diseases of the Lungs,' Dr Weber's translation of Braun's 'Baths and Waters,' and a little book by Mr R. H. Otter, entitled 'Winters Abroad;' also 'The Influence of Climate in the Prevention and Treatment of Pulmonary Consumption,' by Dr Theodore Williams; and Dr Weber's interesting Croonian Lectures on "The Hygienic and Climatic Treatment of Consumption," 'Lancet,' March, 1885.

Within the last few years it has become a common practice to send consumptive patients during the winter to Davos, a village situated in the Grisons, at an elevation of about 5200 feet above the sea. Other places, perhaps, might be found equally favourable; and St Moritz and Samaden, in the Engadine, had in fact been tried before Davos Platz, at least by English invalids. The Maloja Pass has more recently come into favour. The great peculiarity of the weather in these elevated stations is the stillness and the dryness of the air. In the shade the cold is extreme, but as the sun is very powerful, and as the sky is generally perfectly clear, patients are able to take exercise nearly every day—walking, skating, sledging, or "tobogganing." When sitting in the verandahs of the hotels the sunshine is hot. At night the double windows in the bedrooms are left slightly open; yet so motionless is the air that the temperature within scarcely falls below 50° Fahr., even when it is from 2° to 16° Fahr. outside. Many persons who are very liable to take cold elsewhere are free from the tendency at Davos. The proper time for a patient to arrive there is about the first or second week in October, or even earlier. It is generally supposed to be undesirable for him to remain after the beginning of April, when the snow melts. Unfortunately there is no little difficulty in saying where he should then go. He should on no account return to England before the first week in June; and during the interval the choice seems to lie between

Baden-Baden, Wiesbaden, Montreux on the Lake of Geneva, and Monte Generoso above Lago Lugano. The fact that hæmoptysis has been one of the symptoms does not appear to forbid sending a consumptive patient to Davos; but the actual presence of pyrexia is an objection, and still more so irritability of the larynx or trachea.

In America there are mountain climates in which phthisical patients derive great benefit without being exposed to extreme cold. This fact was pointed out long ago by Dr Archibald Smith, who practised for many years in Peru; indeed it seems to have been familiar to the Peruvians themselves, who regard the valleys of the Andes, from 8000 to 10,000 feet above the sea level, as almost omnipotent in the prevention and cure of consumption. As a general rule, it may be said that the nearer the equator the greater the elevation which is necessary to render a mountain region salutary in such cases. The chief resorts in the Cordilleras appear to be Huanuco and Jauja. Dr Walshe recommends, as more accessible, the plateau of Santa Fé de Bogotà in New Granada. A great peculiarity of this place is the equality of its climate at different seasons; the mean temperature of each quarter of the year is within a degree or two of 86° Fahr.

Other mountain regions to which phthisical patients may be sent are the table-lands of California and Mexico, Colorado,* Denver or Kansas City in the States, or Manitoba in Canada. One can hardly doubt that in the Himalayas also there must be valuable resorts; Dr Weber is inclined to think that the present military sanitaria there may not be at a sufficient elevation for the climate. The writer has seen excellent results from a whole phthisical family going to settle in Colorado.

A prolonged stay in the Southern Hemisphere during what would be the winter of Europe, but is of course summer there. Certain parts of Australia are very serviceable to phthisical patients. It must not, however, be imagined that a residence in the large towns is advisable. Melbourne, in particular, is apt to be intensely hot and dusty, with very rapid changes of temperature and cold winds; and Sydney and Brisbane are very hot. The best health resorts appear to be certain places in the interior of New South Wales, especially Bathurst, Goulburn, Boural (3000 feet above the sea), and Currajong, but, above all, the Darling Downs, in the south of Queensland, where the weather is cool, dry, and bracing. Tasmania and certain parts of New Zealand are suitable for consumptive cases; Hobart Town and Wellington or Auckland especially are well spoken of.

Certain parts of South Africa have climates which appear to be very favourable to phthisical patients, but no long stay should be made at the sea-coast; Mr Otter says not within 100 miles of it, nor at a less elevation than 1500 feet. The easiest way of reaching the interior is to land at Port Elizabeth, and to go on by Grahamstown to Cradock or to Bloemfontein, the capital of the Orange Free State. The difficulties are the badness of the roads, and the roughness of the accommodation. Bloemfontein has an exceedingly dry climate; the daily range of temperature is great, but this is said not to act prejudicially.

The working of change of climate, as a curative agent in phthisis, is still uncertain. In many instances the beneficial influence on patients who come from a distance finds its parallel in the fact that natives of the same district are very seldom, if ever, attacked. This appears to be the case, for

* See a paper on the "Climatic Treatment of Phthisis in Colorado," by Dr Charteris, of Glasgow ('Lancet,' November 26th, 1887).

example, in the high Alps, as well as in the valleys of the Andes, and until recently it was so in Australia, although the disease is now rife among the inhabitants of Melbourne and of other large towns there. But Dr Walshe and others have rightly insisted that there is no necessary connection between the two things, and there is no difficulty in finding countries, such as Iceland, to which one would not think of sending a consumptive patient, notwithstanding that the natives escape the disease. To the author it appears evident that the "aseptic" character of the air of a place cannot be the direct reason why phthisis should cease to advance in those who inhale it. Observations showing that meat remains fresh there longer than elsewhere are altogether inapplicable; the only case in point would be for putrefaction once started to be stopt. On the whole, it seems likely that the good effects of change of climate depend partly upon its improving the general health and increasing the resistance of the organism to the further progress of the disease, partly upon its protecting the patient from fresh attacks of bronchial catarrh. The number of bacilli found in the sputum is not diminished in Davos, according to Dr Theodore Williams.

(3) When the complete arrest of phthisis is no longer probable, much may still be done to prolong the patient's life and to give him relief from suffering. The climates which Dr Walshe terms *sedative* seem to have their chief use under such circumstances; for example, Madeira, and the Grand Canary, Pau, San Remo and some other places on the Riviera, Torquay, and Penzance. The climates which he classifies as *stimulant* include St Leonards and Hastings, various towns on the Riviera, Algiers, and even Egypt. Of Nubia he speaks in terms of the highest praise; and probably a voyage up the Nile is the best thing for patients who dislike cold, and who habitually feel better the hotter they are. That special benefit follows from the air having resinous smells from pine forests—as at Arcachon and Bournemouth—is easier to assume than to explain or to prove.

It must of course be remembered that phthisis sometimes becomes quiescent without any change of climate whatever. On the other hand, although it is a heavy responsibility to advise or allow a patient with confirmed phthisis to take a long sea voyage, or to spend a winter in Australia, in South Africa, among the Andes, or even at Davos, there is no question that, if he chooses to run the risks inseparable from such undertakings, he has at least a chance of unexpected benefit.

(4) In the last stages of phthisis, when palliation of symptoms is all that can be hoped for, we have practically to deal with cough, hectic fever, and diarrhœa, and with the other less constant or serious complications. At this stage it is most undesirable to send a patient away from the comforts of home. Opium, in one form or another is now the most valuable of drugs, and its effects are among the most striking instances of the power of the judicious practice of our art to alleviate and to soothe when it can no longer cure.

Physic and Diet.—The following is the list of drugs, regarded by Louis as important fifty years ago:—Steel, digitalis, common salt, carbonate of potash, chloride of ammonium, chloride of lime, hydrocyanic acid, creasote, and iodine. The inhalation of diluted chlorine gas, also, was fully considered and tried in fifty cases of phthisis in the Paris hospitals, and in no instance was there a successful result. Most of the other "remedies" are deservedly forgotten.

Quinine to give appetite or to moderate fever, preparations of iron for anæmia, digitalis to improve the pulse, sedatives to relieve cough or vomit-

ing, and laxatives and stomachics to help the appetite and digestion—these are rational and useful means of treatment; but chlorine has been followed into obscurity by the hypophosphites, by inhalation of carbonic acid gas,* and by inflating the patient's bowels with sulphuretted hydrogen.

Only one medicine has borne the test of long and wide experience, and this is not a specific remedy, but a kind of food, which has remarkable power of fattening the patient. *Cod-liver oil* was introduced for cases of chronic "rheumatism," but was first advocated as peculiarly useful in consumption by the late Dr Hughes Bennett. Now that it is taken by almost all consumptive patients, one is apt to underrate its real importance as a means of preventing emaciation and keeping up the strength. It is said to be less useful in proportion as the age of the individual is more advanced. It is best given after food, in doses of from a drachm to half an ounce two or three times a day. If it gives rise to nausea and vomiting, cream may sometimes be substituted for it, or glycerine, but they are not as good. With perseverance *small* doses of cod-liver oil can almost always be taken, at least during cold weather. It may be taken alone, or in orange or ginger wine, or as an emulsion. Chewing a bit of orange-peel is the best way to prevent the after-taste. In children cod-liver oil is often rubbed into the skin, but the smell is extremely unpleasant, and probably olive oil is just as good.

The first object in the early stages of phthisis, or of threatened phthisis which has not yet begun, is to improve the patient's *appetite and digestion*. This is one object of change of climate. If he gains weight he generally is doing well. Usually small doses of alkalies, with the milder and less astringent bitters and gentle laxatives, act best in this direction. The combination of carbonate of soda with rhubarb and calumba, long famous at Victoria Park and other hospitals, is excellent for the purpose. In the later stages laxatives must be administered with great caution, for nothing is then more injurious than diarrhœa. Sometimes strychnia and gentian or chiretta are borne well and help the appetite.

Fatty food, such as bacon, milk, butter, cream, dripping, is indicated. Wine with meals is as a rule desirable; but in early cases, before there is much cough, malt liquors, and particularly porter, are more useful. Brandy is, as a rule, best adapted for advanced cases, and even then should be given in moderate doses; but rum and milk, either before breakfast or between breakfast and lunch, is a well-known and useful way of giving a nourishing stimulant.

Treatment of special symptoms.—When severe *hæmoptysis* appears, the patient must be kept strictly in the recumbent position for two or three weeks at least. He should not be allowed to talk. His diet should be limited almost entirely to milk. He should have ice to suck, and everything that is given to him should be cold. Of styptics it is difficult to say which is the best; ergot, gallic acid, acetate of lead, opium, have each their advocates, and it sometimes seems necessary to try one after another. A large ice-bag may also be placed over the chest. When the hæmorrhage has ceased, the patient is very cautiously allowed to get up and to move about, and the amount of food is gradually increased, while the pulse and temperature are being carefully watched from day to day.

The *cough* of phthisis must be combated by the usual remedies. Most

* This remedy, recommended in 1795 by Dr Beddoes and James Watt the engineer, was once much employed, and has lately been revived.

prescriptions contain a small dose of opium or morphia, together with tolu, aniseed, benzoic acid, or some other of the so-called expectorants.

In most cases of phthisis there is early and marked *anæmia*, indicating the use of steel. This may be given as the sulphate, with small doses of Epsom salts, or in one of the milder preparations, or (if borne well) in the most efficient form, the muriated tincture, with glycerine and quassia or calumba. Arsenic may succeed when preparations of iron fail, and now and then has a remarkable effect in restoring appetite, and adding fulness as well as colour to the cheeks. Lately it has been given with salicylates.

Diarrhœa is often a most distressing complication, and does much to hasten the fatal termination. It is best treated by bismuth and soda, or by the aromatic chalk powder. Dover's powder is often useful, and occasionally, when the rectum is affected, a starch and opium enema or a compound lead suppository.

The only other symptom that needs special mention is the *night-sweating*. This may sometimes be checked by sponging the chest and the arms at bedtime with vinegar and water. Sometimes it ceases if a subcutaneous injection of atropine ($\frac{1}{200}$ to $\frac{1}{100}$ of a grain) is given at bedtime, or a dose of belladonna, oxide or sulphate of zinc, gallic acid, or strychnia. But in too many cases it persists in spite of all treatment.

Direct pulmonary applications.—Treatment by inhalation of compressed air,* of oxygen, or of other gases, is either useless or of very limited benefit. A mode of treatment, lately introduced by Dr Bergeon, of Lyons, gaseous enemata of sulphuretted hydrogen, has no theoretical probability to recommend it; it is unpleasant in its action, and after being tried (perhaps with more patience than was due to it) has been shown to be useless.

Of late, however, those who believe that the bacilli of tubercle are the efficient cause of phthisis have naturally attempted to destroy them by antiseptic methods. Iodoform and thymol have been given by the mouth with this object, and more recently sodic sulpho-carbolate and phenyl-propionic acid. Creasote, thymol, and eucalyptol have been inhaled by means of ori-nasal respirators (of which Sir William Roberts, Dr Burney Yeo, and others have devised ingenious forms); steam inhalers have been supplied with such antiseptic agents as creasote or carbolic acid, and similar solutions have been administered by Siegel's spray apparatus. Sometimes apparent benefit to the local and general symptoms has resulted, and the number of bacilli in the sputum has diminished. But experiments in the laboratory show that Koch's bacilli are extremely difficult to kill, and survive prolonged immersion in germicide solutions. Iodine and corrosive sublimate seem to have the most power in destroying them. Perhaps the most ingenious method of "bacillicide" treatment is that introduced by Dr Cantani, of Naples, who introduces the common *Bacterium termo* of putrefaction into the lungs in a spray, with the hope of its destroying the specific bacillus. Unfortunately the latter is the more powerful of the two; and Dr Theodore Acland reported, after a visit to Naples, that the results of this treatment are negative.

The eminent pathologist who discovered the bacillus of tubercle has been long engaged in search after some remedy which may render the organism refractory to the growth of the parasite; and it is reported that definite results have already been obtained in Professor Koch's laboratory.

* See some recent lectures by Dr T. Williams ('Brit. Med. Journ.'), 1885, vol. i, p. 769.

MILIARY TUBERCULOSIS OF THE LUNGS.—Both from a clinical and from a pathological point of view it is desirable to treat apart from cases of ordinary phthisis—in which tubercles spread through the lung from the apex downwards and produce softening and excavation—those of miliary tuberculosis, in which each tubercle appears to be the result of the deposition in the pulmonary tissue of a particle of virus (probably a bacillus, or the spore of a bacillus) brought to the organ from elsewhere by the blood-current. The cases which most demand attention in this chapter are those in which the lungs, previously healthy or but slightly affected with phthisis, become suddenly the seat of such immense numbers of tubercles that acute symptoms arise and death by asphyxia follows.

It must, however, be remembered that other organs are almost always attacked at the same time. If tubercles appear in the membranes of the brain, they generally (but not invariably) give to the disease its main clinical features. If the peritoneum is much involved, the abdomen alone may appear to suffer. So that it is often almost an accident whether a case is regarded during life as one of tubercular meningitis, or tubercular peritonitis, or miliary tuberculosis of the lungs; while there are other cases which run their entire course without definite clinical localisation. Nor is the severity of the pulmonary symptoms always proportioned to the number of miliary tubercles in the lungs. In cases clinically classified as tubercular meningitis the lungs are often found as full of tubercles as the pia mater.

Anatomy.—With regard to the morbid anatomy of miliary tuberculosis of the lungs there is little, if anything, to add to what was stated in the chapter upon Tubercle in general. As was there remarked (p. 319), many cases occur in which the greater abundance and more advanced state of the tubercles in the upper lobes, than towards the bases, prove that the proclivities of the pulmonary tissue in different regions produce their effect on this disease as well as in phthisis. It has also been noticed that when a general outbreak of miliary tubercles occurs in a patient who previously had the apex of one lung affected with phthisis, the tubercles in that lung are more numerous (or perhaps larger) than in the opposite one. The characters of the tubercles themselves vary widely in different cases. Sometimes they are grey, and tend not to caseation, but to fibrous change or to horny induration (p. 307). Sometimes they become cheesy almost as soon as they are formed. In some exceptional instances, and only towards the apices, they are found to have already softened in their centres with minute vomicæ.

Physical signs.—These are in most cases vague and doubtful.

In all probability miliary tubercles are never set sufficiently close together, even in the apex of a lung, to impair the percussion-resonance of the corresponding part of the chest. Sometimes it appears doubtful whether the sound is not slightly dull beneath one or both of the clavicles; but if this is so, the dulness is most likely due, not to the tubercles themselves, but to the collapse of the surrounding pulmonary tissue. Dr Eustace Smith remarks that in children this interpretation is borne out by the fact that variations may be observed from day to day, the resonance becoming good where it had been deficient; and the case of an adult patient will be presently mentioned in which the same thing seemed to occur. Again, it is not uncommon for the presence of pulmonary emphysema to render the percussion-sound over-resonant; and the progressive emaciation of the patient makes the still spongy parts of the lung give a more resonant note.

In many cases any dulness under the clavicle is due not to the acute tuberculosis, but to the preceding chronic phthisis.*

With the stethoscope one may be able to detect absolutely nothing abnormal, even when tubercles exist in enormous numbers. But in some cases, especially towards the apex, the vesicular murmur has a harsh quality, the cause of which is not apparent. More frequently the auscultatory signs of bronchitis are present, sometimes to an extreme degree. Not only may sibilus and rhonchus be audible, but also abundant moist sounds. The expiration, too, may be prolonged and wheezing. In such cases the smaller tubes are found reddened and filled with muco-pus—clear proof of bronchitis. Sometimes the moist sounds are so bright and clear—so consonating in quality—over the upper lobes, that it is difficult to believe that there is not diffused infiltration, with "breaking up" of the pulmonary tissue. In one such case which occurred at Guy's Hospital in 1874 there were in fact a large number of small cavities, especially in the left apex. These were evidently of older date than the general eruption of miliary grey tubercles, which filled every organ in the body, and it appeared from the history that the patient had had a cough for three months, whereas his more acute illness began only ten days before death. But in another case, in 1868, it is reported that there were mucous râles at the left apex, gurgling at the left base, and "pneumonic crepitation" over the right upper lobe; and yet the tubercles were nowhere seen softening, the only source of the moist sounds being pus in the smaller tubes. In many instances the autopsy has shown that the lesions were much less advanced than had been thought during life. This occurred in a woman, aged twenty-five, in Guy's Hospital. When she was admitted on July 19th, 1882, the only physical sign was a slight crackling sound heard at the right apex after she coughed. However, on the 28th there was a marked crepitant râle in both upper lobes, and especially along the anterior edges of the lungs; and during the next three or four days its character became so "consonating" that we were almost disposed to look upon the disease as acute pneumonic phthisis rather than as miliary tuberculosis. But at the autopsy, made on August 4th, the lungs, though bulky and œdematous, everywhere contained air; the tubercles were discrete, and caseous only in the upper lobes; the tubes yielded a frothy fluid.

Clinical symptoms.—These fall under two heads: first, pulmonary symptoms; and secondly, pyrexia of a peculiar kind.

There is always more or less troublesome *cough*, generally short and hacking, and often dry.

The *sputum* when present often consists of clear mucus; occasionally it is muco-purulent. There are not infrequently streaks of blood in it, and

* Jürgensen described in the 'Berliner klin. Wochenschrift' for 1872 a case in which during five days he heard over a large part of both sides of the chest a peculiar soft, rubbing sound, perceptible also to the touch; when death occurred, two days later, the only cause to which this sound could be attributed was the presence of a number of miliary tubercles situated on the right side beneath the pulmonary pleura, which was free, and on the left side in the substance of adhesions which obliterated the pleural cavity. From the very first day the soft quality of the sound led him to conclude that it was produced by miliary tuberculosis, and not by pleurisy. The patient complained of no pain, and could draw a deep breath without embarrassment. Jürgensen thinks that in future cases a positive diagnosis may safely be based upon this sign. Burkart has since stated (in vol. xii of the 'Deutsches Archiv') that he has twice detected a rough friction-sound, due to the presence of obsolete tubercles.

still more rarely it may be "rusty," like the sputa of acute pneumonia, or even plum-coloured. Actual *hæmoptysis* in any considerable quantity is not common. But in 1869 there was brought into Guy's Hospital the dead body of a child, aged five, who was said to have been well on the previous evening, and to have eaten some herring for supper. In the course of the night it was found to have brought up blood, and to be in an alarming state, and it died on its way to the hospital. An autopsy showed that there was an acute general tuberculosis; and some of the tubercles in the lungs were already caseating, especially in the upper lobes. No definite source for the bleeding could be discovered; the pulmonary tissue was mottled with blood drawn into it by inhalation. In all probability the cause of hæmoptysis in such cases is the extremely congested state of the vessels immediately around the tubercles, which often gives them the appearance of being surrounded by a reddish-brown border after death; in fact, obvious points of capillary hæmorrhage may sometimes be seen, not only in the lungs, but in other organs.

Far more significant than cough or sputum is *dyspnœa*. At first the breathing is only hurried; the number of inspirations in the minute gradually increase up to fifty or sixty, and in children to eighty or ninety. In the woman aged twenty-five, whose case was mentioned above, it was counted at fifty-six on the very day of her admission. After a time the patient becomes conscious of shortness of breath; there is orthopnœa, the movements of the thoracic muscles are forced, and the nostrils work. The cheeks, the lips, the fingers, and the nails are of a purple colour. This cyanosis, perhaps more than any other symptom, suggests the idea of pulmonary tuberculosis, when there is no long-standing emphysema or heart disease to account for it. Very seldom is it stated to have been absent.

Sometimes albumen appears in the urine. There is not infrequently slight œdema of the lower limbs, and the face becomes puffy and swollen. In the case of the young woman already referred to, the urine contained sugar during the first few days after her admission into the hospital, the proportion being on one occasion 0·4 grain, and on another occasion 0·265 grain in the ounce. Senator's experiments seem to show that this cannot be attributed to deficiency of oxygen in the blood; and, indeed, as the case went on and marked cyanosis developed itself, the glycosuria ceased.

Pyrexia seems to be invariably present, but it varies greatly in its degree and in its course. Sometimes the temperature ranges up to 104° or 105°, but more often it remains at a lower level, perhaps not at any time exceeding 102°. Its progress is irregular. For two or three days there may be scarcely any differences in the thermometric readings at different periods of the twenty-four hours; and then the usual diurnal variations may appear in an exaggerated form, or what is termed the *typus inversus* may show itself, the morning temperature being higher than that of the evening. Brunniche is said to have observed this in fifteen cases out of seventeen.*

The onset of the pyrexia is usually gradual, and the patient does not take to his bed until he has been ailing for some days. But Rühle, in 'Ziemssen's Handbuch,' speaks of an initial rigor as not infrequent. There are the ordinary early febrile symptoms of headache, malaise, depression,

* Jürgensen makes it a point that the pyrexia does not yield to tepid baths or to antipyretic remedies so readily as in the specific fevers. This opinion, however, seems to have been partly theoretical, and based upon the idea that in tuberculosis the high temperature of the body generally is the result of the local morbid process, just as when there is inflam-

intense thirst, and loss of appetite. The skin is often wet with perspiration. Epistaxis occurs in some cases, and herpes may appear about the mouth.

The *pulse* is generally very rapid—often out of all proportion to the height of the temperature. There may be a flush on the cheeks, but the face is more usually pale before it becomes livid. At one time it was taught that in miliary tuberculosis enlargement of the spleen is exceptional. But all observers seem now to be agreed that some degree of swelling of the organ is usually to be detected by careful percussion. Rühle says that if tubercles are developed in the spleen it may be tender on pressure, and become as large as in enteric fever. This was the case in a patient under the writer's care in December, 1888.

Towards the end a typhoid state may develop itself, with sordes, a dry brown tongue, subsultus, delirium, and coma. Death is sometimes preceded by a rise of temperature, sometimes by a fall and by collapse.

Diagnosis.—One is often helped in the recognition of miliary tuberculosis of the lungs by indications of a like affection of some other organ. Thus the case may at any period of its course become complicated with symptoms of tubercular *meningitis* or *peritonitis*. Occasionally a tubercular *arthritis* may perhaps aid in clearing up the nature of the disease.* Cornil and Ranvier have shown that in cases of acute tuberculosis miliary granulations may often be found in the cancellous tissue of the bones, especially in the vertebræ, the sternum, and the ribs; and possibly their presence may sometimes give rise to pains vaguely referred to different parts of the limbs and body.

From a clinical point of view, the most important disease to distinguish from acute tuberculosis is enteric fever. Both diseases occur in patients of the same age, both are attended with pyrexia, both run an acute course, and in both the lungs are constantly, the intestines and peritoneum sometimes, the seat of disturbance. The temperature in tuberculosis is less regular, and interrupted by subnormal readings; the ratio of respiration to pulse and temperature is greater, and cyanosis comes on earlier. Moreover, some chronic disease of the pulmonary apices is often to be discovered. The rash and characteristic stools are absent, but both may be so in enteric fever.

Next to enterica we must remember the pyrexia of pyæmia, and particularly of internal pyæmia, such as from endocarditis or caries of the internal ear.

In the majority of cases of acute miliary tuberculosis of the lungs there are physical signs of previous phthisis, often of old standing or even appa-

mation. But since the discovery of the bacillus it is probable that the presence of this organism in the blood is the chief cause of the pyrexia; and, if so, the distinction from a specific fever can no longer be maintained.

* A striking example of this was recorded by Laveran in the 'Progrès Médical' for 1877. A man, aged twenty-two, was attacked with articular pains, especially in the knees. Effusion occurred into the right knee-joint, and when admitted into hospital he was supposed to be suffering from subacute rheumatism. However, at the end of a week great dyspnœa set in and high fever, the temperature ranging from 102° to 104°. A fortnight later he died, the cause of death being acute tuberculosis. The synovial membrane of the right knee was found to be injected and covered with a large number of greyish granulations the size of pins' heads, which could be felt. Where they had been in contact with a surface of articular cartilage their summits were flattened. A few granulations were present also in the left knee.

In 1867 a woman died in Guy's Hospital of tubercular meningitis, whose right knee had become swollen and painful in the course of her illness. At the autopsy all that was noted was that the synovial membrane was very vascular and œdematous, and that the cartilage over the external condyle of the femur was slightly eroded. But it is not at all unlikely that tubercles were present.

rently obsolete; or if these are masked by the more recent and acute malady, we have a history of cough, wasting, or hæmoptysis; or the clubbing of the fingers may point to long-standing deficient aëration of the blood. The prevalence of phthisis or other tubercular disease in the family is another point which often helps in deciding the diagnosis of an acute febrile attack.

Acute capillary bronchitis, when it occurs in a young adult, is sometimes most difficult to distinguish from miliary tuberculosis of the lungs; or, rather, it is difficult to decide whether capillary bronchitis is due to the presence of tubercles or is the only disease. In young adults the latter is improbable, but the writer had two cases during the winter of 1889–90, in a young man and a young woman, in both of whom there were no tubercles or other complication.

As a help to diagnosis, the most important seat of miliary tubercles is one where they can actually be seen during life.

In 1857 Manz discovered tubercles in the *choroid* of each eyeball in the body of a girl who had died of acute tuberculosis, and he and Busch afterwards recorded similar instances. In 1867–8 Cohnheim, investigating this point carefully in all the cases of miliary tuberculosis—eighteen in number—that came under his notice in the Pathological Institute at Berlin during a period of fourteen months, found that in every instance one or both of the eyes—almost invariably both of them—showed choroidal tubercles. In April, 1867, the ophthalmoscope was for the first time used, apparently by v. Graefe himself, for the discovery of these tubercles during life in a patient of Griesinger's. In November of the same year Mr Soelberg Wells exhibited to the Pathological Society of London a specimen of choroidal tuberculosis, which he had detected in a little girl under the care of Sir Alfred Garrod, five days before her death. Since then many other observers have recorded similar facts. But further experience seems to have shown that the eyeballs are far from being so constantly involved in cases of miliary tuberculosis as Cohnheim supposed.*

In acute cases the number and the size of the tubercles may increase from day to day, as was noticed in a child examined by Fränkel and by Leber. If, however, nothing should at first be detected by the ophthalmoscope in a suspected case of general miliary tuberculosis, the instrument ought to be used again and again as the disease advances. The tubercles vary much in size. The largest seen by Cohnheim was 2·5 mm. in diameter; but Ponfick met with one that measured 5 mm. On the other hand, Cohnheim seems often to have detected them (in the dead body) where they could only be seen after carefully removing the choroidal pigment, and even where they were too small to be visible by the naked eye. Perhaps this may account

* One of the most remarkable cases was recorded by Fränkel in 1872 in the 'Berliner klin. Wochenschrift.' A delicate girl of six was attacked in May of 1871 with slight shiverings, and her temperature rose occasionally to 100·4°. Then partial ptosis appeared, and afterwards paralysis of some of the ocular muscles. On May 22nd the ophthalmoscope showed a white patch to the upper and inner side of the disc in the left eye; it was as large as the disc itself, and had a rounded form, except that in one direction it was drawn out into a point. By the 1st of June it had increased in size by one half. On account of its characters being so different from those generally described as belonging to tubercles in the choroid, Fränkel hesitated to diagnose it as tubercular. The child now went into the country with her parents and remained there until August, when she came back apparently in perfect health. The patch in the fundus of the eyeball, however, was more prominent, though not larger than before. On August 21st she became ill with gastric symptoms and pyrexia, and died on October 1st. On September 10th five fresh miliary tubercles had been detected in the choroid. Vision remained unimpaired until death.

for the discrepancy between his statements and those of other observers as to their inconstancy in general tuberculosis. Where there are but few of them they seem to be developed in the neighbourhood of the disc or of the yellow spot more often than towards the equator of the eyeball. When very numerous, some of them may run together into irregular masses. They almost always become caseous as soon as they reach about 1 mm. in diameter. With regard to the ophthalmological diagnosis between tubercles and the white spots that are seen in disseminated choroiditis, von Graefe and Leber insisted on the rounded form generally presented by tubercles, their projecting above the level of the choroid, the gradual thinning away of the choroidal pigment from their periphery inwards to their centres, which appear white, and the absence of any accumulation of pigment outside them, except perhaps when they are very large. It is very rare for the sight to be affected.

Ætiology.—Pulmonary tuberculosis is seldom the result of caseating tubercular glands which had been recognisable during life. Many of those who are attacked, whether children or adults, are robust and healthy-looking. On the other hand, one must always be prepared for the supervention of miliary tuberculosis in cases of phthisis; for in the vast majority of cases acute miliary tuberculosis is secondary to chronic tubercular and caseous inflammation of the lungs or some other organ.

Acute tuberculosis occurs at all ages, but is most frequent in young adults. Burkart records the case of a woman, aged twenty-eight, who died after a fortnight's illness, and in whom the lungs, the peritoneum, and the kidneys showed recent miliary tubercles, while in the intestine there were the typical lesions of enteric fever. He also cites eight cases recorded by Birch-Hirschfeld, in which acute tuberculosis developed itself after enteric fever. In children the disease often follows measles, scarlet fever, and smallpox.

Among forty cases observed at Guy's Hospital between the years 1857 and 1873, one was in an infant aged nine months, three were in patients between one and ten years, seven between eleven and twenty, thirteen between twenty-one and thirty, six between thirty-one and forty, four between forty-one and fifty, six between fifty-one and sixty. This list does not include the cases in which the clinical symptoms were those of tubercular meningitis; if they were taken in, the proportion of children would be far higher. Burkart gives very similar figures. Among the above cases there were almost exactly twice as many males as females; among Burkart's the proportion was as sixteen to two.

Prognosis.—The duration of the disease is commonly three to four weeks, reckoning from the first commencement of marked symptoms up to the time of the patient's death; but there may be a protracted illness, lasting three or four, or even eight months. Clinically, acute pulmonary tuberculosis resembles severe bronchitis more than any other affection, and is not seldom mistaken for it until an autopsy reveals the real nature of the case. Indeed, as Burkart has remarked, it is common for miliary tuberculosis to develop itself in lungs which are already emphysematous from old bronchitis. Twelve of his eighteen cases were examples of this, and their course was often very prolonged. In no fewer than six of these, in fact, the tubercles were already calcified or fibrous, with a lustre like that of mother-of-pearl, so that they might fairly be considered obsolete; and the cause of death was sometimes pleural effusion, sometimes dilatation of the right side of the heart, sometimes Bright's disease; the presence of the

tubercles seemed to be little more than an accident. A similar obsolescence of pulmonary tubercles has been observed by the author.

This question of the possible involution of miliary tubercles in the lungs is of great importance. It has been usual to regard the fact that a patient recovers as conclusive proof that he was not suffering from miliary tuberculosis; although Dr Bristowe says that the progress of the disease "may be occasionally arrested, but with more or less permanent damage to the tissue of the lung." But, in cases of fatal tubercular meningitis, we often have opportunities of observing that the number of miliary tubercles scattered through the lungs may be very small. And it is difficult to say why, if the meningitis were absent, recovery should not take place.

Wunderlich, as far back as 1860, recorded in the 'Archiv für Heilkunde' cases which he regarded as examples of cured miliary tuberculosis; but he based his diagnosis solely on the fact that the temperature chart failed to correspond with what he regarded as a necessary course of enteric fever. Few observers will be found at the present time to endorse his opinions in this respect. Perhaps the only way in which the occurrence of recovery from an attack of miliary tuberculosis could be proved by clinical evidence alone would be by the discovery of tubercles in the choroid of the eye. Or the patient, after getting well, might die from some other cause, and the tubercles be found in a state of obsolescence. In regard to this, Dr Bristowe speaks of the lung, after the arrest of the discrete tubercles, as becoming "seamed throughout with minute patches of cicatricial tissue, the fibres of which have something of a stellate arrangement, and within the limits of which the lung-tissue presents, from the presence of concurrent emphysema, a coarsely spongy character; occasionally, in the centres of the scars, minute fibroid knots or concretions may be recognised."

With regard to the *treatment* of miliary tuberculosis of the lungs, all that can be said is that the strength of the patient should be maintained, that symptoms should be checked by appropriate drugs, and that perhaps bichloride of mercury or some other parasiticide should be given in the hope of favouring the obsolescence of the tubercles.

CATARRHAL DISORDERS OF THE AIR-PASSAGES, WHOOPING-COUGH AND ASTHMA

"Præcipue sanus, nisi cum *pituita* molesta est."—HORACE.

"What evil star
On you hath frown'd and poured his *Influence* bad?"—SPENSER.

"Anthony Henley's father, dying of an *Asthma*, said, 'Well, if I can get this breath once out, I'll take care it shall never get in again.'"—SWIFT.

CORYZA *or Nasal Catarrh—its symptoms, causes and treatment—Feverish colds.*
INFLUENZA *or Epidemic Catarrh—history—symptoms—events—ætiology and pathology—diagnosis—prognosis and treatment—comparison of* 1762 *and* 1890.
Ozœna or fœtid coryza—origin in atrophy of the nasal tissues—minor forms—treatment. Epistaxis—ætiology—course—treatment.
HAY-FEVER *or Summer Catarrh—onset and course—ætiology—distribution among persons and places—treatment.*
WHOOPING-COUGH—*History and names—Symptoms and course—Events—Complications and sequelæ—Prognosis—Pathology—Treatment.*
ASTHMA—*The term—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Prognosis and treatment.*

IN the present chapter will be described certain affections of the upper air-passages: catarrh and other affections of the nasal mucous membrane; and also whooping-cough and asthma, which are clinically related to bronchitis and dyspnœa, although pathologically the one might find a place among specific fevers, and the other among spasmodic disorders.

NASAL CATARRH.*—Almost everyone, at least in England, is liable from time to time to a "cold in the head." This affection commonly begins with sneezing, repeated again and again. The nose becomes dry and "stuffy," its mucous membrane may be seen to be swollen and injected, and there is difficulty in breathing through it, so that the mouth is kept open for breathing; and the nasal consonants *n*, *m*, and *ng* cannot be pronounced, but become *d*, *b*, and *g* respectively. Then follows a profuse flow of a thin, saltish, watery fluid from the nostrils, so that the handkerchief is continually in use. Smell and taste are completely abolished for the time. If the frontal sinuses are involved, there is dull pain in the forehead, or severe headache. Extension to the lachrymal passages causes the tears to flow over the cheeks, and the conjunctiva becomes injected. Extension from the fauces to the Eustachian tube may give rise to deafness. The skin near the nostrils often becomes inflamed and excoriated by the discharge.

* *Syn.*—Coryza, *κόρυζα*—Pituita capitis vel nasi—Gravedo—Catarrhus endemicus, Catarrhus a frigore.—*Anglice*, Cold in the head.—*Fr.* Rhume de cerveau.—*Germ.* Schnupfen.

In this fluid leucocytes appear to be always present, though only in small numbers; and, according to Hüter, micrococci are also abundant.

Sometimes an attack of nasal catarrh passes off as suddenly as it began. In two or three hours the flow may cease entirely; or the patient having been tormented with a "running cold" up to the very moment of going to bed, may forthwith fall asleep, and on waking in the morning may find himself entirely free. According to Trousseau, such a transitory coryza is sometimes an irregular manifestation of asthma, either leading directly to a genuine attack of that disease, or at least occurring in individuals subject to it. In cases of this kind there is little or no constitutional disturbance.

The more common kinds of nasal catarrh often set in with marked malaise and chilliness, and in children they may be attended with considerable pyrexia. The usual duration of such an affection is from two days to a week, but sometimes it lasts longer, or (it would perhaps be more correct to say) a series of attacks occur in succession, a fresh one setting in each time as the other is passing off. Whenever coryza runs on for more than a day or two, the discharge from the nose alters in character, becoming thick, opaque, and muco-purulent. Sometimes, as the nasal affection subsides, the larynx and the trachea are in their turn attacked with a like catarrhal inflammation. Lastly, a severe cold is in certain cases followed by a long-continued impairment or loss of the sense of smell.

In regard to the *causes* of coryza, it is in the first place to be noted that some individuals are far more liable to it than others, and that it is also especially apt to occur in certain families. There are persons who are sure to take cold if they get their feet wet; but such an over-susceptibility to catarrh may often be controlled by cold sponging in the morning, or by the use of a shower-bath, and by keeping the bedroom supplied with fresh air during the night. Among the public it is almost universally believed that nasal catarrhs are contagious; they are supposed to be easily conveyed from one person to another by handkerchiefs, or by the act of kissing.* It is uncertain whether the great prevalence of coryza at certain seasons of the year is to be regarded as a real epidemic, or as a result of the wide-spread distribution of the common exciting causes of the affection. As a matter of common observation, feverish colds "run through the house," but this generally occurs at times when they are prevalent.

A special variety of nasal catarrh must be mentioned which arises in certain individuals when they are taking iodide of potassium medicinally; this is supposed by Fränkel to be due to direct irritation of the lining of the nose by iodide dissolved in the mucous secretion.

It must not be forgotten that coryza is a prominent early symptom of measles, and sometimes of typhus.

An inflammation of the nasal mucous membrane that may be mistaken for an ordinary catarrh is sometimes dependent upon an infection of th nose with leucorrhœal or gonorrhœal discharge. In such cases, however, the exudation is from the first yellow and purulent, or mixed with blood, and the duration of the affection is much more protracted, lasting several weeks. Dr Hermann Weber, in vol. xliii of the 'Med.-Chir. Transactions,' seems to have first drawn attention to the possibility of the nasal cavities of an infant becoming infected during birth when the mother has leucorrhœa. Fränkel thinks that such an occurrence is by no means infrequent. In the

* Friedreich is said to have inoculated his nasal mucous membrane with discharges from persons suffering from coryza without any result.

'Lancet' for 1857 Mr A. M. Edwards relates a case of nasal gonorrhœa in a woman, who was ultimately shown to have caught it by using a handkerchief that had been employed as a suspensory bandage by her son.

Treatment.—Coryza is not a disease about the treatment of which one is often consulted, for most people manage their attacks themselves. It is well to keep indoors, and better to keep in bed, but few patients can do either. At the commencement ten grains of Dover's powder may be taken at bedtime with a basin of hot gruel, and next morning five grains of quinine. A warm bath is also useful; free perspiration seems to favour subsidence of the affection. Locally, marked relief often follows the insufflation of bismuth mixed with a little morphia, as advised by Dr Ferrier ('Lancet,' 1876, vol. i, p. 523). The late Dr. Williams recommended abstinence from all liquids. If there is no fever and the patient is allowed lozenges or orange-peel to suck, or a few teaspoonfuls of tea at intervals, the restriction is tolerable and prevents the profuse coryza. It applies best to those who are obliged to go about their work.

When nasal catarrh, instead of subsiding, persists for weeks together, change of air is often the best remedy; but the internal administration of arsenic is sometimes very useful under such circumstances, and striking results have been obtained from the same medicine when loss of taste and smell as the result of coryza had continued for many months; sometimes, however, it entirely fails.

Feverish colds.—Sporadic influenza.—The usually trivial disorder just described has considerable pathological interest. It is generally assumed to be the result of a chill, and few who are subject to it but can recall instances in which it follows directly and unmistakably as the result of exposure to a cold wind, or to rain, or to a draught of air. The first effect is a sudden feeling of chilliness, an involuntary shudder (*i. e.* a slight rigor), and a fit of sneezing. The reflex character of this spasmodic neurosis is shown by its being caused by a bright light irritating the eyes, as well as by cold air in the face or snuff in the nostrils, and also by its being prevented (inhibited) by pressure on the upper lip or bridge of the nose, and completely stopped by covering the face.*

But the same question arises which met us in the case of pneumonia. What seems a local inflammation determined by a chill to the surface, like bronchitis or pleurisy, assumes from another point of view the aspect of a specific fever, like measles. It sometimes comes on with no unusual exposure to cold; it "runs through the house" like a contagious disease; it is accompanied by malaise and pyrexia; it lasts for a limited time, and then subsides.

In one point it differs from other specific fevers, in that it does not protect against itself; or rather, perhaps, it protects for only a short time, for while relapses from an ordinary cold in the head are very frequent, a severe feverish cold rarely occurs except at considerable intervals.

The connecting link between the ordinary local coryza, described in the previous section, and the epidemic influenza, to be next considered, is furnished by what is somewhat loosely named a "feverish cold."

This, like the less severe and more local form, is usually the result of exposure to cold—particularly to an east wind blowing on the face; but it

* The action of the domestic remedy of smearing the nose with tallow or cold cream is to prevent sneezing by protecting the skin from cold.

is sometimes undoubtedly caught from another case of the same kind, and sometimes it is idiopathic, *i. e.* of unknown origin.

The symptoms begin with shivering or rigor, then there is usually pain in the throat felt on swallowing, together with headache and severe aching pains in the loins, the thighs, or the back and limbs generally. To this stage succeeds coryza and a short, dry, painful cough. The temperature rises, sometimes to 103° in an adult and often to 102°, the urine becomes febrile, the pulse quick, and the skin dry. After a day of distress, the skin begins to act, and the symptoms are somewhat relieved, but the coryza becomes more severe, a little mucus is expectorated, the bowels are constipated, and headache and pains in the limbs continue. After three, or at the latest four days, the feverish stage is almost always over, but there remains great prostration of strength for so short an illness, and considerable bronchial as well as nasal catarrh. Finally, there remains some cough and expectoration for a week or ten days more. Occasionally a sharp attack of diarrhœa seems to take the place of the nasal catarrh.

The *treatment* is first to avoid fresh chills, and secondly to keep warm. A hot bath or a Turkish bath is excellent, and for the time the patient feels well, but they do not cure; within an hour he is as ill as ever. He must go to bed, drink warm diluents, and get the skin to act. Dover's powder is certainly useful with most people. Some find antimonial wine more effectual in producing perspiration and relieving the pains and oppression. Low diet, salines, and laxatives are indicated, with constant warmth to the feet, the face, and the hands. Thus the patient gets comfortably through his malady. During convalescence quinine is the best tonic.

As prophylactics, covering the face and hands, or wearing cotton wool in the ears and nostrils, are the most effectual.

INFLUENZA.*—There is yet another form of catarrh of the nose, throat, and air-passages which is an epidemic, and probably a specific disease. This is the *influenza*, a name of Italian origin which came into use in 1741, and denoted its external origin and wide-spread sway. Its history is supposed to date from remote antiquity; but, as might be expected, doubts exist as to the real nature of many of the older epidemics.

There was an epidemic catarrh recorded in 1173, four similar epidemics in the fourteenth, and four more in the fifteenth century. According to Dr Parkes (in his article in 'Reynolds' System'), there were eleven in the sixteenth century, sixteen in the seventeenth, and eighteen in the eighteenth. Between 1800 and 1850 there were ten, of which three were the most important: one, in its spread over different countries, occupied the years 1830 to 1833; another occurred in 1837, when "half London" was attacked; and the third in 1847–8. This last was a very severe form. Many died from pleurisy, bronchitis, pneumonia, or pericarditis. At one time it was said that the whole staff of Guy's Hospital was laid up. It seems to be doubtful whether there was any reappearance of the disease, at least as an epidemic, during the second half of this century until the year 1889.

After having died out of the memory of the present generation, a severe and wide-spread epidemic of influenza has spread over the whole of Europe

* *Synonyms.*—Tussis epidemica (Sydenham)—Catarrhus a contagio (Cullen)—Catarrhus epidemicus—"the Chinese catarrhal fever"—"the Russian influenza."—*Fr.* La grippe.—*Germ.* Grippe.—*Ital.* Influenza. The word *grippe* is said to be derived from the Polish *crypka* or *grypka* (=*raucedo*).

and some other parts of the world while this edition is passing through the press, and is only now subsiding after several months.

The newspapers of November 26th, 1889, announced "a curious epidemic raging in St Petersburg; some authorities identify it with the dengue fever prevalent in Greece and Turkey (p. 346). The Russian doctors call it 'influenza,' the symptoms being fever and headache, accompanied by a running cold." Regiments were incapacitated and railways interrupted by the suddenness and severity of the outbreak. The disease spread quickly through Poland to Hungary and Vienna, where there was a considerable mortality. At the k. k. allg. Krankenhaus between December 7th and 14th, 165 patients, 57 nurses and 77 physicians were attacked. Other parts of Germany were visited, though less severely, as well as Italy, Spain, and France; afterwards the United States, Australia, and New Zealand. At the end of the year several cases had been reported at and near Chiswick, but the malady was slow in establishing itself, and some of the earlier cases reported were probably not genuine. In January and February, 1890, it was frequent in London, and our hospital wards and out-patient rooms were crowded with cases of catarrh and influenza. In the Foundling Hospital in January, 95 out of 315 children were attacked; and at the London Orphan Asylum 115 out of 500. At Hanwell, of 174 attendants 93 suffered, and of 1141 inmates of all ages 178. The disease after appearing in London visited the great provincial towns successively, and afterwards smaller places like Colchester and Cambridge, where at one college, St. John's, there were 70 cases. The severity of the epidemic was over in April, but scattered cases occurred until the end of May.

Course.—Most of the symptoms of influenza are those of a common feverish cold; but it is attended with greater pyrexia, and with more severe depression of strength. The patient perhaps suddenly becomes chilly and shivers, and for some hours he may feel exceedingly ill before any definite local affection appears. Early vomiting is not uncommon. When Sir Thomas Watson was first called to two cases on April 3rd, 1833, the symptoms were just those which mark the commencement of an attack of fever, and he did not then know what was about to happen; but by the close of the following day all London was smitten with the disease.

The pyrexia runs high, and there is intense pain "behind the eyes." The pulse is rapid, remarkably weak and small, and sometimes intermittent. After an interval the patient usually begins to sneeze, and a thin acrid fluid runs from his nose; his eyes grow red and watery, and his fauces are injected. In some cases, however, there is no coryza throughout. He becomes rapidly prostrate, so that he cannot keep up, and is obliged to take to his bed. He may suffer from giddiness or faintness; in some epidemics drowsiness is a common symptom, in others many patients become delirious. Pains in the limbs or cramps in the calves may be complained of. With the headache there is often a severe pain at the root of the nose, which is attributed to extension of the catarrh to the frontal sinuses. At the same time, or a little later, the patient is attacked with an irritating cough, dry, or attended with scanty expectoration of mucus without froth. Sometimes there is great dyspnœa, with a sensation of distress in the præcordial region, and frequent suffocative attacks. Loss of appetite is a marked symptom; the tongue is thickly furred; there may be nausea or vomiting; sometimes diarrhœa is present; the skin may have an icteric tinge. The urine is scanty and high-

coloured. In pregnant women abortion may occur, as the result (it is said) of the violent cough; or, if the menstrual functions have been suppressed from other causes, they are sometimes re-established. Facial herpes is sometimes observed.

At the end of from three to five days the attack passes off. If it lasts much longer either pneumonia has developed itself, or the catarrh of the air-passages has passed into ordinary bronchitis. The subsidence is sometimes gradual, sometimes by a critical sweating or diarrhœa; sometimes it seems to set in with epistaxis. The patient's convalescence is always slow, and he is long in regaining his strength. The subsequent prostration is remarkably severe and long continued. Relapses are not infrequent; few persons suffer more than one attack in the same epidemic. On the other hand, to have had influenza appears to confer no immunity against its recurrence on a subsequent occasion.

In some cases the disease assumes a rudimentary form, and the patient merely complains of slight coryza accompanied with sore throat and cough, and attended by a little malaise, headache, and disinclination for work—in fact, a feverish cold.

Sir George Baker, in his 'Opuscula Medica' (second edition, London, 1871), gives the following account of the epidemic of influenza in the year 1762. It was characterised by alternate heats and chills, by a constant cough, sometimes dry, occasionally accompanied by a little thin mucous expectoration. There was depression of strength, a sense of weight, and severe pain in the forehead and temples; inflamed, swollen, and watery eyes with photophobia; frequent sneezing and altered voice. There was painful rawness felt in the windpipe and chest, with, in some cases, a feeling of choking, and wandering pains in the arms, legs, and sides. The fever was chiefly nocturnal, but even then so slight that it rarely interfered either with sleep or with food. There was more or less perspiration, and when it was profuse the disorder was relieved or cured. The tongue was white and thickly furred; the urine was dark, and threw down a furfuraceous or lateritious deposit. In all cases there was more depression of spirits and loss of strength than the character of the disease seemed to account for, and convalescence was often tedious and imperfect. Many cases of abortion or premature birth were observed in London from this disease. In a few of the more severe cases there was a miliary eruption.

In the recent epidemic these symptoms were again observed. So far as the writer could judge from a limited number of cases, the attack usually began abruptly; the temperature rose higher on the first day than is common in any disease except scarlatina; the headache, lumbago, and pains in the limbs were unusually severe, and, with the not infrequent vomiting, resembled the onset of variola. The sore throat, coryza, and subsequent bronchitis were not different from those of a "severe cold." The bowels were usually confined, but sometimes there was sharp diarrhœa. A scarlatiniform rash was not infrequently observed. The urine was febrile, but very seldom contained albumen. The pulse was extremely rapid, and usually full and with rather high tension—rarely dicrotic. The course of the fever, as a rule, was not above three days, and sometimes less. The fall of temperature was very rapid, more so than in pneumonia. In one case, a strong, healthy young man was in full health on Saturday, had a temperature of 104·5° F. on Sunday, and by Tuesday afternoon was free from fever and from pain.

In some cases great prostration followed, and in many there was muscular weakness with malaise for several weeks after recovery.

Bronchitis was a frequent sequela, but the most important one was acute lobar pneumonia. In January, 1890, the writer saw in consultation with Dr Addison, of Colchester, a young man of twenty-seven ill with acute pneumonia of the left lung after influenza. He made a good recovery; but a brother three years younger had died a week before of the same disease, double lobar pneumonia after influenza; and a maid who had nursed him also died with acute double pneumonia. His mother, a younger brother, and a man-servant suffered at the same time from influenza, and the servant from pneumonia, but these three patients recovered.

Recovery from influenza has sometimes been followed by parotitis; often it appears to be the starting-point of phthisis.

Mortality.—It seems paradoxical to say that influenza is seldom fatal, and yet that it always causes a great increase of the death-rate; but the explanation is that almost all the mortality is brought about indirectly, and that the number of those who fall ill is greater, beyond all comparison, than in the case of any other epidemic disease. In London in 1847 not less than 5000 persons are said to have died of influenza in six weeks; but then it was computed that 250,000 persons were attacked. In Paris above one fourth of the population suffered; in Geneva not less than one third. Those who die are chiefly old and debilitated subjects, who have previously laboured under emphysema of the lungs, or who have feeble and dilated hearts. It is sometimes dangerous to very young children.* Parkes states that patients with lesions of the valves of the heart, and some at least of those who have phthisis, pass through influenza without being the worse for it; but other writers have remarked that after its subsidence phthisis often takes a very rapid course. Dr Farr pointed out, in 1847, that the mortality was much greater in those districts of England in which the death-rate was generally high than it was in healthier places. In February, 1762, the weekly death-rate at Warsaw ran from 30 or 40 to 150. In 1837 the mortality in 2347 cases was 2·3 per cent., in 1847 about 3 per cent. (Peacock). In 1889–90 it was probably lower than this throughout England, but in Vienna and for a time in Paris there was a higher death-rate, owing chiefly to subsequent pneumonia.

No special morbid changes are seen in the bodies of those who have died during an attack of influenza. The lungs and air-passages are congested, sometimes to an extreme degree, and there may be great œdema of the pulmonary tissue. Pneumonia is not infrequent, but this is regarded as a complication; it may be either catarrhal or fibrinous. Plastic exudation into the bronchial tubes has now and then been found.

Ætiology and pathology.—There have been many speculations as to the cause of influenza. No other disease diffuses itself so widely over the earth's surface. Not only is it capable of existing in all inhabited regions, so far as is known, but in some epidemics it has ranged over every quarter of the globe, and has established itself in places presenting all kinds of soil and every variety of climate. It therefore cannot be attributed

* But Sir George Baker writes of the epidemic in 1762, "*Leviter plectebantur infantes et liberabantur facillime.*" He found it most serious in advanced age, and particularly in asthmatic old men. The elder Dr Babington died of the influenza in 1833. Graves found the epidemic of 1837 very fatal among the aged, yet saw Judge Day recover at ninety-three.

to any "telluric emanation" or miasm; nor is it related to ague, for Holland, which is infected with malaria, has escaped some European epidemics.

Again, the progress of influenza from district to district occupies time. Many observers have thought that it commonly keeps to a definite direction, namely, from the east or north-east towards the west or south-west.

The epidemic of 1762 appeared at Warsaw in February, reached Vienna at the end of March, and Magdeburg in April; at which time it also invaded Hamburg and London. It was much more severe at Venice and at Warsaw than in England and Germany, and did not visit Paris at all. In June it was epidemic throughout Alsace,* and in July attacked the British fleet in the Mediterranean. After that month no cases were known to occur in Europe. Beside London, the towns of Manchester, Lincoln, Leicester, Exeter, and Norwich were seats of the disease, apparently by conveyance from the capital. Similar epidemics of catarrh or influenza had appeared at Norwich in 1733 and 1743. It was less common in country places—an argument in favour of its being infectious.

The epidemic which raged in London in 1833 is supposed by Hirsch to have been related to one which occurred in 1830 in China, and which reached Moscow later on in that year. In 1831 it spread over Russia, Poland, Germany, France, Sweden, Italy; it next appeared in the Isle of Man, and lastly in New Jersey, on the other side of the Atlantic. In 1832 it occurred chiefly in Spain and in some of the United States. In 1833 it broke out again in the north of Europe, and after extending over Russia and Germany, and passing to Denmark, it reached London in April. It was also observed at different dates of this year in Switzerland, the Tyrol, France, Italy, and Egypt.

The recent epidemic of 1889–90 was first observed in Russia and spread westward over the continent, attacking as usual the large towns earliest and most severely. It spread from Central Europe southwards over Italy and Spain, and northwards to England, where the smaller country towns were visited long after London, while many out-of-the-way places escaped altogether. In America and Australia the epidemic appeared later, and travelled over the United States in a westerly direction from the Atlantic seaboard to California.

It is particularly worthy of notice that both in England and France epidemic influenza seems only to occur as the result of extension from other countries. Some writers doubt whether it can arise in any part of Europe, and believe that it has its home in some remote part of Asia, perhaps Chinese Tartary. Hence the Russian name of the "Chinese catarrh," and the English name of the "Russian influenza."

Influenza often suddenly breaks out at the same time in places far distant from one another, and at once attacks a large proportion of their inhabitants; and it has been said to appear on exactly corresponding dates on board ships which had been long at sea, and which had sailed from ports where influenza was not prevailing. Thus in 1782, Admiral Kempenfeldt's squadron sailed from Spithead on May 2nd to cruise between Brest and the Lizard. On the 29th, there having been no communication with the shore, the men who formed the crew of one of the ships were attacked with influenza, and soon afterwards so many of the sailors on the other ships that by the second week in June the whole squadron had to

* These accounts were received by Sir Geo. Baker from Drs Jackwitz, Mertens, Kothen, Pringle, and Reimarus.

return to port. In the meantime another fleet, under Lord Howe, had sailed, all in perfect health, for the Dutch coast. Towards the end of the month of May the disease appeared in several of his vessels also, although there had been no intercourse with the land. So, again, on April 3rd, 1833, the day on which Sir Thomas Watson saw his first two cases of influenza in London, a vessel called the "Stag" was coming up the Channel and arrived at two o'clock off Berry Head on the Devonshire coast, all on board being well. The breeze was blowing from the land, and in half an hour forty men were down with influenza; by six o'clock the number was increased to sixty, and by two o'clock on the following day to 120. The very same evening a regiment at Portsmouth was in a perfectly healthy state, but by the next morning so many of the soldiers were affected by the disease that the garrison duty could not be performed. Parkes, indeed, expresses some doubts as to whether these instances can be entirely relied upon. But, as he says, if they are not altogether without foundation, they effectually disprove all chemical theories as to the cause of influenza; for neither a vapour nor any particles wafted in the air could travel such distances without undergoing dispersion and destruction. Nor is it possible that the materies morbi can be any substance, such as ozone, which, naturally present in the air in small quantities, might conceivably become enormously increased in amount over a wide area. For the disease, instead of affecting the whole of a town or city, sometimes confines itself to certain districts, or even to particular streets; and it may leave adjacent villages free.

Thus, by a process of exclusion, we are brought to the conviction that, unless the cause of influenza is something of the nature of which we have no conception, it must be a living thing, which is capable of reproducing and multiplying itself when once it has been introduced into a particular district or country. Nevertheless, though the discovery of the microbe of influenza was repeatedly announced in 1890, it is still unknown.

There have been numerous instances in which the complaint has first broken out in those particular houses of a town at which travellers had recently arrived from infected places; and there have also been examples of its having spared the inmates of prisons or convents, as though their isolation had served to protect them. But all that such cases can prove is that the contagion is capable of adhering to the human body, or to clothes or luggage, so as to be conveyed from one place to another. Cullen, indeed, defined influenza as *catarrhus a contagio*, but if he meant that it is contagious in the sense in which we now use that term, it is tolerably certain that such a view is incorrect. The disease seems to occur as frequently among persons who are confined indoors as among those who go about; it often attacks bedridden people; it does not spread from one patient to another, nor to relatives or nurses of the sick. It is decidedly more common in women than in men; children suffer less than adults, and in some epidemics they seem very generally to escape.*

* The absence of a stage of incubation is a remarkable exception from its general resemblance to the specific fevers. At the commencement of an epidemic of influenza an immense number of persons fall ill simultaneously or nearly so. If the disease took several days to hatch, one may be quite sure that the preliminary morbid process would not thus come to an end in every patient at the same time. Parkes, indeed, alludes to cases "in which the incubation period must have been two or three weeks," but on looking up the reference which he gives to Dr Robert Williams's work on morbid poisons, we find that the only instances given there are those already alluded to, in which the disease appeared on board ship when there had been no communication with the land.—C. H. F.

Epidemics of influenza have often been observed to last from four to six months, and their subsidence is scarcely less sudden than their commencement. In 1831 the disease prevailed in Paris for nine or ten months at a stretch. The recent epidemic lasted in England for about five months.

It is possible that the contagion may be derived from horses or other of the lower animals. Inoculation of horses with the blood of patients suffering under influenza was tried by Hertwig and failed. It is, however, not certain that the human disease is identical with the influenza to which horses are liable, and which, for example, raged in the United States in 1872, when it is said to have attacked about 16,000 horses in New York alone. During epidemics of human influenza, horses, dogs, cats, and even birds are said to suffer. But the disease in horses does not appear to spread to stablemen and grooms.

During the prevalence of an epidemic of influenza the only point to be mentioned in regard to its *diagnosis* is the risk of misinterpreting the early stages of other febrile complaints, such as enteric fever, rheumatism, or the exanthemata.

Sir Thomas Watson says that "in the years immediately succeeding an epidemic it generally shows itself again, but in a milder and less general form." And he goes on to say that "many of the colds and bronchial disorders of the seasons which follow a period of genuine influenza are attended with much more languor, debility, muscular aching, and distress than belong to an ordinary attack of catarrh." Parkes, however, states that sporadic cases are not met with; and we ought to protest against the practice of applying the term influenza to any severe catarrh, in order to add dignity to a common complaint.*

A further question remains as to the nature of certain localised forms of catarrh, affecting a greater or less part of the population of particular towns or districts. Thus in 1864 influenza is said to have existed in Switzerland, during the spring of 1867 in Paris, and so recently as 1874 in Cape Breton. As Parkes observes, strict proof that the disease in such "local epidemics" is really influenza ought to be required before they are admitted; but it is difficult to say how the proof is to be obtained.

With respect to *prognosis* we have already stated that an epidemic is only generally dangerous to infants and aged persons. But in anyone influenza is liable to end in ordinary acute lobar pneumonia, which as above stated is not infrequently fatal.

Treatment.—From an historical point of view it will always be interesting to know that bleeding and the administration of antimony were recognised by universal experience to be injurious in this disease at a time when they were regarded as almost essential to the cure of pneumonia and other inflammations. In the later epidemics quinine was found useful, even from the commencement of the attack; but the most usual practice was, after having given one purgative dose, to prescribe salines during the first day or two, and afterwards ammonia with senega or serpentary. Parkes insists that the custom of feeding the patient with hot beef-tea is a bad one; it invariably, he says, increases the headache and the languor. As there is complete loss of appetite, and as the attack may be expected to come to an end in a few days, it may be sufficient, if the patient is young and healthy, to let him

* Yet, as Niemeyer remarks, "diese Unsitte hat gewissermassen ihr Gutes. An einem Katarralfieber will Niemand erlieblich erkrankt sein und längere Zeit im Bette liegen; hat er aber die Grippe so ist er zufrieden wenn er nur in acht bis zehn Tagen genest."

have such beverages as iced-milk and soda-water, barley-water with lemon-juice, or weak white-wine whey. Except to elderly patients stimulants should not be given during the early stages. Afterwards, a good supply of food should be allowed, and at this stage tonics are often necessary. In the recent epidemic hot baths and diaphoretic drugs gave much relief, and antipyrin proved the most useful remedy for the severe headache.

OZÆNA.*—*Fœtid coryza.*—A very intractable and distressing form of chronic nasal catarrh is one in which the discharge from the nostrils, and even the air expired through the nose, has constantly a peculiar and disgusting fœtor.

With regard to many points about it there is still much uncertainty. Formerly it was thought to be generally dependent either upon "the scrofulous diathesis," or upon a syphilitic taint, congenital or acquired. There is, of course, no doubt that disease of bone in the interior of the nose, whether due to syphilis or to any other cause, may produce great fœtor. But the special odour that characterises ozæna exists without caries of the bones or ulceration of the mucous membrane.

All recent observers who have made autopsies in cases of ozæna appear to be agreed that in this affection there is an *atrophic* condition of the tissues within the nose, including even the turbinated bones. At one time it was supposed that this was the ultimate stage of a chronic "rhinitis," attended at an earlier period with thickening and swelling of the mucous and submucous structures. There is, however, no evidence of the occurrence of such an antecedent condition. Nor is it clear how the atrophy, when it has developed itself, is related to the fœtor. Some have thought that the current of air through the widened nasal passages is too slow to clear away the mucus by sneezing, and it therefore dries up and undergoes decomposition. Others hold that atrophy of the glands leads to an insufficient formation of mucus. In either case it appears probable that the process of desiccation is essential to the production of the offensive odour.

But there can be no doubt that mucus may and often does form dry crusts in the interior of the nose without any fœtor resulting. The explanation seems to be that for the production of ozæna a special ferment or microbe is required, the presence of which induces a particular kind of putrescence.

So penetrating is the smell in the worst cases of this distressing malady that it pervades the air for some distance round the patient, and renders him unfit for society. Yet he himself is often quite unconscious of it.

It must not be supposed, however, that the affection is always so severe as this. Fränkel remarks that about some persons, when they first wake in the morning, very faint indications of the ozæna-odour can be plainly recognised, which are altogether absent at other times. And, again, he says that in some cases in which mucus leaves the nose free from smell, it acquires more or less of the characteristic fœtor after drying on the handkerchief. This statement is confirmed by one's own observation. On the other hand, the contents of a spittoon or of a patient's spit-jar never smell like ozæna, perhaps because they are not allowed to dry up.

Treatment.—The usual method of treating ozæna has been by systematic

* *Synonyms.*—Chronic atrophic fœtid coryza—Rhinitis chronica (Fränkel)—Ὄζαινα (from ὄζω, I smell) was applied by Greek writers in the second century to a fœtid polypus in the nose. *Le punais, la punaisie,* are the vernacular names in French.

irrigation of the nose with water, or with saline solutions, or by injections of antiseptic agents, such as boracic acid or salicylate of soda. Of late a considerable advance seems to have been made by Gottstein, of Breslau, who simply introduces a plug of cotton wool into one nostril, leaving it there for twelve or twenty-four hours, and then withdrawing it, and plugging the other nostril in its turn for a like period. The effect is often successful so far as concerns the temporary removal of the fœtor. But almost as soon as the treatment is discontinued the case becomes as bad as ever. Ozæna is, in fact, at present incurable.

So serious is the discomfort entailed and so unsatisfactory have milder remedies proved, that more than once surgeons have undertaken the severe operation of extirpating the turbinated bones. (See on this subject a paper by Mr Warrington Haward in the 'Lancet' for 1877, vol. i, p. 784.)

EPISTAXIS.*—*Bleeding at the nose.*—Epistaxis occurs as a complication of many maladies. It is apt to accompany purpura and all hæmorrhagic diseases, and in splenic leuchæmia it is often an early symptom. It is more frequent in aortic than in mitral affections of the heart. Persons who have granular kidneys with hypertrophy of the heart are exceedingly liable to it, and in them it may often be taken as a warning of the probable supervention of uræmic seizures or of cerebral hæmorrhage. In old men it denotes vascular degeneration, and it is also frequent in cases of cirrhosis of the liver, with more or less jaundice and with dilatation of the small veins of the cheeks. "Bleeding at the nose," wrote the elder Heberden, "is a usual attendant upon the diseases of the liver in hard drinkers." It may accompany some of the acute infective diseases, especially variola and enteric fever. In cases of ague not only is it sometimes associated with the ordinary paroxysms of the disease, but it is said to have occurred periodically as the sole symptom and effect of malarial poisoning, until stopped by the administration of quinine.

But in the majority of cases bleeding at the nose is due to none of these causes. Boys towards the age of puberty are very subject to it spontaneously from time to time; and at any moment a slight blow upon the face, touching the nasal mucous membrane, or forcibly blowing the nose, may bring it on. In the 'Lancet' for 1865 Dr Guy Babington gave the history of a family in which marked liability to habitual and violent epistaxis was traced through five generations, and in thirteen out of twenty or more individuals. Some of those who suffer from recurrent or "habitual epistaxis are pallid, with apparently little blood to spare. But others are plethoric, with flushed cheeks and injected conjunctivæ; and they may experience from time to time sensations of oppression and giddiness, noises in the ears, throbbing in the head, fulness and heat in the nose, which they recognise as indications of the approach of an attack of hæmorrhage, and which are at once relieved when it occurs.

In some young women epistaxis has been observed to be distinctly vicarious of the catamenia. A striking instance of this is one recorded by Obermeier in vol. liv of 'Virchow's Archiv.' A girl of fifteen, after once menstruating in the ordinary way, began to suffer at regular intervals of a month from bleeding at the nose; this occurred two or three times a day for three successive days, and it was attended with malaise and other symptoms like those which had accompanied the natural flux. She became

* Ἐπίσταξις sc. αἵματος, a continual dropping of blood.

pregnant and the hæmorrhage then ceased, to return six weeks after her delivery. No doubt caution is required in accepting the statements of patients as to any form of vicarious menstruation; but this case appears to be beyond question.

In epistaxis the blood almost always comes from one side of the nose only; but sometimes part of it passes round behind the nasal septum and ultimately escapes from the opposite nostril, so that both sides may appear to be bleeding simultaneously. It may either flow in drops or in a more or less continuous stream; the quantity lost is in some cases very great. It is commonly of a bright red colour, this being perhaps due to exposure to the air after it has escaped from the vessels. It forms a solid coagulum. One can very seldom discover, whether by rhinoscopy or by inspection through the nostril, the exact point from which the oozing of blood takes place; nor have pathological anatomists as yet ascertained whether in cases of habitual epistaxis the veins in the submucous tissue of the nose (which are normally very wide and numerous) are in a varicose condition or affected with any degenerative change. It is, however, certain that not infrequently repeated epistaxis in adults is due to an ulcer of the septum, and in many cases to a perforating ulcer. This may depend on syphilis, but probably has more often a traumatic or accidental origin.

Sometimes part of the blood escapes through the posterior nares into the pharynx and is swallowed. Indeed, if the patient should happen to be asleep or recumbent from any cause, the whole of it may take this course; and when a large quantity of blood is subsequently ejected from the stomach by vomiting, the real seat of the hæmorrhage may be altogether overlooked unless a careful examination of the nose is made, when clots will be discovered in one or the other of the nostrils or in the pharynx.

The ordinary course of epistaxis is to cease spontaneously sooner or later. But it sometimes continnes for many hours, or even for days, without intermission. In such cases the patient may rapidly pass into a condition of extreme anæmia, and may possibly die of syncope; and in other cases the frequent repetition of attacks of bleeding at the nose brings about a chronic state of bloodlessness that itself tends to favour the occurrence of further hæmorrhage. The process by which the natural arrest of epistaxis is effected appears to be by the formation of coagula, which adhere firmly to the mucous membrane and close up the vessels. Hence, after the cessation of an attack, any disturbance of the parts may at once cause a return of bleeding. Fränkel, in 'Ziemssen's Handbuch,' remarks that obstruction of the free part of the nose by clots, with apparent cessation of the epistaxis, affords no guarantee that oozing into the pharynx may not still continue, the patient swallowing the blood without knowing it; in the case of enteric fever, for example, great danger may arise in this way.

Epistaxis should not always be actively treated; as already observed, it may give relief to other symptoms of which the patient had been complaining, and it then generally ceases of its own accord. On the other hand, when anæmia is beginning to result, one must be careful not to delay too long in carrying out whatever measures may be requisite. Sometimes compression of the side of the nose against the septum by the finger placed just below the nasal bone is sufficient to arrest the flow of blood, at least for the time. It is important to notice whether this is the case; for, if so, one can be sure that the bleeding spot is in the forepart of the nasal cavity, and that one can stop all further hæmorrhage by the "anterior tam-

ponnade," that is by systematic plugging with a long strip of lint introduced through the nostril. But before adopting this method of treatment it is as well to make trial of astringents, such as gallic or tannic acid, which may be sniffed up in the form of a powder. To inject cold water or a solution of alum or any other astringent liquid is less advisable, because it tends to disturb any clots that may have formed. Cold may be applied to the outside of the nose, as well as to the whole of the patient's neck and chest. At the same time he may have his feet placed in a hot mustard bath. He should sit upright with his head slightly bent forward, so as to prevent the blood passing backwards towards the pharynx, and should keep the hands raised above the head. The most efficient internal medicine is ergot or subcutaneous injection of ergotin.

But if the hæmorrhage should continue, and if the source of it should appear to be from the back part of the nasal cavity, one must not wait long before having recourse to the radical method of the "posterior tamponnade," or "plugging the posterior nares." This is generally effected by means of an instrument which is described in every surgical work, and which is known as a "Belloc's cannula;" the plug being a piece of folded lint of suitable size and shape. It has, however, its disadvantages; for if the lint is not left undisturbed for some days, epistaxis is apt to begin again as soon as it is interfered with; while, on the other hand, if it be allowed to remain long *in situ*, there is often great difficulty in loosening it, and the mucous membrane may be torn. In any case it should certainly never be left to become fœtid by putrefaction of the blood and other fluids which soak into it. A better method is to fill up the nasal cavity with an elastic pouch, which, having been introduced in a collapsed state, is afterwards inflated with air until it exerts considerable pressure in every direction.

HAY-FEVER.*—Certain persons are liable every summer to a very troublesome affection, which sometimes assumes the characters of catarrh, sometimes those of asthma, and therefore may find its place provisionally between coryza, influenza, and spasmodic asthma. Attention was first directed to it by Dr Bostock in a paper read before the Royal Medical and Chirurgical Society in 1819.

Symptoms.—The *asthmatic* form will be considered separately (p. 1148). The *catarrhal* form sets in with a feeling of irritation in the nose, throat, and eyes. Then the patient begins to sneeze—perhaps twenty or thirty times in succession. A thin watery secretion pours from his nostrils. The nasal submucous tissue rapidly swells until in a little while no air can be drawn through the nose. If, however, he lies down and turns on his side, the nostril which is now uppermost becomes in a short time free (apparently as the result of gravitation of the œdematous fluid), while the other one becomes more occluded than before. The swelling affects the lachrymal passages also, so that the tears run down over the cheeks. The eyes become inflamed, and there may be œdema of the eyelids. So distressing are these symptoms that it is almost impossible for the patient to avoid giving way to them and suspending his usual occupations. From day to day they vary in severity, but they commonly last three or four weeks,

* *Synonyms.*—Hay-asthma—Periodic vaso-motor coryza—Specific or nervous coryza—Catarrhus æstivus—Catarrhus ex fœnisicio—Rose-cold and autumnal catarrh (U.S.A.).—*Fr.* Fièvre, asthme, ou catarrhe de foine, asthme d'été.—*Germ.* Heu-asthma, Heufieber, Bostock'sher Katarrh.

or even longer. Paroxysms of sneezing continue to recur from time to time. The nasal discharge presently becomes thicker and more purulent, or it may be stained with blood. Ultimately the affection passes off, leaving more or less weakness and prostration behind.

Exciting cause.—One of the first points observed about this "summer catarrh" was that its onset often corresponded closely with the beginning of the hay season, and that persons were attacked immediately after being in or close to a hayfield. But it was not until 1873 that Dr Blackley, of Manchester, discovered by careful observations and experiments upon himself that the true cause of the affection is the diffusion in the air of the pollen of certain plants, and especially of grasses, which settles upon the mucous membrane of the nose and eyes, and acts as a local irritant. Up to that time some medical writers had attributed hay-fever to coumarin,* others to ozone, others to "common dust," and others to the heat of early summer. Dr Blackley found that by introducing a small quantity of pollen into the nostrils he could bring on all the symptoms of the disease almost simultaneously. During the summers of 1866 and 1867 he made daily observations upon the amount of pollen which was deposited upon glass slides moistened with glycerine and exposed to the air; and he found that there was in general a close relation between the quantities collected and the severity of the symptoms of hay-fever under which he laboured from day to day. Dr Blackley appears to have accounted satisfactorily for certain observations which had seemed to Dr Bostock and others to prove that the supposed connection between summer catarrh and the emanations from flowering plants was a mistake. At any rate, he showed that when one might imagine that there can be no pollen in the air, it really may be present in abundance. Once he was suddenly seized while on the shore, with a sea breeze blowing; between him and the sea there was but a narrow belt of land, but upon this he found a field of wheat in full bloom. Another time an attack was brought on by the dust of an unfrequented country lane; it was summer-time, and on examining the superficial layer of dust with the microscope he discovered that it was full of the pollen grains of grasses.

Predisposing causes.—Hay-fever most commonly appears for the first time about the age of puberty, but sometimes it is observed in children four or five years old. Persons who have reached the age of forty without being affected with it are probably never attacked afterwards. It is more common in males than in females.

One very curious circumstance about this disease is that those who suffer from it appear always to belong to the educated classes. It is never seen among gardeners or farm labourers, who are of course more exposed to the influence of pollen than any other set of men. This fact has led to the suggestion that the individual predisposition to the disease, which plays so important a part in its ætiology, is perhaps a result of an indoors life, especially in towns or cities. Should that be the case, one can understand how it is that hay-fever appears to have become so much more common of late years than in the earlier part of the present century. In addition to the personal peculiarity, which we may call an idiosyncrasy or neurotic disposition or diathesis, Sir Andrew Clark believes that there is a local peculiarity in the nasal mucous membrane of those subject to this curious malady, which leads it readily to swell and become vascular.

* Or coumaric anhydride ($C_9H_6O_2$), an aromatic compound belonging to the Benzene group. It gives its scent to the grass *Anthoxanthum odoratum*, and to *Asperula odorata*.

It is supposed to prevail in England much more than on the Continent, but it is also very common in the United States, where it has been carefully investigated by Dr Mackenzie, of Baltimore. In most cases the susceptibility to the disease increases with each successive year. At first the patient may be attacked only when he is actually in a meadow where the grass is in full bloom; ultimately he suffers as soon as he attempts to go into the country during the hay season. Sometimes hay-fever assumes the asthmatic form after it has for several years recurred as a catarrh.

The treatment of hay-fever is extremely unsatisfactory. Neither quinine nor arsenic, nor any other medicine appears to have the power of enabling those who are liable to the disease to bear exposure to its exciting cause without being at once attacked by it. The local application of quinine in solution was first used in his own case by Professor Helmholtz, but it has not proved so successful with meaner sufferers. Sir Andrew Clark (after trying and discarding every kind of internal remedy, including aconite and belladonna) finds that a solution of cucaine (5-15 per cent.) applied locally with a brush, or as a spray or a bougie, is sometimes successful, though it often fails. As a more effectual plan, the same physician recommends the thorough local application of a solution of carbolic acid to the interior of the nostril (see the formula and method described in the 'Brit. Med. Journ.,' June 11th, 1887, p. 1256).

The most thoroughgoing treatment of all is to destroy the secretive parts of the Schneiderian membrane by caustics or galvano-cautery. This severe treatment has been carried out in the United States, and it is said with complete and permanent success.

For those who suffer severely, the only course (apart from local treatment) is to remain in a large town through the whole of the summer months, or else to go to the sea-side, choosing some narrow peninsula or island, or to take a sea voyage. Staying indoors during the middle of the day, even in a country house, often mitigates the symptoms.

The continuance of hay-fever during several weeks—generally from some time in May until the middle of July—depends upon the continued exposure of the patient to the irritant. If he can get away to a place where there is no pollen in the air, the attack quickly passes off.

When the symptoms begin considerable relief is afforded by the use of a smelling-bottle containing ammonia, iodine, and carbolic acid, made into a paste with wood-charcoal and compound tincture of benzoin.

WHOOPING-COUGH.*—Like so many other epidemic diseases, whooping-cough can be traced to only a comparatively recent period—the earliest notice of it is said to have been by Schenck in the year 1600. It is a very frequent and wide-spread disease, and, next to scarlatina, more fatal than any other in childhood; indeed, for infants under one year it is probably the most fatal of all.

Clinical course.—The *incubation* of whooping-cough after infection is perhaps variable—probably a fortnight as a rule, but often less.

* *Synonyms.*—Tussis clangosa (Glisson)—Pertussis infantum (Sydenham, 'Proc. Integr.,' cap. xliv, 1695)—Tussis puerorum convulsiva (Sydenham, 'Obs. Med.,' iv, 5, § 8, 1685; also Heberden and Cullen).—*Fr.* Coqueluche, Toux-qui-houpe (whence "hooping" cough, rationalised into "whooping").—*Germ.* Keuchhusten.—*Vernacular.* Chin-cough, a corruption of Chink-cough.—*Scottice.* Kink-cough, or Kink-host (Dutch Kinkhoest).—Kink or Chink means a catch in the breath (Skeat).

The *prodromal stage*, as it is sometimes called, is characterised by an ordinary bronchial catarrh, with a more or less troublesome cough and some pyrexia. Sometimes there is also nasal catarrh, with running at the nose and sneezing. The child (for whooping-cough chiefly affects children) is pale, out of sorts, and restless. According to Trousseau, the cough is sometimes remarkably frequent, recurring fifteen, twenty, or thirty times in the minute; in such cases there is generally high fever. He speaks of having been able to diagnose the disease at this period by the incessant repetition of the cough. But, as a rule, there are at this stage no symptoms by which its nature can be suspected, unless other cases have recently occurred in the same family or neighbourhood.

The duration of the prodromal stage is very uncertain. Sometimes in young children it lasts only a day or two, or it may perhaps be entirely absent. Sometimes it runs on for two, four, or even five weeks. In some cases it is believed to constitute the whole of the disease, the patient recovering without more distinctive symptoms; this was suggested by Cullen, and it has been recently supported by Dr R. J. Lee and Dr Eustace Smith.

In all but these somewhat doubtful exceptions a *developed stage* succeeds, characterised by the appearance of the "whoop" with the cough. The change in question may occur either suddenly or very gradually, so that the boundary-line between the two stages is often not to be fixed with any certainty. The cough may on some one or two occasions be attended with a sound which raises the suspicion of an experienced nurse or visitor, but several days may pass without confirmation of their suspicion. On the other hand, some healthy children, especially if they happen to cough while crying, make a crowing inspiratory noise that is not very different from the true whoop.

But when whooping-cough is well marked, there can be no mistake about it. Pyrexia is now absent, but the pulse continues very frequent. There may be a good appetite, and except for the cough the child may appear to be well. The cough comes on in paroxysms, of which there may be only a few in the twenty-four hours, or as many as sixty or eighty. They are usually more frequent in the night than during the day. Each begins with a series of short explosions in rapid succession, and these expiratory efforts have no noticeable inspiratory movements between them; then, after the air in the lungs has been reduced to the utmost, there comes a long-drawn inspiration, attended with a loud whooping or crowing sound that gives the name to the disease. The short explosive coughs follow, and then the whoop. Such a succession of attacks may continue for several minutes, and generally end by expectoration of a viscid mucoid fluid, or sometimes by ejection of the contents of the stomach.

It may well be imagined that the paroxysms of whooping-cough, when severe, cause great distress. The child, when it feels one coming on, runs to its nurse for support, or it clings to a chair or table so as to diminish the shock as much as possible. Patients old enough to take notice describe a tickling sensation in the larynx, or a feeling of compression about the throat, as though the air could not pass. Those who are younger betray the approach of an attack by restlessness and anxiety of face.

As the paroxysms continue, the child may become intensely cyanotic. The eyes protrude, the face and the neck become swollen, and a cold sweat breaks out. *Hæmorrhages* are by no means infrequent; the expectoration may be stained bright red by blood (which probably comes from the fauces or from the larynx), the nose may bleed, one or both of the conjunctivæ may

become ecchymosed, the tears even may be mixed with blood, or the tympanic membrane may be ruptured, with the escape of blood from the ear on one side or on both. Steffen (in 'Ziemssen's Handbuch') says that albumen sometimes appears in the urine. He adds that a momentary stoppage of the heart is not uncommon, and cites a case, in a girl six years old, in which there was a temporary loss of sight during each attack. He also speaks of a boy nine years of age, in whom, when severe paroxysms occurred, internal strabismus of the right eye was noticed, whereas the left looked straight forward, being held fixed with tonic spasm. Sometimes spasmodic movements of the muscles of the face occur. General *convulsions* are not of very infrequent occurrence, and they may prove fatal.

After the subsidence of the paroxysm, the child may be out of breath for a time and glad to lie down; sometimes he complains of headache, and sometimes is dull or fretful. But in many cases he begins to play again at once, and seems as gay and lively as though nothing had happened. When there has been vomiting, he often asks for food and eats it greedily.

Sequelæ.—Among the more remote effects of the violence of the cough is the formation of one or more shallow whitish ulcers on the under surface of the tongue by the side of the frænum; they appear to be caused by the lower teeth, against which the tongue is forced outwards during the paroxysm. In this country Dr Thomas Morton first drew attention to these ulcers in a paper read before the Harveian Society in 1876; but on the Continent they had been previously described by Bouchard and others. Dr Morton detected them in about 40 per cent. of his cases, generally between the third and the fifth week. He once saw an ulcer in an infant who had no teeth, when the tongue may possibly have been injured by the edge of the gums. The recognition of these sublingual ulcers may sometimes be useful in diagnosis.

Bronchitis and broncho-pneumonia are the most frequent and most important sequelæ. They are usually combined with lobular collapse.

Another result of whooping-cough is the production of pulmonary emphysema, and even the extravasation of air into the interlobular and subpleural areolar tissue, whence in some rare cases it reaches the mediastinum, and may ultimately diffuse itself under the skin of the chest, throat, and limbs. Or a pulmonary alveolus may rupture, so that pneumothorax results. Either of these affections may cause a fatal result.

Ascending paralysis has been once or twice observed after whooping-cough, probably due to peripheral neuritis.*

Prognosis.—It very seldom happens that a paroxysm of whooping-cough directly destroys life. A very young child, however, may die as the result of a complete closure of the glottis, or perhaps from syncope, or from the rupture of an intra-cranial blood-vessel. When the attacks are very violent and follow one another with extreme frequency, they sometimes give rise to a condition of apathy and stupor which is attributed to the effusion of serum upon the brain and its membranes, and this may terminate in death.

Much more often fatal are the pulmonary complications of the disease, bronchitis and broncho-pneumonia; it is said that they kill half or two thirds of all the children attacked. As may easily be supposed, patients who before were weakly and delicate are more likely to succumb to whooping-cough than the strong and healthy. It is far more dangerous

* Dr Samuel West has recorded a case of right hemiplegia with aphasia and athetosis, probably due to cerebral hæmorrhage during a paroxysm of whooping-cough ('Brit. Med. Journ.,' Jan. 22nd, 1887).

among the poor than among the rich. There are, however, differences of severity in different epidemics. It is more serious in its results during the cold seasons of the year than in the summer. When it affects adult patients it is very distressing, but it is not dangerous, nor is its duration generally prolonged.

The tendency of whooping-cough is, after a variable period, to subside. The expectoration which ends the paroxysms becomes looser, more abundant, and more puriform. The violence of the cough lessens, and the characteristic whoop disappears. Perhaps the length of time during which it continues to be heard may be from six weeks to two or three months. In a case related by Trousseau its duration was only three days; the patient was a child three years old, an inmate of the Necker Hospital. After the paroxysms have ceased, symptoms of ordinary bronchial catarrh may remain for a time. The child may be a long while in regaining its appetite and strength, especially if the season happens to be winter, so that there is difficulty in getting it out of doors and into the fresh air. Sometimes, when the whoop has apparently passed off, a fresh attack of catarrh brings it back again for a few days in as marked a form as ever. Even after the lapse of a year it may be noticed that the cough arising out of a simple cold is attended with a somewhat similar sound.

In some cases, even when the paroxysmal stage of whooping-cough has completely passed off, the child nevertheless fails to recover its strength, and ultimately dies of marasmus. Not infrequently pulmonary phthisis, with caseous bronchial glands, develops itself as a sequela. Permanent deafness and otorrhœa are said to be occasional results of the injury sustained by the tympanic membrane during the paroxysms.

Pathology.—There is still considerable uncertainty with regard to the nature of whooping-cough. That its proper place is among the infective diseases is suggested not only by its marked contagiousness, but also by the fact that those persons who have once had it are protected against future attacks. Indeed, Steffen says that its occurrence for the second time in the same individual, although not absolutely impossible, is far more rare than that of scarlet fever, smallpox, or any other exanthem.

It is said to be one peculiarity of the contagion of whooping-cough that it is far less apt than most other contagia to be transmitted to a distance in an active state. At the Evelina Hospital for Children the whooping-cough ward is separated by a short passage only from other wards on the same floor, whereas the wards for cases of measles and scarlet fever are isolated in a different building. We very rarely find the contagion of whooping-cough conveyed by persons not themselves affected with the disease. However, one can hardly doubt that it might be spread by the use of handkerchiefs or towels contaminated by dried secretions from the air-passages of patients; and a case is recorded in which linen sent to be washed on some island from a ship, on board which there were children affected with whooping-cough, conveyed it to the inhabitants of the island. In vol. xi of the Clinical Society's 'Transactions' there is recorded an observation, made by Dr Bristowe, of a case in which a lady appeared clearly to have conveyed the contagion of the disease from Sydenham to London upon her dress. During a visit to the former place a boy affected with whooping-cough, was climbing upon her knee and coughing and sneezing over her; she returned home the same evening, and early next morning one of her children was found playing over her dress, which had

been laid upon an ottoman. This girl took the disease, and afterwards gave it to two other children. A further point of interest is that the boy himself had only begun to have a constant troublesome cough on the very day on which the lady visited him; in fact, he was staying away from home in the hope that he might escape the disease, which was prevailing among his brothers and sisters. The case is also important as tending to show that the period of incubation in whooping-cough is about a fortnight; for the girl fell ill exactly thirteen days after she was exposed to the contagion, and the two other children sickened after about the same interval.

Climate does not appear to have much influence upon the prevalence of the disease, except that perhaps cold and damp countries are more favourable to it. And Hirsch has shown that it is not more apt to be epidemic at one season of the year than another. There are doubtless great individual differences as regards susceptibility to the contagion.

Female children are decidedly more liable to be attacked than males. The age at which whooping-cough is most common is between the first year and the eighth. Of Dr Goodhart's 314 cases 62 were under a year old, 212 were between one and four, 65 between four and six, and 13 between six and ten. Barthez and Rilliet recorded the case of an infant whose mother had had the disease for three weeks before its birth, and in whom severe paroxysms occurred on the second day. Sir Thomas Watson relates in his lectures how the grandchild of his bedmaker at Cambridge whooped on the very day of birth, there having been another child affected with the disease in the same house for three weeks before. Dr Eustace Smith found more than a fourth of a series of cases occurred in children under one year old. On the other hand, whooping-cough is sometimes observed in adults up to forty or fifty, or even a still greater age.* Heberden met with one case in a woman aged seventy, and another in a man aged eighty.

An association is often traceable between epidemics of measles and those of whooping-cough, children falling ill with the latter disease soon after having passed through the former. Such cases are peculiarly apt to be accompanied with severe broncho-pneumonia, and to have a fatal termination, the exanthem having often already brought the lungs into a morbid condition. Sometimes, however, the relation between the two diseases is reversed, measles breaking out after whooping-cough has existed for some time. It is then sometimes noticed that the paroxysms become much less frequent and much less severe than before, and that they remain so as long as fever persists; but the same thing may happen in cases of whooping-cough when pyrexia appears from any cause.

As already remarked, whooping-cough differs in one very important feature from infective diseases in general, namely, in not being attended with pyrexia at the time when its more characteristic symptoms are manifested. There is, however, a parallel instance in hydrophobia, and it is not impossible that Pasteur's discovery of the actual presence of the virus of hydrophobia in the nervous centres, and of its multiplication there, may hereafter be found to supply the key to the pathology of whooping-cough. One can easily imagine that the poison of this disease, having originally entered the air-passages from without, and having set up a catarrh there, is during the prodromal stage conveyed to some part of the central nervous system, and there sets up the peculiar spasmodic cough.

* An eminent London physician suffered severely from an attack of whooping-cough when more than sixty-five years of age.

None of the theories propounded hitherto really throw light upon this disease. Some have regarded it as a pure neurosis; some have attributed it to pressure upon the vagus nerve by swollen tracheal or bronchial glands; some have maintained that it is a mere catarrh of the respiratory mucous membrane. Beare, on the ground that the paroxysms of whooping-cough resemble those that follow the entrance of a foreign body into the larynx, would have it that the fundamental lesion is an inflammation of the tract immediately above the vocal cords. Observers differ as to whether or not reddening of the laryngeal mucous membrane can be seen with the laryngoscope during life; according to Rossbach ('Berl. klin. Woch.,' 1880) no morbid change is discoverable, either in the larynx or in the upper part of the trachea; according to Meyer-Hüni ('Ztsch. f. klin. Med.,' 1880) there is marked reddening, and even slight swelling, of most parts of the larynx (but not of the cords), as well as of the trachea.

Recent observations render it probable that the contagious principle of whooping-cough is an organism analogous to those which produce so many other infective diseases, and that it has already been seen with the microscope. As far back as 1870 Letzerich described and figured in 'Virchow's Archiv' a fungus, consisting of thallus-filaments as well as of spores. This is found abundantly in the sputa of patients affected with whooping-cough, and he asserted that he had succeeded in cultivating it, and also in producing a like malady in rabbits by inoculating the trachea of these animals with the product of his cultivation experiments. In the 'Jahrbuch der Kinderheilkunde' for 1876 Tschamer supported Letzerich's views, and also maintained that an identical fungus, commonly found adhering to the surfaces of oranges or apples, is capable of giving rise to whooping-cough when inhaled into the human air-passages. Afterwards, however, Burger, in the 'Berliner klin. Wochenschrift' for 1883, cast doubt on the accuracy of Letzerich's observations, while at the same time he asserted that the sputum in whooping-cough always contains large quantities of bacteria, which appear as rods of oval form, sometimes constricted in the centre, which are generally scattered quite irregularly, but occasionally arranged in chains. They are said to be easily brought into view by staining with fuchsin or methyl violet.

Treatment.—We have no specific or effectual treatment of whooping-cough. Sydenham depended on venesection; Fothergill and Armstrong advocated nauseating doses of antimony, and many less probable modes of treatment, as arsenic and nitric acid, have been from time to time introduced, to be in turn forgotten.*

In bad weather the patient should be kept in a spacious room, warm and equable in its temperature; for exposure to cold has a marked tendency to bring on the paroxysms, although according to Hauke the presence of an excess of carbonic acid in the air has a like effect. But in fine weather it is right to allow a walk out of doors in the middle of the day. When the disease lingers in its course, nothing is so likely to bring it to an end as change of air, especially to the sea-side. The meals should be nourishing and frequent, and should be given directly after a paroxysm. Talking, crying, and excitement of every kind should as much as possible be avoided.

* Moreover some of them are not free from danger. In the same number of a medical journal which narrated successful treatment of whooping-cough by antipyrin and antifebrin, there appeared an account of a doctor in Germany who nearly killed his own child by repeated doses of antipyrin during whooping-cough.

As regards drugs, *belladonna*, hydrocyanic acid, chloral, bromide of potassium or ammonium, hemlock, appear each of them to diminish the frequency and the severity of the paroxysms in some cases, and even to shorten the duration of the disease. But not one of these medicines can be depended on even in most cases; and they often fail altogether. Dr Eustace Smith strongly recommends sulphate of zinc (gr. $\frac{1}{6}$) and solution of atropine (B. P.) $\mathfrak{m}$ss, gradually increased in amount and frequency. Alum was introduced by the late Dr Golding Bird as an astringent in the later stages of the disease, and has often proved useful. Quinine also is sometimes serviceable; and the supposition that it checks the growth of the specific microphytes has led to its administration by insufflation into the air-passages.

In fact, it has lately become a common practice to treat whooping-cough by inhalations. The earliest attempts of this kind consisted in placing patients in the purifying chambers of gas-works, where the air is laden with tarry products, as well as with sulphuretted hydrogen and ammonia. According to Roger ('Bull. de l'Acad.,' 1880) the evidence of the value of this treatment is very doubtful.

A more recent plan is to impregnate the air of the patient's chamber with turpentine, or with petroleum, or with carbolic acid. A solution of carbolic acid, for example, is diffused through the room by means of a spray apparatus, or by simply heating a vessel containing it. Children who are old enough may be made to inhale a weak carbolic spray for fifteen or twenty minutes two or three times a day. Thorner, in the 'Deutsches Archiv' for 1878, reports very favourably of this practice; for about a week there was little change, but at the end of that time the symptoms of the disease began rapidly to subside. Successful results by similar means are reported in this country. Inhalations of the vapour from a boiling 2 per cent. solution of salicylic acid have also been recommended. Dr Goodhart has found both carbolic and salicylic acids disappointing.

Marshall Hall's suggestion of protecting the infant at night from draughts by a mosquito curtain has been tried with success.

ASTHMA.*—Until the present century the word asthma was used to mean what we now call dyspnœa; hence the phrase "cardiac asthma" was often used, and even at the present day it is common to hear persons spoken of as "asthmatic" who are suffering from bronchitis and emphysema. Indeed, soon after the discovery of auscultation Rostan and some other French physicians were strongly disposed to deny the existence of any disease occurring in persons with healthy lungs which required the name of asthma. But that there is such a disease there can be no doubt; and now that spasmodic or true asthma has been separated from other affections with which it used to be confounded, its characters are found to be exceedingly definite and well marked.

Symptoms of an attack.—Asthma is a paroxysmal affection. It sets in generally with remarkable suddenness; most frequently in the middle of the night, between 2 and 4 a.m., but in some cases at other times, between 6 and 8 a.m., or in the afternoon. The forenoon is almost always the period in the day when the patient is freest from it. In the same case it commonly begins at about the same hour. When this is, as usual, between two and four in the morning, the patient, who may have gone to sleep in perfect health, wakes up with a sense of oppression of the chest which soon

* Ἄσθμα (*ἀσθμαίνω*), *panting*, is a Homeric as well as a Hippocratic word.

passes on to the most extreme distress of breathing. But sometimes the seizure is preceded by symptoms which previous experience enables him to recognise as premonitory; among them are a peculiar drowsiness, flatulence, a slight degree of sneezing, a troublesome itching under the chin, the passing of a quantity of pale limpid urine like that secreted in hysteria. The urgent dyspnœa of the developed attack compels him to sit up, and perhaps drives him to the window, which he throws wide open, in the hope of getting air more freely. Or he may be obliged to sit with his elbows planted upon a table, or to stand with his hands grasping the mantelpiece or some article of furniture above his head; such attitudes being adopted for the purpose of fixing the shoulders and so assisting the muscles of forced respiration in their action. His face, at first pallid, becomes livid or purple, his eyeballs start from their sockets, his hands and feet are cold, his skin is covered with a profuse sweat, and his expression indicates extreme anxiety. In fact, he may appear to be at the point of death. There is sometimes a preliminary sensation, paræsthesia of various kinds, which Romberg happily called an asthmatic aura.

Examination of the chest shows that the physical conditions are as follows:—The breathing is not accelerated but of normal frequency, or even slower than natural. Its rhythm is perverted, the inspiration being short, whereas the expiration is greatly prolonged. With the inspiration there may be slight wheezing, but nothing in comparison with that of expiration, which is audible all over the room. The shape of the chest is such as corresponds with a very deep inspiration; the upper ribs are raised to the fullest possible extent, and the diaphragm has descended towards the abdomen, so that the area of pulmonary resonance extends considerably lower than natural. During inspiration the sternomastoidei and the scaleni are brought into action, but there is scarcely any advance in the degree of expansion; during expiration there is but little recession, although the rigid abdominal muscles can be seen and felt to be doing their utmost to expel air from the lungs. Percussion shows much less than the natural amount of difference in the relation of the edges of the lungs to the heart and liver during inspiration and expiration. The percussion-note over the chest is over-resonant. On auscultation, the inspiratory vesicular murmur is found to be almost or quite inaudible; frequently sibilus is heard in its stead, or sonorous rhonchi. With expiration there is heard through the stethoscope the same loud wheezing sound which has been already mentioned as being audible at a distance.

So entirely occupied is the patient with the mere act of breathing that he can scarcely utter a word, or turn his head, or even stop to cough; but after a time a slight cough comes on, leading to the expectoration of a few greyish-white pellets of mucus. Not infrequently the mucus is stained with blood, and sometimes there is considerable hæmoptysis. Expectoration generally indicates that the symptoms are about to subside.

The duration of a paroxysm of asthma is very variable; usually it lasts from one to three hours, sometimes only a quarter of an hour, and occasionally with but slight remissions for a whole night or a day. As it passes off the patient falls asleep, and when he wakes in the morning his breathing may be quite easy. The temperature is not raised. The urine after a fit is, according to Ringer, deficient in both urea and salines. But sometimes the disease continues for several days in succession with scarcely any abatement, except that there is almost always some increase in its severity at night, and some lessening during the early part of the day. In

such cases the patient's condition causes extreme alarm, although a fatal termination scarcely ever occurs. In the following instance, however, the breathing actually ceased, and life was maintained only by artificial respiration. Several years ago, one of our students, a strong healthy young man, devoted to football, was admitted into the Clinical Ward labouring under a severe attack of asthma, to which he was subject. A few hours later, the house physician, who was sleeping in an adjoining room, was hastily summoned on account of an alarming failure of the patient's breathing. His respirations became more and more shallow, and at last they ceased altogether; he fell forwards in a state of insensibility, and remained unconscious for several minutes. Artificial respiration was resorted to at once, and with success. If medical aid had been less prompt there is little doubt that he would have died.

When asthma passes off in the usual way it is apt to return during the following night. The paroxysms may, in fact, recur for several successive nights, and may then cease, leaving the patient entirely free for weeks or months together; but there are other cases in which the disease shows itself night after night for years. A friend of the author's, who was liable to asthma for the last twenty-five or thirty years of his life, was never able to lie down to sleep; when night came on he dressed himself in a flannel suit, and seated himself in a large chair, where he remained till morning.

Diagnosis.—From the above description of asthma it will be apparent that the disease ought never to be mistaken for those tracheal or laryngeal affections (such as bilateral paralysis of the abductors of the cords) in which the dyspnœa is mainly inspiratory.

But it is often only by the history that one can tell whether a patient is suffering from asthma or from bronchitis and emphysema. According to Trousseau, a child (or even an adult) may be seized with what appears to be an acute and dangerous attack of broncho-pneumonia, with abundant moist sounds over the chest; and yet the subsidence of the symptoms in the course of a day or two, and the recurrence of like attacks on future occasions, ultimately show that the affection is really asthma.

The relations of bronchitis and emphysema to asthma are somewhat complicated. On the one hand, it is not uncommon for patients who have chronic bronchitis to suffer from time to time from paroxysms of dyspnœa, which cannot be accounted for by any increase of the bronchial inflammation, and which seem referable to a concomitant spasm of the air-tubes; if such spasm is regarded as the essential condition in asthma, it may be said that in these cases *secondary* asthma is present as a complication of the bronchitis. "Bronchitic asthma" was recognised by Salter; it is constantly worse in the winter than in the summer, which is not generally the case with primary asthma.

On the other hand, if a person with healthy thoracic viscera becomes subject to frequently recurring attacks of asthma, his lungs sooner or later become emphysematous. We have seen that during the paroxysm of asthma the ribs are raised, and the diaphragm at a lower level than natural; in other words, the lungs are in a state of over-distension. When the symptoms quickly pass off, as is usually the case, the chest walls return in a few hours to their normal position and the lungs to their normal size. But if similar attacks recur again and again at short intervals, the inevitable result is that the elasticity of the pulmonary tissue is impaired, and thus the alveoli become permanently over-stretched and emphysematous; ultimately the right side of the heart dilates, dropsy sets in, and there follow

the other mechanical results of general venous congestion, as in the last stage of chronic bronchitis.

Patients with confirmed asthma gradually acquire a characteristic aspect, which was well described by Salter. They are round-backed, high-shouldered, and stooping; the chest is obviously rigid and without pliancy, and from it the arms hang suspended, but inclined rather backwards and bent at the elbows. They are thin almost to emaciation, with prominent veins, cold thin hands, and a dusky complexion. The cheeks are hollow, the eyeballs turgid and watery, the mouth generally open, and the jaw rather hanging. The voice is feeble and somewhat hoarse and rough.

Ætiology.—With respect to the causes of asthma two questions have to be asked: (1) What are the *predisposing conditions* which render certain persons susceptible of the disease, whereas other persons seem to be incapable of being affected by it? (2) What are the various *exciting causes* which, in such individuals, are found to bring on the paroxysms?

The answer to the first of these questions must at present be incomplete, while of the *causa efficiens*, the invariable antecedent of asthma, either regarded as a paroxysm or as a diathesis, we are quite ignorant.

Inheritance has a share in the ætiology of asthma. Salter gives many striking instances of the transmission of asthma from generation to generation; and also mentions cases in which several brothers and sisters in a family were asthmatic without the parents being so. Walshe likewise gives instances of three or four brothers or sisters being asthmatic without the disease having appeared in the family before for at least the preceding generation. This kind of connection, which is not hereditary so much as consanguineous, we have already noticed in the case of certain undoubtedly nervous maladies, *e. g.* Thomsen's disease (p. 734). It is a point of great interest, and requires more investigation than it has yet received.

In early life many cases appear to be directly traceable either to *measles*, to *whooping-cough*, or less certainly to an attack of ordinary bronchitis; at the time the child seems to recover perfectly, but it becomes for the future liable to asthma. Other facts suggest that the disease is a *paroxysmal neurosis*. Thus Salter relates the case of an epileptic patient whose fits, after the usual premonitory symptoms, were on several occasions replaced by asthmatic paroxysms. This is, however, very exceptional; nor is it pretended that there is any connection between asthma and hysteria. Another of Salter's cases is that of a gentleman in whom a violent attack of asthma was twice suddenly excited by fear. Walshe also tells how an asthmatic patient, who had forgotten to take his cigarettes of stramonium and belladonna out with him, was so alarmed on discovering his omission that the dreaded attack at once came on.

Again, asthma is said to be sometimes closely related to gout. It has been observed to alternate with cutaneous eruptions, becoming worse when the skin has got better, and *vice versâ*. Sir Andrew Clark has particularly called attention to the frequent coexistence or alternation of asthma and *urticaria*, and has founded thereon the hypothesis that the immediate cause of a fit may be urticarial swelling of the bronchial mucous membrane, both the cutaneous and the mucous wheals being the result of sudden vaso-motor disturbance.*

The *age* at which patients first become affected with asthma is very variable. It sets in during childhood much more often than used to be

* See also a case in point related by Dr T. D. Pryce in the 'Lancet' for May, 1886.

supposed. Salter found that in a fourth of his cases it had begun before the tenth year, and he saw two cases in infants, one fourteen and the other twenty-eight days old. More *males* than females suffer from asthma, in the proportion of two to one.

The *exciting causes* of the asthmatic paroxysm vary widely in different cases. Indeed, hardly any two patients agree in their statements as to the precise conditions which bring on their attacks.

Particular kinds of weather, certain winds, cold air, the confined air of crowded rooms or railway carriages, act as exciting causes in some cases. Or the attack may follow the inhalation of dust, flue, or smoke, even the smoke of an extinguished candle or of a lucifer match.

Some patients are sure to be seized with asthma if they come near certain kinds of animals: cats, rabbits, dogs, horses, guinea-pigs, or the smaller Carnivora of a menagerie. Salter relates many remarkable cases of this kind, and what is especially noteworthy is that years have often passed before the patient discovered to what his sufferings were due. One man, a livery-stable keeper, was continually asthmatic until he retired from business, and then became almost entirely free; but whenever he went back among the horses the disease returned, and so at last he found out what was for him the special exciting cause. The writer once knew a lady who was attacked with asthma whenever she was in the same room with a cat; the animal could not be hidden anywhere near her without her discovering it by a painful sense of constriction in the air-passages.

The asthma produced by hay, or rather by the pollen of grasses, is one form of the disease known as hay-fever (p. 1134). Some patients never have asthma unless they are exposed to the influence of the pollen; others are habitually asthmatic, this being only one of many causes capable of exciting the disease in them. Many persons are attacked if they inhale the powder of ipecacuanha diffused in the air, and odours of various kinds act as exciting causes in particular cases.*

Diet plays an important part in setting up the paroxysms in almost all asthmatic patients. Heavy suppers and late dinners are very injurious; many asthmatic persons are unable to eat any solid food for several hours before bedtime. Special articles of food, among which are cheese, nuts, coffee, bottled stout, and wine, are apt to provoke the disease.

Another occasional cause of asthma is the presence of polypi in the nose. This was first pointed out by Voltolini; it has since been confirmed by Haenisch. Removal of the nasal growths frees the patient from the liability to the recurrence of the bronchial affection.

Salter mentions one case in which a paroxysm was sure to occur if the rectum was allowed to remain loaded. Sometimes, again, the attacks are clearly traceable to uterine irritation, as when they return with each catamenial period, or come on only during pregnancy or parturition.

But of all the exciting causes of asthma the most important in its influence is *locality*. And here, again, there are the strangest differences between different cases, so that it almost seems as if the disease were regulated by caprice. In certain places the patient is sure to be attacked; in others he is as sure to escape. As a rule, the places most favourable for asthmatic subjects are large, crowded, smoky towns, like London, Glasgow, and Manchester. The most extraordinary stories are related by Salter of the effects of London air. Persons whose lives had been rendered

* See the graphic narratives of such cases of asthma given by Watson and Trousseau.

miserable for years have become entirely free from asthma after moving to London. He thought that three fourths or seven eighths of all cases of asthma might be cured in this way. A patient of Dr Walshe found that he could only live in perfect freedom from his asthma in the Seven Dials. It is to be noted that the influence of locality extends to neutralising the ill effects of some other exciting causes of the paroxysms; the patient may be able while in London to eat what he pleases and at whatever hours, whereas in the country the strictest dieting may be required to keep off the disease. On the other hand, there are a few cases in which the air of the sea-side or of a bracing hilly district is found to be the best.

Pathology.—Various theories have been framed to account for the paroxysms of asthma, but even now the pathology of the disease cannot be said to be fully established. The *expiratory* character of the dyspnœa, exactly like that which accompanies capillary bronchitis, seems clearly to show that it must depend upon a morbid affection of the very small bronchial tubes within the lungs. During the act of expiration these tubes are as much exposed to pressure as the pulmonary alveoli themselves, and it is not difficult to suppose that, when from any cause they are partially obstructed, they may admit air into the lungs in inspiration, and yet refuse to allow it to pass out in expiration. The question is, What is the nature of the obstruction in asthma? The most obvious suggestion certainly is that it results from spasm of the muscular fibres in the walls of the tubes. That these fibres are capable of contracting, so as to narrow to some extent the calibre of the tubes, has been established by physiologists. The suddenness of the onset of the asthmatic paroxysm, the eqnally sudden way in which it sometimes subsides under the influence of a violent mental shock or emotion, the marked effect of such remedies as chloral, belladonna, and stramonium in bringing it to an end, the relation which appears to exist between asthma and certain neuroses, all favour the view that it is essentially spasmodic in nature. On the other hand, it is by no means clear that spasm can account for such considerable narrowing of the tubes as must be present in asthma; or that spasm can be kept up for so long a time as is sometimes occupied by a prolonged paroxysm of the disease.

Another hypothesis is that the mucous membrane of the tubes becomes very rapidly swollen by what German writers term a "fluxionary hyperæmia," or (as Weber put it) by "a dilatation of its blood-vessels through the influence of the vaso-motor nerves." The fact that the catarrhal form of hay-fever is attended with an obvious swelling of the mucous membrane of the nose is a strong point in favour of this view; since it seems unlikely for the asthmatic form of the same disease to be altogether different in pathology. Sir Andrew Clark's theory of the nature of this supposed swelling has been already referred to. Störk is said to have actually observed with the laryngeal mirror that during an asthmatic attack the whole length of the trachea and part of the right bronchus were deeply congested. It is possible that the small bronchial tubes may be affected both with hyperæmia and with spasm.

In 1871 Leyden discovered in the sputa certain pointed octohedral crystals, identical with those which are found in the blood and viscera in cases of leuchæmia, and commonly known as Charcot's crystals, having been first described by him. Leyden's idea was that these crystals might perhaps constitute the starting-point of the asthmatic paroxysm, by irritating the peripheral ends of the branches of the vagi in the bronchial

mucous membrane, and so setting up a reflex spasm of the muscular fibres beneath. But this is not at all probable, and they have been observed in the sputa of patients not suffering from asthma.

Prognosis.—When asthma occurs in childhood, it generally subsides about the age of puberty, leaving the patient free for the rest of his life. On the other hand, persons above the age of forty or forty-five seldom if ever get rid of a liability to it. The longer and the more frequent the paroxysms the more serious is the case. It is important to notice whether in the intervals between the attacks there is any shortness of breath, or cough with expectoration. For such symptoms indicate that the asthma is complicated with chronic bronchitis, or emphysema; and the presence of any permanent organic lesion of the lungs seriously adds to the gravity of the disease. The popular notion that "asthma is a lease of long life" is quite mistaken. It only points to the fact that death in the fits is extremely rare, and that so apparently severe a disease does not kill rapidly. All life insurance offices know that asthma leads in time to emphysema and its consequences, and reject or heavily "load" such cases.

Asthma does not tend to develop phthisis; although, on the other hand, there are cases which prove that it does not prevent its supervention.

Treatment.—This falls under two heads: we have, first, to prevent the recurrence of the asthma; and secondly, to relieve the attacks when they develop themselves, and, if possible, to cut them short.

In endeavouring to prevent the attacks, the most important thing is to study the exciting causes in the individual patient, and as far as possible to remove them. The digestive organs and the diet demand the first attention; then the place of residence. A drug which is in some cases very serviceable, although its mode of action is obscure, is the iodide of potassium; but it often utterly fails.

For the paroxysms of asthma different modes of treatment are useful, some in one case, some in another. Many patients are at once relieved when they are made faint and sick by an emetic dose of ipecacuanha or by smoking tobacco. The latter is said to be the best remedy for hay-asthma; unfortunately, those who smoke habitually get no benefit from it. In some cases nothing does so much good as strong hot coffee taken on an empty stomach, or hot whisky and water. In other cases the inhalation of chloroform gives very rapid, but generally only temporary relief. Smoking the leaves of stramonium (or of one of the other species of Datura) is often very effectual. Or the patient may try stramonium as a tincture or an extract, or the ethereal tincture of lobelia in full doses, or tincture of belladonna, or chloral. Some patients derive great benefit from the fumes of nitre-paper, burnt so as to fill the room with white smoke. In other cases nothing does so much good as the inhalation of a green powder which is sold as Himrod's cure for asthma in the United States, and is said to consist of nitre, stramonium, and aniseed. Chloroform and chloral hydrate are uncertain in their effects; and galvanism is probably useless.

For details in the management of cases of asthma the reader should consult the late Dr Hyde Salter's work, based as it is upon a large experience as well as upon his own sufferings from the disease. It contains on almost every page practical hints of the greatest value.

END OF VOL. I.

INDEX OF SUBJECTS

IN BOTH VOLUMES

The Roman numeral refers to the volume, the Arabic to the page.

A.

Abdomen in enteric fever, i, 145
Abdominal section, ii, 252, 271
Abiogenesis, i, 16
Abortion in chorea, i, 741
— in relapsing fever, i, 126
— in syphilis, i, 298
— in typhus, i, 43
— in variola, i, 211
Abscess of brain, i, 624
— of heart, ii, 50
— of hypochondrium, ii, 311
— of kidney, ii, 538
— of liver, ii, 358
— of mediastinum, i, 918
— of tonsil, ii, 140
Acarus scabiei, ii, 788
Accentuated second sound, ii, 63
Acetonæmia in diabetes, ii, 576
Achorion, ii, 868
Achromatopsia, i, 805
Acne, ii, 839
— atrophica, ii, 907
— cornée, ii, 844
— frontalis, ii, 844, *foot-note*
— rosacea, ii, 878
— tarsi, ii, 843
— varioliformis, ii, 844
Aconite in cardiac hypertrophy, ii, 43
Acrodynia, ii, 922
Acromegaly, i, 866
Actinomycosis, i, 365
Acupuncture for cardiac dropsy, ii, 85
— for renal dropsy, ii, 509
— for sciatica, i, 394
Acute bulbar paralysis, i, 512
— yellow atrophy of liver, ii, 375
Addison's disease, ii, 593
— anæmia, ii, 640
Adenoid growths in pharynx, ii, 144
Adenoma, i, 89
— sebaceum, ii, 927
Adherent pericardium, ii, 97
Adrenals, in *Addison's* disease, ii, 594
— diseases of, ii, 593
Ægophony, i, 940, 1025
Ætiology, i, 6
Age of cerebral aneurysm, i, 555
Age of cerebral embolism, i, 550
— of syphilitic arteritis of brain, i, 552
Agraphia, i, 571
Ague, i, 327 *et seq.*, 587, 786
Alalia, i, 569
Albinism, ii, 932
Albumen, estimation of, in urine, ii, 447
Albuminuria, ii, 444
— in morbus *Brightii*, ii, 480, 490, 497
— in cholera, i, 231
— in diabetes, ii, 587
— in diphtheria, i, 269
— in enteric fever, i, 140
— in erysipelas, i, 252
— in glanders, i, 361
— in hæmoglobinuria, ii, 436
— in lardaceous nephritis, ii, 490
— in mitral disease, ii, 79
— in pemphigus, ii, 818
— in pneumonia, i, 994
— in scarlatina, i, 187, 191
— in typhus, i, 110
— in variola, i, 208
— in yellow fever, i, 340
— febrile, ii, 453
— from congestion, ii, 452
— and life insurance, ii, 452
— physiological, ii, 449
— theory of, ii, 448
— toxic, ii, 453
Albuminuric retinitis, ii, 452
Alcohol, effect on brain, i, 791
— in treatment of diphtheria, i, 272
— — of irregular cardiac action, ii, 12
— — of typhus, i, 121
— — of enteric fever, i, 170
— — of pneumonia, i, 1001
— — of phthisis, i, 1112
— in relation to cirrhosis, ii, 369
— — to atrophy of brain, i, 689
— — to gout, ii, 677
— — to phthisis, i, 1091
— — to delirium tremens, i, 792
Alcoholic insanity, i, 842
— nausea, i, 797
— paralysis, i, 424
— paraplegia, i, 458
— tremor, i, 797
Alcoholism, acute, i, 791

Alcoholism, chronic, i, 796
Aleppo boil, ii, 922
Alexia, i, 569, *foot-note*
Alkaline urine, ii, 413
Allorrhythmia, i, 901
Alopecia, ii, 871
— areata, ii, 872
— febrilis, ii, 871
— syphilitica, ii, 899
— universalis, ii, 873
Alphos, ii, 804
Amaurosis from uræmia, ii, 470
— from cerebral tumour, i, 605
Amblyopia, crossed, i, 563
Ambulatory enteric fever, i, 142
Ammoniacal fermentation in urine, ii, 427
— urine without decomposition, ii, 430
Ammonium chloride in neuralgia, i, 393
— in myalgia, ii, 715
Amnesia, i, 572, 575
Amnesic aphasia, i, 575
Amphoric breathing, i, 934
— percussion note, i, 930
Amyloid disease, ii, 482; *see also* Lardaceous
Anæmia, ii, 608—649
— *Addison's*, ii, 640
— and dilatation of heart, ii, 41
— from Bilharzia, ii, 557
— cerebral, in epilepsy, i, 757
— Egyptian, ii, 294
— essential or idiopathic, ii, 640
— lymphatica, ii, 627
— in phthisis, i, 1063
— progressive pernicious, ii, 640
— primary, ii, 617
— from Sclerostomum, ii, 452
— of spinal cord, i, 451
— splenica, ii, 624, 626
— symptomatic, ii, 616
Anæmic murmurs, ii, 613
Anæsthesia, i, 402
— in chronic alcoholism, i, 797
— in hemiparaplegia, i, 469
— in hemiplegia, i, 564
— in hysteria, i, 804
— in leprosy, ii, 920
— in paraplegia, i, 480
— in peripheral paralysis, i, 402
— in sciatica, i, 391
— in tabes, ii, 526
Analgesia, i, 403, 804
— in paraplegia, i, 480
Anarthria, i, 508
— in bulbar paralysis, i, 508
Anasarca, *see* Dropsy
— without albuminuria, ii, 459
Anchylosis, *see* Ankylosis
Ancylostomum duodenale, ii, 293
Aneurysm, ii, 101–115
— abdominal, ii, 111
— of the aortic arch, ii, 104
— cerebral, a result of embolism, i, 555
— compressing trachea and bronchi, i, 917
— of heart, ii, 48
— miliary, of cerebral arteries, i, 559
— of pulmonary artery, i, 1070
— pulmonary, in phthisis, i, 1069
Aneurysm, thoracic, ii, 104
— treatment of, ii, 109
Angina, ii, 139; *see also* Cynanche
— pectoris, ii, 18
— — vaso-motoria, ii, 22
Angioma pigmentosum atrophicum, ii, 927
Angulus Ludovici in emphysema, i, 969
Anidrosis, ii, 855
Ankle-clonus, i, 434
Ankylosis in dengue, i, 347, 348
— from gonorrhœal synovitis, ii, 720
— osteo-arthritis, ii, 725
— from rheumatism, ii, 688
Anomalous fever, i, 176
Anorexia, ii, 169
— nervosa vel hysterica, i, 822
Anosmia, i, 421, 572, 609
Antagonism of other diseases to phthisis, i, 1105
Anthracosis of lungs, i, 1094
Anthrax, i, 350
— in animals, i, 350
— diagnosis of, from erysipelas, i, 254
— gastro-intestinal, i, 355
— pulmonary, i, 356
Anticipation of ague fit, i, 323
Antipyretics, i, 171, 173
Antipyrin in sick headache, i, 785
— in fever, i, 173
— in pneumonia, i, 1000
Aorta undeveloped in anæmia, ii, 636
Aortic aneurysm, i, 990; *see also* Aneurysm
— incompetence, ii, 67
— stenosis, ii, 67
Apex beat of heart, ii, 37
— of lung in phthisis, i, 1055
— pneumonia, i, 987
Aphasia, i, 569
— amnesic, i, 575
— association with anosmia, i, 576
— — hemiplegia, i, 570
— expression by gestures in, i, 577
— localisation of lesion in, i, 573
— theories of, i, 574
Aphemia, i, 569, *foot-note*
Aphonia, inflammatory, i, 890
— in hysteria, i, 805, 814
— neurotic, i, 875
Aphtha epizootica, i, 363
Aphthæ, ii, 132, 134
Apoplectic cicatrix, i, 557
Apoplexy, i, 572, 578
— in *Bright's* disease, ii, 588
— in general paralysis, i, 684
— in insular sclerosis, i, 545
— in *Menière's* disease, i, 769
— meningeal, i, 580, 590
— from organic disease of brain, i, 593
— prognosis and treatment of, i, 594
— simulation by malarial poisoning, i, 337
— from syphilitic arteritis, i, 585, 592
Arachnitis, *see* Meningitis
Arch of aorta, aneurysms of, ii, 104
Arcus senilis, ii, 52
Area, ii, 872
Argyll-Robertson pupil, i, 529
Arm, peripheral paralysis of, i, 396

Arsenic in treatment of ague, i, 338
— in cardiac disease, ii, 84
— in chorea, i, 744
— in eczema, ii, 781
— in *Hodgkin's* disease, ii, 633
— in idiopathic anæmia, ii, 648
— in lichen ruber, ii, 798
— in neuralgia, i, 393
— in osteoarthritis, ii, 730
— in pemphigus, ii, 820
— in prurigo, ii, 803
— in psoriasis, ii, 810
— in purpura, ii, 663
Arsenical eruption, ii, 837
— paralysis, i, 523
Arseniuretted hydrogen as a cause of hæmoglobinuria, ii, 433
Arteries, cerebral, distribution of, i, 552
— — obstruction of, i, 549
Arterio-capillary fibrosis, ii, 466
Arteritis, ii, 103
— syphilitic, i, 552
— in renal cirrhosis, ii, 464
Arthritis in dengue, i, 346, 348
— in secondary syphilis, i, 284
— atrophica, i, 530; ii, 731
— deformans, ii, 722
— gonorrhœal, ii, 720
— rheumatoid, ii, 722
— tabid, i, 530
— tubercular, i, 320
Arthropathic ataxique, i, 530
Ascaris lumbricoides, ii, 288
Ascites, ii, 323
— from cancer of biliary ducts, ii, 388
— from cancer of liver, ii, 387
— from cancerous peritonitis, ii, 322
— from cirrhosis of liver, ii, 372
— from perihepatitis, ii, 374
— from chronic peritonitis, ii, 317
— from pylephlebitis adhæsiva, ii, 328
— from syphilitic hepatitis, ii, 374
— tubercular peritonitis, ii, 320
Aspect in phthisis, i, 1065
Aspiration, ii, 366
— in empyema, i, 1041
— in pleuritic effusion, i, 1040
Asteatosis, ii, 846; *see also* Xerodermia
Asthma, i, 1142
— from hay, i, 1134, 1146
Atavism in gout, ii, 676
— in hæmophilia, ii, 664
— in phthisis, i, 1067
Ataxia of muscles, i, 525
Atelectasis pulmonum, i, 958
Atheroma, ii, 103
— of cerebral arteries, i, 558
— of pulmonary artery, i, 961
Athetosis, i, 569, 701, 746
Atrophic arthritis, ii, 731
— paralysis, i, 488 *et seq.*
Atrophy of the brain, i, 680
— of kidney from calculus, ii, 527
— of liver, ii, 374
— of muscles after paralysis, i, 435
— — in acute atrophic paralysis, i, 490
— — in chronic atrophic paralysis, i, 495
Atrophy of muscles in progressive atrophic paralysis, i, 497
Atropine, *see* Belladonna
Auditory effects in paroxysmal vertigo, i, 769
— — in meningitis, i, 652, 656
— vertigo, i, 771
Aura epileptica, i, 751
Auscultation, i, 931
— theory of, i, 935
— in diagnosis of tracheal stenosis, i, 921
— of the breathing, i, 933
— of the heart, ii, 63
— of the voice, i, 938
— of the œsophagus, ii, 150
Azoturia, ii, 408

B.

Bacillicides in phthisis, i, 1113
Bacilli in genere, i, 14
Bacilluria aseptica, ii, 430
Bacillus anthracis, i, 351, 357
— of cholera, i, 238
— of lepra, ii, 920
— of lupus, ii, 909
— of malaria (?), i, 335
— of rhinoscleroma, ii, 917
— of tuberculosis, i, 305, 312, 1055; ii, 214
— — in sputum, i, 1067
— subtilis, i, 351
Bacteria in genere, i, 13
— in bromidrosis, ii, 856
— in diphtheria, i, 261
— in enteric fever, i, 160
— in pyæmia, i, 63
— in scarlatina, i, 186
Bacterium lactis, i, 14 *et seq.*
— termo, i, 262
Banting treatment of obesity, ii, 390
Barium chloride in paralysis agitans, i, 719
Barrel-shaped chest, ii, 969
Basal phthisis, i, 1056
Basedow's disease, ii, 126
Baths, cold, in enteric fever, i, 171
— — in pneumonia, i, 1000
— — in rheumatic hyperpyrexia, ii, 710
— in eczema, ii, 778
— in paraplegia, i, 449
— in spastic paraplegia, i, 483
— in tabes, i, 537
— of Bath, in dyspepsia, ii, 164; also, ii, 731
— of Buxton, in osteo-arthritis, ii, 731
— of Droitwich, in spinal meningitis, i, 487
— of Gastein, in hemiplegia, i, 596
— — in hypochondriasis, i, 825
— of Harrowgate, ii, 164, 778
— of Nauheim, in spinal meningitis, i, 487
— — in locomotor ataxy, i, 537
— of Pfeffers, in hemiplegia, i, 596
— of Ragatz, in myelitis, i, 449
— of Rehme, in spinal meningitis, i, 487
— of Schlangenbad, in myelitis, i, 449
— of Strathpeffer, in pityriasis rubra, ii, 814
— of Teplitz, in myelitis, i, 449
— of Vals, in gout, ii, 685
— of Vichy, in gout, ii, 685
— of Wiesbaden, in myelitis, i, 449

Baths, hot air, in uræmia, ii, 475
— warm, in *Bright's* disease, ii, 508
— — in croup, ii, 889
Beaded hair, ii, 875
Beading of ribs in rickets, ii, 735
Bedsore acute, i, 565
— electrolytic treatment of, ii, 448
— in enteric fever, i, 149
— in paraplegia, i, 438
Belladonna in aortic disease, ii, 86
— in constipation, ii, 212
— in diaphoresis, i, 1113, ii, 856
— in epilepsy, i, 766
— in pertussis, i, 1142
— toxic rash from, ii, 836
Bell-sound, i, 1048
Belloc's cannula, i, 1134
Bellows murmur, ii, 64
Beri-beri, i, 426
Bile acids, tests for, ii, 339
Bilharzia hæmatobia, ii, 304, 557
Biliary colic and obstruction, ii, 269
— cirrhosis, ii, 369
Bilious disorders, ii, 331
Bilirubin, ii, 338
Bird-claw hand, i, 398, 497
Bird's-nest cells, i, 91
Birth-palsy, i, 746
Bismuth as a test for glycose, ii, 567
— in diarrhœa, ii, 219
— in gastritis, ii, 164
— internally in erythema, ii, 833–4
Black vomit in yellow fever, i, 340
Bladder, hypertrophied, in diabetes, ii, 578
— in paraplegia, i, 435
Bladder-worm, ii, 545
Bleb, *see* Bulla
Bleeders, ii, 664
Blepharospasmus, i, 701
Blistering for pericarditis, ii, 100
— for pleurisy, i, 1036
— for rheumatism, ii, 701
— for ringworm, ii, 863
— for sciatica, i, 394
Blister-test of gout, ii, 673
Blood, in *Bright's* disease, ii, 457
— in chlorosis, ii, 637
— in cholera, i, 233
— diseases of, ii, 608
Blood, estimate of hæmoglobin in, ii, 611
— — corpuscles in, ii, 611
— in gout, ii, 675
— in *Hodgkin's* disease, ii, 629
— in leuchæmia, ii, 621
— in purpura, ii, 661
Blood-letting in apoplexy, i, 595
— — in aneurysm, ii, 109
— — in capillary bronchitis, i, 978
— — in epilepsy, i, 765
— — in pneumonia, i, 999, 1001
— — in uræmia, ii, 509
Blue line on gums, ii, 207
Boils, ii, 852
— in diabetes, ii, 574
— in smallpox, i, 210
Bones in congenital syphilis, i, 302, 303
— in mollities ossium, ii, 747–8
Bones in osteitis deformans, ii, 749
— in rickets, ii, 734
— in sarcomatosis, i, 86
Boroglycerine in ringworm, ii, 865
Bothriocephalus latus, ii, 283
Bovine tuberculosis, i, 315; ii, 201
Brain, diseases of, i, 548–692; *see* Cerebral
— abscess of, i, 624–632
— anæmia of, in epilepsy, i, 802
— arteries, distribution of, i, 552
— — obstruction of, i, 549
— and cord in diabetes, ii, 578
— cortical centres of, i, 613
— cysts of, i, 599
— glioma of, i, 601
— gumma of, i, 599
— hydatid of, ii, 600
— hypertrophy of, i, 691
— injury to, diagnosis from apoplexy, i, 584
— in rickets, ii, 737
— softening of, i, 553, 632
— tubercle of, i, 597
— tumours of, i, 597, 610, 624
Brass-founders' ague, i, 523
Breast-pang, ii, 18
Breath in apoplexy, i, 579
— odour of, in diabetes, ii, 573
Breathing, amphoric, i, 934
— bronchial, i, 934
— cavernous, i, 934, *foot-note*
— cog-wheel, i, 1072
— puerile, i, 934
— tubular, i, 934
Bright's disease, ii, 439–510.
— — forms of, ii, 441, 506
— — and cerebral hæmorrhage, i, 558
— — and gout, ii, 681
Bromide rash, ii, 835
Bromides in epilepsy, i, 765
— in eczema, ii, 779
Bromidrosis, ii, 856
Bromism, i, 800
Bronchial breathing, i, 934
— — in phthisis, i, 1073
— — in pneumonia, i, 990
Bronchiectasis, i, 972
— in cirrhosis of lung, i, 1010
Bronchitic asthma, i, 1144
Bronchitis, i, 951—984
— acute, i, 955
— capillary, i, 955
— chronic, i, 960
— typhoid, i, 148
— fœtid, i, 974
— plastic, i, 980
— as a cause of phthisis, i, 1098
Bronchocele, *see* Goitre
Bronchophony, i, 939, 990
Broncho-pneumonia, i, 1003
— association with pleurisy, i, 1004
Bronchorrhœa serosa, i, 962
Brow-ague, i, 328, 787
Brown induration of lung, ii, 60
Brucia, poisoning by, i, 724
Bruit d'airain, i, 1048
— apical, in Bright's disease, ii, 500
— in chorea, i, 780

Bruit de diable, ii, 613
— de pot fêlé, i, 930, 1073
— de râpe, ii, 64
— de scie, ii, 64
— de souffle, ii, 64
— diastolic, in arteries, ii, 68, *foot-note*
— d'oboe, ii, 64
— without valvular lesion, ii, 38, 613
Bruits, cardiac, *see* Murmurs
Bubo in plague, i, 221
— in syphilis, i, 283
— in typhus, i, 113
Bucnemia, *see* Elephantiasis
Bulbar paralysis, i, 506
Bulimia, ii, 169
Bulla, ii, 757, 815, 816, 828, 898
Burmese ringworm, ii, 866
Butyl chloral in sick headache, i, 785
— — in facial neuralgia, i, 393

C.

Cachexia, cancerous, i, 72
— malarial, i, 329, 338
Caffein in cardiac disease, ii, 86
Caisson-disease, i, 453
Calcareous concretions in phthisis, i, 1071
— degeneration of arteries, ii, 103
— — of heart, ii, 52
Calcic oxalate in urine, ii, 422
— phosphate in urine, ii, 424
— sulphate in urine, ii, 427
Calculi, biliary, ii, 350
— urinary, ii, 511
— — fusible, ii, 515
— — mulberry, ii, 513
— — operative treatment of, ii, 542
— — of calcic carbonate, ii, 514
— — of calcic oxalate, ii, 512
— — of calcic phosphate, ii, 514
— — of cystin, ii, 513
— — of indigo, ii, 514
— — of triple phosphate, ii, 515
— — of urate of soda, ii, 512
— — of uric acid, ii, 512
— — urostealith, ii, 514
— — of xanthine, ii, 513
Calculous pyelitis, ii, 537
Callosities, ii, 880
Cancer, i, 71, *foot-note*; *see* Carcinoma
Cancer-bodies, i, 90
Cancer-cell, i, 88
Cancer-juice, i, 90
Cancer of the biliary passages, ii, 387
— of the bowel, ii, 217
— of brain, i, 599
— of gall-bladder, ii, 353, 387
— of kidney, ii, 549
— of larynx, i, 912
— of liver, ii, 384
— of lung, ii, 121
— of œsophagus, ii, 148
— of pancreas, ii, 60
— of peritoneum, ii, 321
— of rectum, ii, 235
— of skin, ii, 930
Cancer of stomach, ii, 189
— of vertebræ, i, 472
Cancroid, i, 91
Cancrum oris, ii, 133
Canities, ii, 933
Capillary bronchitis, i, 956
Carbolic oil in ringworm, ii, 863
Carbuncle, ii, 855
— facial, diagnosis from anthrax, i, 354
— in diabetes, ii, 574
— in plague, i, 222
Carcinoma, i, 71, 89 *et seq.*; *see also* Cancer
— and tubercle, i, 1105
— colloid, i, 94
— columnar, i, 92
— criteria of, i, 72
— cylinder, i, 92
— distribution of, i, 74
— epithelial, i, 90
— keratoid, i, 90
— lipomatosum, ii, 550
— origin of, i, 92
— scirrhous, i, 90
Carcinomatin, i, 72
Cardiac disease, ii, 1 *et seq.*
— — from rheumatism, ii, 690
— hypertrophy, ii, 30
— — in Bright's disease, ii, 463 *et seq.*
— sounds in phthisis, i, 1072
— — in valvular disease, ii, 63
Cardialgia, ii, 24
Cardinal's case (hydrocephalus), i, 667, 669
Cardio-vascular changes in morbus *Brightii*, ii, 463 *et seq.*
Carditis, ii, 53
Caries of spine causing paraplegia, i, 471
— — in Addison's disease, ii, 600
Carnification of lung, i, 1021
Carotid ligature causing hemiplegia, i, 549
Carpopedal contractions, i, 711
Carswell's grapes in phthisis, i, 1057
Caseation in caries of spine, i, 481
— of carcinoma, i, 93
— of inflammatory products, i, 56
— of lungs in phthisis, i, 1056
— of sarcomata, i, 81
— of tubercle, i, 306, 311
— a source of general tuberculosis, i, 214
Casts, renal, ii, 454, 480, 490, 498
— — blood, ii, 454
— — epithelial, ii, 454
— — false, ii, 455
— — fatty, ii, 454
— — granular, ii, 454
— — in hæmoglobinuria, ii, 436
— — hyaline, ii, 454
— — inflammatory or corpuscular, ii, 454
— — lardaceous, ii, 456
Catalepsy, i, 814
— epileptic, i, 750
— hysterical, ii, 817
Cataract, diabetic, ii, 574
Catarrh, nasal, i, 1121
Catarrhal inflammation, i, 51
— pneumonia, i, 1003
Catarrhe sec, i, 961
Causalgia, i, 404

Cauterisation in hydrophobia, i, 877
— in spasmodic torticollis, i, 698
Cavernous breathing, i, 934, *foot-note*
Cephalalgia, *see* Headache
Cerebellum, tumours of, i, 610
Cerebral abscess, i, 624; *see* Brain
— aneurysm, i, 559
— atrophy, i, 688
— embolism, i, 551
— hæmorrhage, i, 555
— — miliary aneurysms in, i, 559
— — in renal disease, i, 556
— — seats of, i, 556
— — traumatic, i, 628
— — from tumour, i, 592
— — statistics of locality, i, 556
— hypertrophy, i, 691
— meninges, new growths in, i, 597
— — inflammation of, i, 535 *et seq.*
— softening, i, 554, 601, 632
— symptoms in cirrhosis of liver, ii, 372
— — in diabetes, ii, 576
— — in enteric fever, i, 140
— — in rheumatism, ii, 698
— — from salicylates, ii, 708
— — in typhus, i, 107
— — in yellow atrophy of liver, ii, 378
— thrombosis, i, 551
— tubercle, i, 597
— tumours, i, 597, 624
Cerebritis, *see* Encephalitis
Cerebro-spinal fever, i, 653
— — epidemic, i, 654
— — simple, i, 662
— fluid in chronic hydrocephalus, i, 664
— — normal characters of, i, 664
Certificates in cases of insanity, i, 848
Cestoidea, ii, 277
Chalicosis of lungs, i, 1096
Chancre, i, 280
— infecting and mixed, i, 281
— non-infecting, i, 280
Chaps or fissures, ii, 758
Charbon, i, 354
Charcot's crystals in asthma, i, 1147
— — in leuchæmia, ii, 621
— joint disease, i, 529; ii, 731
Cheloid, ii, 922
Cheyne-Stokes respiration, i, 945
— — in apoplexy, i, 579
— — in cardiac disease, ii, 62
— — in uræmia, ii, 470
Chicken-pox, *see* Varicella
Chilblain, ii, 880
Children, diabetes in, ii, 587
— disease of heart in, ii, 90
— dyspepsia in, ii, 165
— enteric fever in, i, 143, 168
— phthisis in, i, 1082
— pneumonia in, i, 998, 1003
— psoriasis in, ii, 808
— rheumatism in, ii, 701
— scabies in, ii, 791
— treatment of constipation in, ii, 212
Chiro-pompholyx, ii, 819
Chloasma, ii, 935
Chloral hydrate in chorea, i, 745
Chloral hydrate in eczema, ii, 779
— — in tetanilla, i, 713
Chlorate of potash a cause of hæmoglobinuria, ii, 433
— — in stomatitis, ii, 133
Chloroform in angina pectoris, ii, 27
— in biliary colic, ii, 354
— reducing copper in urine, ii, 566
Chlorosis, ii, 635
Choked optic disc, i, 606
Cholecystitis, ii, 355
Cholelithotomy, ii, 355
Cholesteatoma, i, 600; ii, 846
Cholera, i, 226
— nostras, or English cholera, i, 242
Cholera-typhoid, i, 232
Choleraic diarrhœa, i, 232
Cholerine, i, 232
Cholesterin, ii, 350
Chorea, i, 728
— "electrical," i, 747
— gravis, i, 731
— *Huntingdon's*, hereditary, i, 747
— major, i, 701, 818
— of dogs, i, 734
— post-hemiplegic, i, 746
— relation to rheumatism, i, 741
Choroid, tubercle of, i, 1119
Chromidrosis, ii, 856
Chrysophanic acid in psoriasis, ii, 809
— — in ringworm, ii, 864
Chyluria, ii, 555
Cicatrices and cheloid, ii, 876
— of lupus, ii, 905
— syphilitic, ii, 900
Cirrhosis of kidneys, ii, 444
— of liver, ii, 367
— — in ague, i, 330
— — hypertrophic form, ii, 373
— — a cause of jaundice, ii, 343
— — syphilitic, ii, 374
— of lung, i, 1007
— of stomach, ii, 302
Classification of diseases, i, 7, 9, 98, 330
— — of the nervous system, i, 382, 433, 713
— — of the skin, ii, 752, 753
— of mental disorders, i, 826, 827
Clavus, ii, 881
— hystericus, i, 775
Clergyman's sore throat, ii, 143
Climate in treatment of m. *Brightii*, ii, 510
— — of bronchitis, i, 980
— — of osteoarthritis, ii, 731
— — of phthisis, i, 1109
Clinical thermometer, i, 27
Clonus, i, 434
Cnidosis, *see* Urticaria
Coagulation of blood, ii, 123
Cocaine in laryngeal phthisis, i, 879
Codeia in treatment of diabetes, ii, 591
Cod-liver oil in lupus, ii, 913
— — in osteo-arthritis, ii, 731
— — in phthisis, i, 1112
Coffee-ground vomit, ii, 194
Cog-wheel respiration, i, 1072
Colchicum in gout, ii, 683
— in sciatica, i, 392

Cold as a cause of bronchitis, i, 976
— — hæmoglobinuria, ii, 435
— — myelitis, i, 442
— — nephritis, ii, 476
— — phthisis, i, 1098
— — pleurisy, i, 1033
— — pneumonia, i, 996
— — rheumatism, ii, 692
— baths in treatment of fever, i, 171
— — — of gout, ii, 685
— — — of meningitis, i, 487
— — — of pneumonia, i, 1000
— — — of rheumatism, ii, 710
Colic, biliary, ii, 269
— intestinal, ii, 203
— plumbic, ii, 205
— renal, ii, 521
Colitis, acute catarrhal, ii, 240
— dysenteric, ii, 242
— membranous, ii, 241
— ulcerative, ii, 241
Collapse, ii, 15
— in biliary colic, ii, 269
— in cholera, i, 228
— in hysteria, i, 821
— in acute obstruction of intestine, ii, 268
— in peritonitis, ii, 306
— in pneumonia, i, 994
Collapse of pulmonary tissue, i, 958
Colloid cancer, i, 94
— — of peritoneum, ii, 323
— — of stomach, ii, 198
— milium, ii, 927
Colotomy, ii, 267
Colour-blindness in hemiplegia, i, 567
— in hysteria, i, 804
— in locomotor ataxy, i, 529
Columnar sclerosis, i, 488
Coma in ague, i, 327
— in alcoholic poisoning, i, 791
— in apoplexy, i, 578
— from cerebral abscess, i, 630
— — tumour, i, 604
— diabetic, ii, 576
— in enteric fever, i, 144
— epileptic, i, 752
— in general paralysis of the insane, i, 684
— from heat-stroke, i, 788
— in meningitis, i, 651
— from narcotic poisoning, i, 585
— traumatic, i, 584
— uræmic, i, 587
— in delirium tremens, i, 586
— in variola, i, 202
Coma-vigil in typhus, i, 111
Comedo, ii, 844
Comparative pathology of anthrax and other epizootic diseases, i, 350–379
— of *Bright's* disease, ii, 508
— enterica, i, 136
— osteoarthritis, ii, 729
— rickets, ii, 743
— tuberculosis, i, 315; ii, 1107
— variola, i, 214
Compensation in valvular disease, ii, 69
Composite portraits of phthisis, i, 1089
Compound granule-masses in acute spinal sclerosis, i, 490
— — in cerebral softening, i, 632
— — in chronic atrophic paralysis, 496
— — in myelitis, i, 440
Compressed air in bronchitis, i, 979
Compression of intestine, ii, 254
Compression-paraplegia, i, 470
Concretions, gastric, ii, 202
— pulmonary, in phthisis, i, 1069
Concussion of spinal cord, i, 454
Condyloma, ii, 884
— flat, i, 287
— of larynx, i, 901
Congenital disease of heart, ii, 87
— ichthyosis, ii, 888
— syphilis, i, 297–304
— — affecting larynx, i, 903
Conium in facial neuralgia, i, 393
Conjugate deviation of eyes, i, 563
Conjunctiva in scurvy, ii, 663
Consecutive Bright's disease, ii, 444, 502
Consonating râles, i, 938
Constipation, functional, ii, 210
— from acute intestinal obstruction, ii, 261
— from chronic obstruction, ii, 263
— from peritonitis, ii, 306
Contagia, destruction of, i, 23
Contagia, persistence of, i, 23
— criteria of, i, 11
— transference of, i, 20
Contagion, i, 11–26; *see also* Infection
— of ague, i, 335
— of anthrax, i, 352
— of cholera, i, 237
— of dengue, i, 348
— of diphtheria, i, 260
— of enteric fever, i, 160
— of erysipelas, i, 247
— of glanders, i, 359
— of hydrophobia, i, 371
— of influenza, i, 1129
— of measles, i, 178
— of pertussis, i, 1139
— of phthisis, i, 1083
— of plague, i, 223
— of relapsing fever, i, 129
— of rubeola, i, 196
— of scarlatina, i, 185
— of tubercle, i, 313
— of typhus, i, 113
— of varicella, i, 219
— of yellow fever, i, 341, 343
Contagious pus, i, 15
Contagium vivum, i, 13 *et seq.*
Contraction of intestine, ii, 255
Contracture of muscles in hysteria, i, 806
— — after facial paralysis, i, 409
Convallaria in cardiac disease, ii, 86
Convulsions—
— in acute alcoholism, i, 791
— in chronic alcoholism, i, 797
— from cerebral abscess, i, 629
— from cerebral tumours, i, 604
— from disseminated sclerosis, i, 683
— epileptic, i, 752
— in general paralysis, i, 683

Convulsions—
— in hysteria, i, 808, 809
— from hydrocephalus, i, 668
— infantile, i, 761
— from meningeal hæmorrhage, i, 679
— from meningitis, i, 650
— puerperal, i, 761
— in scarlatina, i, 190
— in uræmia, ii, 469
Copaiba rash, ii, 834
Copper test for sugar in urine, ii, 564
Cord, *see* Spinal cord
Corneal ulcers, *see* Keratitis
Corns, ii, 881
Cornu cutaneum, ii, 888
Coronary arteries in angina pectoris, ii, 23
— thrombosis, ii, 46 *et seq.*
Corpuscular richness of the blood, ii, 611
Corrigan's pulse, ii, 68
Cortical centres, i, 614
Corymbose smallpox, i, 209
Coryza, i, 1121
— in measles, i, 179
— in rötheln, i, 197
Coster's paste, ii, 864
Cough, i, 946
— from affections of the ear, i, 947
— in bronchitis, i, 951
— in pertussis, i, 1137
— in phthisis, i, 1066
— in pleurisy, i, 1027
— in pneumonia, i, 992
— from throat, i, 947
Couperose, *see* Gutta rosea
Cow-pox, i, 211
Cracked-pot sound, i, 930, 1073
Craniotabes in congenital syphilis, i, 303
— in rickets, ii, 737
Crateriform ulcer, ii, 931
Crepitation, i, 937
— pneumonic, i, 989
Cretinism, i, 860 *et seq.*
— sporadic, i, 861
Cretinoid condition in adults, i, 863
Crisis in intermittent form of ague, i, 825
— in remittent form, i, 329
— in enteric fever, i, 141
— in erysipelas, i, 252
— in measles, i, 180
— in acute pneumonia, i, 994
— in pyrexia generally, i, 37
— in relapsing fever, i, 127
— in scarlatina, i, 187
— in typhus, i, 110
Criteria of pathogenetic organisms, i, 12
Croton chloral, *see* Butyl-chloral
Croup, i, 881
— relation to diphtheria, i, 257, 264
— symptoms of, i, 887
— treatment of, i, 889
Crust or scab, ii, 757
Crutch paralysis, i, 396
Cruveilhier's paralysis, i, 497
Crystals of fatty acid in sputum, i, 974
Cubebs-rash, ii, 834
Cultivation of microzymes, i, 15, 18
Cupping in treatment of pneumothorax, i, 1052
— — acute nephritis, ii, 508
Curare in hydrophobia, i, 377
— in tetanus, i, 727
Cutis anserina in ague, i, 824
— pendula, ii, 893
Cyanosis and tubercle, ii, 88
— from bronchitis, i, 952
— from cardiac disease, ii, 61
— from emphysema, i, 968
— of fingers of nervous origin, ii, 118
— of skin, ii, 756
Cyclic albuminuria, ii, 449
Cylindroids in urine, ii, 455, *foot-note*
Cylindroma, i, 92
Cynanche, ii, 139
Cyphosis, ii, 736
Cyrtometer, i, 942
Cystic disease of kidneys, ii, 444, 504
— liver and kidneys, ii, 505
Cysticercus, ii, 280—282
— cellulosæ, ii, 280
— of brain, i, 600
— of subcutaneous tissue, ii, 281
Cystine in urine, ii, 426
— calculus, ii, 427
Cystitis in paraplegia, i, 437
— tubercular, ii, 546
— with ammoniacal urine, ii, 429
Cysts of brain, i, 600
— in granular kidney, ii, 496
— large single renal, ii, 533–4
— mucous, of larynx, i, 911
— in pancreas, ii, 604
— parovarian, ii, 326

D.

Dancing mania, i, 819
Dandy fever or dengue, i, 346
Dartrous diathesis, ii, 770
Deaf-mutism from meningitis, i, 656
Deafness from cerebral tumour, i, 610
— and meningitis, i, 652
— from salicylates, ii, 708
— in epidemic meningitis, i, 656
— in paroxysmal vertigo, i, 769
— in typhus, i, 107
Death by asphyxia, ii, 14
— by collapse, ii, 15
— by syncope, ii, 14
Decubitus acutus in hemiplegia, i, 568
— in paraplegia, i, 438
Deep inflammation of the skin, ii, 876
Defervescence, i, 30
Deformities from atrophic paralysis, i, 491
— from gout, ii, 679
— from osteo-arthritis, ii, 724
— from mollities ossium, ii, 747
— from rickets, ii, 734–737
Degeneration, lardaceous, ii, 482–487
— calcareous, i, 1071; ii, 52, 103
— fatty, ii, 50
Delayed sensations, i, 526
Delhi boil, ii, 922

Délire des grandeurs with disseminated sclerosis, i, 544
— in general paralysis of the insane, i, 682
Delirium in epilepsy, i, 754
— in enteric fever, i, 141
— in meningitis, i, 651
— in pneumonia, i, 994
— in rheumatism, ii, 698
— in smallpox, i, 208
— in typhus, i, 105, 107
— tremens, i, 791
Deltoid, paralysis of, i, 397
Delusional insanity, i, 837
Delusions in acute delirious mania, i, 830
— in chronic mania, i, 832
— in general paralysis, i, 682
— in melancholia, i, 835
Dementia, acute, i, 836
— from chronic alcoholism, i, 797
— in epilepsy, i, 755
— in general paralysis, i, 684
— paralytica, i, 680
Demodex folliculorum, ii, 839
Dengue, i, 346
Dentition in rickets, ii, 736
Depth of cutaneous diseases, ii, 759
Dermatitis, chronic, ii, 713
— in Bright's disease, ii, 460
— deep, ii, 876
— due to scabies, ii, 790
— herpetiformis, ii, 829
— papillomatosa capilitii, ii, 852
— superficial, ii, 763
Dermatology, historical sketch, ii, 754
Dermatolysis, ii, 893
Dermato-syphilis, ii, 896
Desquamation in cholera, i, 348
— in dengue, i, 231
— in eczema, ii, 766
— in erysipelas, i, 252
— in measles, i, 181
— in psoriasis, ii, 804
— in pityriasis rubra, ii, 813
— in rubeola, i, 197
— in scarlatina, i, 189
Dextro-sigmoid disease, ii, 79
Diabetes, ii, 563–592
— and xanthoma, ii, 895
— insipidus, ii, 408
— mellitus, ii, 563
— — phthisis in, ii, 578
— — theories of, ii, 580
Diabetic coma, ii, 575
Diagnosis, general, i, 4
Diapedesis, i, 44
Diaphoretics in *Bright's* disease, ii, 509
Diaphragm, its degeneration in bronchitis, i, 961
Diarrhœa, ii, 213
— choleraica, i, 232
— from lardaceous disease of bowel, ii, 215
— in enteric fever, i, 139, 174
— in phthisis, i, 1065
— — treatment of, i, 1113
— in smallpox, i, 208
— in uræmia, ii, 471
Diathesis, ii, 755, 770
Diathesis, gouty or arthritic, ii, 676
— rheumatic, ii, 694
— scrofulous, i, 1068
— tubercular, i, 1068
Dicrotism, ii, 4
Diet in constipation, ii, 213
— in convalescence from enteric fever, i, 174
— — from rheumatism, ii, 712
— in diabetes, ii, 587
— in dyspepsia, ii, 166
— in eczema, ii, 777
— during infancy, ii, 744
— in scurvy, ii, 656
Diffused atrophic paralysis, i, 495
Digitalis in acute alcoholism, i, 795
— in *Bright's* disease, ii, 508
— in cardiac dilatation, ii, 44
— in functional cardiac disorders, ii, 12
— in general paralysis, i, 687
— in mitral disease, ii, 85
Digitorum nodi, ii, 725
Dilatation of heart, ii, 31
— of stomach, ii, 157
Diphtheria, i, 256
— and croup, i, 264, 882
— and diphtheritic inflammation, i, 258
— in course of measles, i, 182
— of air-passages, i, 264, 267, 274
— laryngeal, i, 267, 274
— of nasal fossæ, i, 267, 274
— sine diphtheritâ, i, 258
— traumatic, i, 263; ii, 19
Diphtheritic colitis, ii, 226
— dysentery, ii, 226
— inflammation, i, 257, 261
— — of skin in smallpox, i, 207
— membrane of larynx in measles, i, 182
— — of pharynx in glanders, i, 361
— — of pharynx in scarlatina, i, 190
— — of pharynx in smallpox, i, 209
Diplegia facialis, i, 410
Diplococcus pneumoniæ, i, 997
Diplopia in locomotor ataxy, i, 529
Dipsomania, i, 798
Direction of cardiac murmurs, ii, 65
Discharging lesions, i, 609
— and destroying lesions, i, 617
Disease, meaning of the term, i, 2
— contagious, i, 11
— epizootic, i, 350
— functional, i, 3
— infective, i, 11
— miasmatic, i, 12
— miasmatic-contagious, i, 12
— specific, i, 98
— zymotic, i, 12
Diseases of the adrenals, ii, 594–603
— of the alimentary system, ii, 299–462
— of the blood, ii, 606–668
— of the blood-vessels, ii, 101–126
— of the bones, ii, 733–750
— of the brain, i, 547–692
— of the heart, ii, 1–100
— — congenital, ii, 86–89
— — in children, ii, 90 *et seq.*
— of the intestines, ii, 203–275
— of the joints, ii, 722–732

Diseases of the kidneys, ii, 406–562
— of the larynx, i, 866–916
— of the liver, ii, 331–405
— of the lungs, ii, 925–1120
— of the mouth, ii, 132–141
— of the nervous system, i, 880–865
— of the œsophagus, ii, 147–151
— of the pancreas, ii, 603
— of the peritoneum, ii, 305–330
— of the respiratory system, i, 866–1148
— of the salivary glands, ii, 135 *et seq.*
— of the skin, ii, 751 *et seq.*
— of the spinal cord, i, 427–547
— of the spleen, ii, 604
— of the stomach, ii, 152–202
— of the thyroid, i, 860; ii, 126
Disinfectants, comparative value of, i, 24
Disinfection, i, 23
Dislocations in epilepsy, i, 754
Dissecting aneurysm, ii, 113
Disseminated sclerosis, i, 541 *et seq.*
Distension of stomach, ii, 157
Distoma hæmatobium or bilharzia, ii, 304
Distribution of lardaceous disease, ii, 484
— local, of skin diseases, ii, 753, 759
Dittrich's plugs, i, 974
Diver's palsy, i, 453
Diverticulum ilei leading to strangulation, ii, 258
Dochmius duodenalis, ii, 293
Dothien-entérique, i, 134
Double quartan ague, i, 325
— tertian ague, i, 325
Dracunculus, ii, 304
Drink, *see* Alcoholism
— and gutta rosea, ii, 879
— and phthisis, i, 1091
— as a cause of renal cirrhosis, ii, 493
— in diabetes insipidus, ii, 409
— in diabetes mellitus, ii, 588
Dropsy in *Bright's* disease, ii, 456
— from chronic bronchitis, i, 961
— from cirrhosis of kidneys, ii, 500, 501
— in heart disease, ii, 60
— from lardaceous nephritis, ii, 490, 491
— from parenchymatous nephritis, ii, 479
— from phthisis, i, 1063
— in scarlatina, i, 191
— of cardiac origin in the course of *Bright's* disease, ii, 500
Drugs, action on colour of urine, ii, 415
— eruptions caused by, ii, 834
Drunkenness, diagnosis from apoplexy, i, 586
Dubini's chorea, i, 747
Duchenne's paralysis, i, 514
Dumb-bell crystals of oxalate of lime, ii, 422
Duodenum, ulcer of, ii, 180, 185
Dura mater, hæmatoma of, i, 676
— — inflammation, i, 634
— — thrombosis of sinuses, i, 674, 676
Dust-inhalation producing asthma, i, 1146
— producing phthisis, i, 1091
Dysæsthesia in hysteria, i, 804
— in paraplegia, i, 477
Dyscrasia, ii, 754
Dysentery, ii, 224
— and hepatic abscess, ii, 359
Dysentery, catarrhal form, ii, 224
— diphtheritic form, ii, 226
— endemic form, ii, 228
— sporadic form, ii, 228
Dysidrosis, ii, 857
Dyspepsia, ii, 159–166
— and gutta rosea, ii, 879
— bilious, ii, 332
— chronic, ii, 159
Dysphagia from aneurysm of aorta, ii, 106
— from bulbar paralysis, i, 513
— from laryngeal carcinoma, i, 913
— from œdematous laryngitis, i, 904
— from tubercular laryngitis, i, 896
— in hydrophobia, i, 369
— in hysteria, i, 805
Dyspnœa, i, 943; ii, 614
— from anæmia, ii, 613
— from aneurysm, ii, 106
— from paralysis of diaphragm, i, 944
— in Bright's disease, ii, 500
— in bronchitis, i, 952
— in cardiac disease, ii, 42, 61
— in diabetic coma, ii, 576
— in phthisis, i, 1065
— in pleurisy, i, 1026
— in pneumonia, i, 992
— from uræmia, ii, 470
— in acute pulmonary tuberculosis, i, 1116
— laryngeal, i, 922
— nervous, i, 943

E.

Ear-cough, i, 947
Ears, affections of, *see* Otorrhœa
— — in gout, ii, 674
— — in measles, i, 182
— — in scarlatina, i, 192
Eburnation of cartilages, ii, 724
Ecchondroses, ii, 725
Echinococcus, ii, 393; *see also* Hydatid
Echinorhynchus, ii, 304
Eclampsia, i, 750; *see also* Convulsions
— nutans, i, 701
Ecstasy, i, 818
Ecthyma, ii, 898
— syphiliticorum, ii, 898
Eczema, ii, 762
— and dermatitis, ii, 762
— and gout, ii, 770
— local varieties of, ii, 782
— marginatum, ii, 866; *see* Tinea marginata
— pustular, ii, 785
— treatment of, dietetic, ii, 777
— — internal, ii, 779
— — local, ii, 778
Electrical chorea, i, 747
— reactions, i, 399
— — in catalepsy, i, 816
— — in chorea, i, 729
— — in hemiplegia, i, 565
— — in lesions of nerve-trunks, i, 401, 403
— — in locomotor ataxy, i, 528
— — in paralysis—
— — — acute atrophic, i, 490

Electrical reactions in paralysis—
— — — agitans, i, 716
— — — bulbar, i, 510
— — — chronic diffused atrophic, i, 495
— — — diaphragmatic, i, 944
— — — facial, i, 408, *foot-note*
— — — peripheral, i, 405
— — — plumbic, i, 520
— — — pseudo-hypertrophic, i, 516
— — in paraplegia, i, 435
— — in hysterical paraplegia, i, 463
— — in spastic paraplegia, i, 481
— — in progressive muscular atrophy, i, 500
Electricity (faradic, galvanic, and static) as used in the treatment—
— of bedsore, ii, 448
— of bulbar paralysis, i, 512
— of exophthalmic goitre, ii, 131
— of hemiplegia, i, 596
— of hysteria, i, 814
— of infantile paralysis, i, 494
— of mercurialism, i, 523
— of spinal meningitis, i, 487
— of neuralgia, i, 394
— of osteo-arthritis, ii, 730
— of paralysis agitans, i, 720
— of acute atrophic paralysis, i, 494
— of chronic atrophic paralysis, i, 497
— of facial paralysis, i, 412
— of general paralysis of insane, i, 687
— of laryngeal paralysis, i, 873
— of peripheral paralysis, i, 406
— of pseudo-hypertrophic paralysis, i, 518
— of acute paraplegia, i, 448
— of spastic paraplegia, i, 483
— of progressive muscular atrophy, i, 506
— of sciatica, i, 390
— of spasmodic torticollis, i, 697
— of syncope, ii, 17
— of tetany, i, 709
— of writers' cramp, i, 707
Electrolysis for bedsore, ii, 448
— in aneurysm, ii, 111
— in hydatid disease, ii, 401
Elephantiasis, ii, 891
— Græcorum, ii, 918; *see also* Leprosy
— telangiectodes, ii, 928
Emaciation, *see* Wasting
— in chorea gravis, i, 731
— in fever, i, 38
— in phthisis, i, 1063
— from vomiting, ii, 172
Embolism, i, 62; ii, 124
— and aneurysm, ii, 104
— — diagnosis from hæmorrhage, i, 571
— — in chorea, i, 742
— — in enteric fever, i, 147
— pulmonary, ii, 128
— from endocarditis, ii, 57
— of cerebral arteries, i, 549
— of spleen, ii, 57
— of splenic arteries in enteric fever, i, 147
Emetics in croup, i, 889
— in plastic bronchitis, i, 984
Emphysema, atrophic, i, 966
Emphysema, hereditary, i, 967
— of lungs, i, 962–971
— interstitial or interlobular, i, 962
— senile, i, 966
— — in pertussis, i, 1038
— subcutaneous, in phthisis, i, 1074
— subpleural, in asthma, i, 1144
— — in diphtheria, i, 268
— — in pertussis, i, 1038
— theory of, i, 964
— vesicular or pulmonary, i, 962
— — friction-sound in, i, 971
Emprosthotonos in tetanus, i, 722
— in hydrophobia, i, 270
Empyema, i, 1020, 1029
— and abscess of brain, i, 625
— diagnosis from serous effusion, i, 1031
— in course of pneumothorax, i, 1043
Encephalitis, i, 633
Enchondroma, i, 78
Endemic hæmaturia, ii, 557
Endocarditis, acute, ii, 53
— chronic, ii, 55
— in chorea, i, 730, 742
— in diphtheria, i, 270
— in rheumatism, ii, 690
— in scarlatina, i, 192
— ulcerative, ii, 56
English cholera, i, 242
Enteric fever, i, 134 *et seq.*
— and general tuberculosis, i, 1119
— diagnosis of, i, 151
— — from ague, i, 152
— — from colitis, i, 154
— — from genito-urinary diseases, i, 154
— — from hepatic abscess, i, 154
— — from perinephric abscess, i, 154
— — from pneumonia, i, 153
— — from pyæmia, i, 153
— — from trichiniasis, i, 153
— — from tubercular meningitis, i, 153
— — from tuberculosis, i, 152, 153
— — from typhlitis, i, 154
— — from ulcerative endocarditis, i, 153
— differentiation from typhus, i, 135, 152
— hæmorrhage in, i, 145
— perforation of bowel in, i, 146
Enteritis, ii, 219
Entozoa, ii, 276
Ephelides, ii, 934
Ephemeral fever, i, 175
Epidemic meningitis, i, 653
— dysentery, ii, 228
— influenza, i, 1124
Epilation in favus, ii, 869
— in ringworm, ii, 865
Epilepsia major, i, 751
— minor, i, 750
Epilepsy, i, 749
— apoplectiform, i, 589
— diagnosis from apoplexy, i, 589
— — from hysteria, i, 768
— *Jacksonian*, i, 613, 753
— relation to paroxysmal insanity, i, 761
— simulation of, i, 762
— temperature in, i, 754
Epileptic centre, i, 755

Epileptic coma, i, 752
— dementia, i, 755
— hemiplegia, i, 754
— mania, i, 774
— vertigo, i, 767
Epileptiform convulsions, *see* Eclampsia
Epistaxis, i, 1132
— in *Bright's* disease, ii, 468
— in enteric fever, i, 138
— in hæmophilia, ii, 664
— in leuchæmia, ii, 622
— in migraine, i, 778
— in miliary tuberculosis, i, 1117
— in purpura, ii, 660
— in smallpox, i, 209
Epithelial reproduction, i, 57
Epithelioma, i, 88
— of skin, ii, 930
Epulis, i, 82
Equinia, *see* Glanders
Ergot in acute paraplegia, i, 448
— in hæmoptysis, i, 1112
Ergotism producing tetanilla, i, 712
Erroneous projection from strabismus, i, 415
Eructation, ii, 170
Eruptions or exanthems—
— in cholera, i, 230
— in dengue, i, 347
— from drugs, ii, 834
— in enteric fever, i, 138
— in epidemic meningitis, i, 656
— factitious, i, 803; ii, 942
— in foot-and-mouth disease, i, 365
— in glanders, i, 360, 361
— in measles, i, 180
— in migraine, i, 779
— in relapsing fever, i, 126
— in rubeola, i, 197
— in scarlatina, i, 187
— in smallpox, i, 201, 203
— in syphilis, i, 284, 301
— in typhus, i, 106
— in varicella, i, 217
Erysipelas, i, 246 *et seq.*
— faucial, i, 251
— laryngeal ulcers in, i, 898
— phlegmonous, i, 248
— surgical, i, 246, 248
Erythema, ii, 822
— and gutta rosea, ii, 878
— annulatum, ii, 826
— bullosum, ii, 828
— definition of, ii, 823
— due to drugs, ii, 835 *et seq.*
— exudativum, ii, 828
— fugax, ii, 828
— gyratum, ii, 828
— iris, ii, 828
— leve, ii, 460, 825
— marginatum, ii, 827
— multiforme, ii, 826
— nodosum, ii, 830
— vesicular, ii, 826
Essera Vogelii, i, 198
État pointillé of intestine, i, 156
Exalgine in facial neuralgia and tabes, i, 537
Exaltation of ideas—
— in disseminated sclerosis, i, 544
— in general paralysis of the insane, i, 683
Exanthems, *see* Eruptions
Excision of gall-stones, ii, 355
— of larynx, i, 914
— of pylorus, ii, 201
— of renal calculi, ii, 542
Exercise in dyspepsia, ii, 336
Exfoliative dermatitis, ii, 811
Exophthalmic goitre, ii, 126
Exudation, catarrhal, i, 51
— croupous, i, 52
— diphtheritic, i, 52
— plastic, i, 51
— purulent, i, 51
Eyeball, paralysis of, i, 413

F.

Facial paralysis, i, 407 *et seq.*
— spasm, i, 694
Factitious chromidrosis, ii, 856
— lesions of the skin, i, 803; ii, 942
— urticaria, ii, 935
Fæcal accumulation, ii, 254
Family or consanguineous diseases, i, 734, 1145
Famine fever, i, 124
Faradisation, *see* Electricity
Farcy, i, 359
Fastigium, i, 30
Fat-embolism in diabetes, ii, 577
Fatty degeneration in anæmia, ii, 614
— — of heart, ii, 51
— — of kidneys, ii, 478
— — of liver, ii, 388
— infiltration of heart, ii, 51
— overgrowth of heart, ii, 50
— stools, ii, 345
Fauces, affections of, ii, 138
Favus, ii, 868
Febricula, i, 175
Febrile albuminuria, ii, 453
— jaundice, ii, 341
— nephritis, ii, 443
Febris varioloea, i, 203
Fermentation, alcoholic, i, 14
— lactic, i, 14
— as a test for sugar in urine, ii, 567
Ferments in disease, i, 12
Ferrocyanide as a test for albumen, ii, 446
Fever, i, 27; *see also* Pyrexia
— hospitals, i, 120
— summary of symptoms, i, 41
Fevers, specific characters in tabular form, i, 102
Fibrillary tremors, i, 498
Fibrinous bronchitis, i, 980
Fibroid disease of heart, ii, 44
— phthisis, i, 1054
— pneumonia, i, 1007
Fibroma, i, 77, 607; ii, 53, 1013
— molluscum, ii, 925
— of skin, ii, 925
— recurrent, i, 80

Fibro-sarcoma, i, 81
— papillare, i, 909
Fibrous rheumatism, ii, 687, *foot-note*
Filaria sanguinis hominis, ii, 300
Fistula in phthisis, i, 1080
Flatulence in hysteria, i, 828
— in cardiac disease, ii, 86
— in dyspepsia, ii, 165
Flexibilitas cerea, i, 816
Floating kidney, ii, 560
Fluctuation in ascites, ii, 323
"Fluid vein" of anæmic murmurs, ii, 613
— — of cardiac murmurs, ii, 66
— — of respiratory murmur, i, 936
Follicles, solitary, in enteric fever, i, 155
Food, *see* Diet
Foreign bodies in the larynx, i, 915
Formication in paraplegia, i, 430
Foot-and-mouth disease in cattle, i, 363
— in man, i, 364
Fragilitas crinium, ii, 874
— ossium, ii, 742
Framboesia, ii, 922
Freckles, ii, 934
Frémissement cataire, ii, 72
Fremitus, hydatid, ii, 895
— vocal, i, 938
Friction-sound in emphysema, i, 971
— in pericarditis, ii, 94
— in pleurisy, i, 1019
Friedreich's disease, i, 539
Front tap contraction, i, 434
Fungi, i, 14
— in pertussis, i, 1141
— in phthisical vomicæ, i, 1060
— in thrush, ii, 134
— of favus, ii, 868
— of ringworm, ii, 860
Fungus hæmatodes, i, 81
Furunculus, ii, 852; *see also* Boils
Fusible calculus, ii, 515

G.

Gait in general paralysis, i, 683
— in paralysis agitans, i, 715
— in paraplegia, i, 433
— in tabes dorsalis, i, 525
— in cases of cerebellar tumour, i, 610
Gall-stones, ii, 350
— obstructing intestine, ii, 253
Galvanic cautery in pharyngitis, ii, 144
Galvanism, *see* Electricity
Gangrene, i, 53
— in cholera, i, 233
— in diabetes, ii, 574
— in enteric fever, i, 148
— in smallpox, i, 210
— in typhus, i, 112
— of genitalia in measles, i, 182
— of lung, i, 1014
— of mouth, ii, 133
— — in typhus, i, 112
— of spleen in ague, i, 330
Gargling, ii, 143
Gas, its composition in pneumothorax, i, 1045
Gastralgia, ii, 166
— from vertebral disease, ii, 169
Gastric carcinoma, ii, 191
— catarrh, acute, ii, 152
— — chronic, ii, 159
— concretions, ii, 202
— crises, i, 529
— dilatation, acute, ii, 157
— — chronic, ii, 168, 200
— disorder in chronic alcoholism, ii, 161
— induration, ii, 202
— ulcer, ii, 178
— sarcoma, ii, 191
Gastritis, acute, ii, 152
— — chronic, ii, 159
— membranous, ii, 159
— phlegmonous, ii, 158
Gastro-colic fistula, ii, 197
Gastro-cutaneous fistula from cancer, ii, 190
— — from simple ulcer of stomach, ii, 185
Gastrodynia, ii, 168
Gastro-enteritis, i, 176
Gastro-intestinal anthrax, i, 355
Gastrostomy, ii, 151
General paralysis of the insane, ii, 680
Geographical distribution—
— of ague, i, 328, 333
— of anthrax, i, 350
— of Bright's disease, ii, 507
— of bronchitis, i, 977
— of calculi, ii, 516
— of cholera, i, 226, 242
— of dengue, i, 346
— of diabetes, ii, 586
— of diphtheria, i, 264
— of eczema, ii, 778
— of epidemic dysentery, ii, 228
— of epidemic meningitis, i, 653
— of elephantiasis, ii, 892
— of enteric fever, i, 136, 167
— of favus, ii, 869
— of gout, ii, 677
— of influenza, i, 1024
— of leprosy, ii, 919
— of lichen tropicus, ii, 795
— of measles, i, 178
— of osteo-arthritis, ii, 729
— of phthisis, i, 1106
— of plague, i, 231
— of relapsing fever, i, 125
— of rheumatism, ii, 693
— of rickets, ii, 741
— of rubeola, i, 196
— of smallpox, i, 199
— of sweating sickness, i, 225
— of syphilis, i, 277
— of typhus, i, 104, 117
— of yellow fever, i, 339, 342
Giant-cells, i, 80, 308
— in lupus, ii, 904
— in rhinoscleroma, ii, 917
— in sarcomata, i, 80
— in simple inflammation, i, 56
— in tubercle, i, 308
Gingivitis, ii, 133
Girdle-feeling in paraplegia, i, 414

Girdle-feeling in locomotor ataxy, i, 529
— in myelitis, i, 444
Glanders, i, 359
— acute, i, 360
— chronic, i, 361
Glioma, i, 78
— of brain, i, 601
— of cord, i, 451
Globulin in urine, ii, 445
Globus hystericus, i, 907
Glomerulo-nephritis, ii, 443
Glosso-labio-pharyngeal paralysis, i, 508
Glossy skin, i, 404
Glottis, œdema of, i, 903
— spasm of, in laryngismus stridulus, ii, 877
— — in spurious croup, i, 886
— — in tetanus, i, 720
Gluge's corpuscles, i, 440
Glycogen, ii, 579
Glycose in urine, tests for, ii, 564
Glycosuria, ii, 564
— theory of, ii, 580 *et seq.*
— without diabetes, ii, 570
Gmelin's test, ii, 838
Goa powder, ii, 864
Goitre compressing trachea, i, 917
— endemic, i, 860
— exophthalmic, ii, 126
Gonorrhœal synovitis, ii, 716
Gout, ii, 669–685
— and diabetes, ii, 585
— and eczema, ii, 770
— and insanity, i, 844
— and phthisis, i, 1105
— as a cause of renal cirrhosis, ii, 493
von Gräffe's symptom, ii, 127
Granular degeneration of heart, ii, 52
— kidney, ii, 479
— pharyngitis, ii, 142
Granulation-tumours, i, 87
Granuloma, i, 60
— fungoides, ii, 928
Graves's disease, ii, 126
Greenstick fractures in rickets, ii, 735
Green-sickness, ii, 635
Grinder's phthisis, i, 1096
Gruyère cheese—condition of cerebrum so called, i, 685, 688
Guaiacum test for blood in urine, ii, 518
Guarana in migraine, i, 785
Gummata, i, 290
— in congenital syphilis, i, 299, 303
— of brain, i, 598
— of heart, i, 46
— of larynx, i, 901
— of liver, ii, 374
— of lung, i, 1013
— of placenta, i, 292
— of spinal meninges, i, 486
Gums in phthisis, i, 1065
— in scurvy, ii, 652
— in stomatitis, ii, 133
Gurgling in enteric fever, i, 140
Gurjun oil in leprosy, ii, 922
Gutta rosea, i, 798; ii, 877
Gyration, i, 820

H.

Hæmatemesis, ii, 174
— causes of, ii, 176–7
Hæmatidrosis, ii, 857
Hæmatinuria, intermittent, ii, 434
Hæmatoma of dura mater, i, 676, 709
Hæmato-myelia, i, 465
Hæmato-thorax, i, 1032
Hæmaturia, ii, 517–521, 552
— endemic, ii, 557
— from acute nephritis, ii, 480
— from bilharzia, ii, 557
— from renal calculus, ii, 517
— from renal cancer, ii, 552
— from renal tuberculosis, ii, 548
— from tubercular pyelitis, ii, 548
— parasitic, ii, 557
— treatment of, ii, 521
Hæmochromometer, ii, 611
Hæmocytometer, ii, 611
Hæmoglobin, estimate of, in blood, i, 611
— in urine, ii, 431
Hæmoglobinometer, ii, 611
Hæmoglobinuria, ii, 431, 434
— infantile, ii, 434
— paroxysmal, ii, 434
Hæmophilia, ii, 664
Hæmoptysis—
— from mediastinal tumour, ii, 119
— in cancer of the lung, ii, 119
— in cirrhosis of lung, i, 1010
— in emphysema, i, 968
— in heart disease, ii, 62
— in miliary tuberculosis of lungs, i, 1116
— in phthisis, i, 1067
— in plastic bronchitis, i, 982
— in pneumonia, i, 992
— in syphilitic laryngitis, i, 902
— non-phthisical, i, 1076
— treatment of, i, 1112
— vicarious, i, 1068
Hæmorrhage—
— cerebral, i, 555, 578, 580
— in Bright's disease, ii, 468
— from cirrhosis of liver, ii, 372
— into cord, i, 449
— cutaneous, ii, 935
— from invagination of intestine, ii, 246
— in enteric fever, i, 145
— in leuchæmia, ii, 621
— meningeal, i, 580
— in remittent fever, i, 329
— renal, treatment of, ii, 521
— in scurvy, ii, 652
— of spinal cord, i, 454
— spinal meningeal, i, 485
— in yellow fever, i, 340, 341
Hæmorrhagic arthritis, ii, 666
— diathesis, ii, 664
— erosion, ii, 161
Hæmostatics, *see* Styptics
Hair, affections of, ii, 858
— collected in stomach, ii, 202
— in phthisis, i, 1063
Hallucinations in hysteria, i, 902
Hammer palsy, i, 707
Handicraft spasms, i, 701

Handwriting in general paralysis, i, 662
Harlequin fœtus, ii, 888
Haut mal, i, 751
Hay-asthma, i, 1146
Hay-fever, i, 1134
Headache from cerebral tumour, i, 602
— from abscess of brain, i, 629
— from dyspepsia, ii, 164
— from meningitis, i, 640
— sick, i, 777
— in syphilis, i, 284
— in uræmia, ii, 470
Health-resorts in phthisis, i, 1108
Heart, diseases of, ii, 1–91
— aneurysm of, ii, 49
— calcareous degeneration of, ii, 52
— congenital disease of, ii, 86
— dilatation of, ii, 31
— — in chronic bronchitis, i, 961
— — in cirrhosis of lung, i, 1011
— displacement in pleuritic effusion, i, 1023
— — in pneumothorax, i, 1049
— in exophthalmic goître, ii, 127
— fibrosis of, ii, 44
— fatty disease of, ii, 50 *et seq.*
— granular degeneration of, ii, 52
— hydatid of, ii, 400
— hypertrophy of, ii, 30
— — in Bright's disease, ii, 463
— palpitation of, ii, 11
— paralysis of, in tetanus, i, 725
— in pyrexia, i, 30
— rupture of, ii, 52
— valvular diseases of, ii, 53
Heat-apoplexy, i, 787
Heat-regulation in fever, i, 32
Heat-stroke, i, 36, 787
Hectic fever, i, 61
Heller's test for albumen in urine, ii, 446
Hemeralopia, ii, 653, *foot-note*
Hemianæsthesia, i, 804
— diagnosis of cause, i, 592
— in hemiplegia, i, 568
— in hysteria, i, 804
Hemicrania, i, 775
Hemichorea, i, 729
Hemilaryngoplegia, ii, 869
Hemiopia, i, 609
— in hemiplegia, i, 567
— lateral, i, 567
— in meningitis, i, 641
— in migraine, i, 776
Hemiopsia, i, 776
Hemiparaplegia, i, 468
Hemiplegia, i, 561
— alternate, i, 564
— association with aphasia, i, 570
— crossed, i, 564
— diagnosis of causes, i, 593
— from cortical lesions, i, 565
— from lesion of basal ganglia, i, 565
— from meningitis, i, 565, 643
— in insular sclerosis, i, 544
— treatment of, i, 596
— with early rigidity, i, 565
— with hemianæsthesia, i, 568
— with lateral hemiopia, i, 567
Hemiplegia with late rigidity, i, 568
Hemispasm, i, 613
Hemitritæus, or semitertian fever, i, 221
Hepatic abscess, ii, 358
— diagnosis from enteric fever, i, 154
— — pleuritic effusion, i, 1031
Hepatitis, ii, 358
— syphilitic, ii, 374
Hepatization of lung, i, 986
Hereditary syphilis, i, 297
Heredity in asthma, i, 1145
— in cancer, i, 94
— in chorea, i, 747
— in diabetes, ii, 585
— in disseminated sclerosis, i, 545
— in emphysema, i, 967
— in epilepsy, i, 759
— in general paralysis, i, 661
— in gout, ii, 676
— in hæmophilia, ii, 664
— in insanity, i, 828
— in leprosy, ii, 921
— in migraine, i, 781
— in molluscum fibrosum, ii, 926
— in neuroses generally, i, 772
— in phthisis, i, 1083, 1087
— in progressive muscular atrophy, i, 499
— in pseudo-hypertrophic paralysis, i, 517
— in rheumatism, ii, 694
— in tabes, i, 589
Hernia, internal, ii, 258
Herpes, ii, 826
— circinatus, ii, 826
— cretaceous, ii, 907, 915
— facialis, ii, 827
— — in epidemic meningitis, i, 656
— — in pneumonia, i, 992
— gestationis, ii, 829
— iris, ii, 828
— labialis, ii, 827
— of the pharynx, ii, 139
— of the tongue, ii, 123
— præputialis, ii, 827
— symptomaticus, i, 992
— zoster, ii, 826, 935
Heubner's disease of cerebral arteries, i, 551
Hiccough, ii, 173
— in uræmia, ii, 471
High tension pulse, ii, 465
Histrionic spasm, i, 694
Hobnail liver, ii, 367
Hodgkin's disease, i, 88; ii, 627
Horseshoe kidney, ii, 560
Humoral pathology, ii, 337, *foot-note*
Huntingdon's chorea, i, 747
Hutchinson's triad, i, 303, 304
Hyaline casts in urine, ii, 454
Hydatids, ii, 227, 403
— of brain, i, 600; ii, 404
— of heart, ii, 400, 404
— as a cause of jaundice, ii, 343
— of kidney, ii, 553
— of liver, ii, 392
— of lung, ii, 404
— of pelvis, ii, 404
— of spine, i, 404
— of spleen, ii, 404

Hydatids, suppurating, ii, 399
Hydradenoma, ii, 927
Hydrargyria, ii, 837
Hydroa, ii, 820
Hydrobilirubin, ii, 414
Hydrocephalic cry, i, 640
Hydrocephalus, chronic, i, 663
— acute, i, 635
— following epidemic meningitis, i, 658
— from tumour of brain, i, 621
— of adults, i, 672
— of childhood, i, 639
— spurious, i, 645
Hydromyelus, i, 504
Hydronephrosis, ii, 529
Hydropericardium, ii, 98
Hydrophobia, i, 368
— spurious, i, 376
Hydropneumopericardium, ii, 94
Hydropneumothorax, i, 1045
Hydrothorax, i, 1020
Hyperæmia of cord, i, 453
— of skin, ii, 756
Hyperæsthesia in epidemic meningitis, i, 659
— in hysteria, i, 804
— in leprosy, ii, 920
— in neuralgia, i, 385
— in rickets, ii, 739
Hyperæsthetic zone in hemiparaplegia, i, 470
Hyperalgesia in hysteria, i, 804
Hyperidrosis, ii, 855
Hyperpyrexia, i, 31
— from heat-stroke, i, 789
— in chorea, i, 730, *foot-note*
— in erysipelas, i, 250
— in hysteria, i, 813
— paradoxical, i, 31
— in rheumatism, ii, 697, 710
— in scarlatina, i, 190
— in smallpox, i, 208
— in tetanus, i, 721
Hyperresonance, i, 929
Hypertrophic affections of the skin, ii, 876
Hypertrophy of brain, i, 691
— of heart, ii, 30
— — from aortic incompetence, ii, 70
— — from *Bright's* disease, ii, 463, 491
— — affecting right side only, ii, 43
— — from over-exertion, ii, 39
— — in youth, ii, 39
— of liver, ii, 373, 388
— of lungs, i, 971
— of skin and chronic dermatitis, ii, 877
Hypnotism, i, 815, *foot-note*
Hypochondriasis, i, 823
— distinction from hysteria, i, 823
— — from insanity, i, 824
— with melancholia, i, 834
Hypodermic syringe in diagnosis, i, 1037
Hypoplasia of aorta in anæmia, ii, 636
— of blood-vessels in hæmophilia, ii, 665
Hyposulphites for sarcinous vomiting, ii, 200
Hysteria, i, 801
— from cerebral tumour, i, 649
— diagnosis from epilepsy, i, 812
— epileptiform, i, 809
— in boys, i, 822
Hysteria, mental condition in, i, 803
— relation to neuroses, i, 804 *et seq.*
Hysterical contracture, i, 806
— cough, i, 948
— fit, i, 808
— paraplegia, i, 462
Hystero-epilepsy, i, 821

I.

Ichthyosis, ii, 885
— congenita, ii, 887
— hystrix, ii, 885
— linguæ, *see* Leucoplasia
Icterus, ii, 336, *see* also Jaundice
— gravis, ii, 343, 377
— neonatorum, ii, 344
— simplex, ii, 339
Idiocy, i, 855
— congenital, i, 856
— epileptic, i, 857
— from deprivation of senses, i, 857
— hydrocephalic, i, 857
— traumatic, i, 857
Idiopathic anæmia of Addison, ii, 617, 640
Idiosyncrasy, i, 7
Ileus, *see* Intestinal obstruction
Illusions in chronic dementia, i, 838
— in mania, i, 830
Imbecility, i, 855
Imitation, a cause of chorea, i, 740
— a cause of hysteria, i, 811
Impaction of bowel, ii, 253
Impetigo, ii, 785
— contagiosa, ii, 787
— rodens, ii, 907
Impotence in diabetes, ii, 571
— in tabes, ii, 527
Incontinence of fæces in paraplegia, i, 438
Incoordination in tabes, i, 525
Incubation, i, 17
— of ague, i, 323
— of cholera, i, 227
— of diphtheria, i, 264
— of dysentery, ii, 232
— of enteric fever, i, 136
— of erysipelas, i, 249
— of foot-and-mouth disease, i, 364
— of hydrophobia, i, 368
— of influenza, i, 1125
— of mumps, ii, 137
— of pertussis, i, 1136
— of plague, i, 221
— of relapsing fever, i, 125
— of rubeola, i, 196
— of scarlatina, i, 186
— of smallpox, i, 200
— of syphilis, i, 279
— of tubercle, i, 315
— of typhus, i, 104
— of vaccinia, i, 213
— of varicella, i, 216
— of whooping-cough, i, 1136
— of yellow fever, i, 339
Indican in urine, ii, 414
Indigo calculus, ii, 514
Infantile convulsions, i, 711

Infantile hæmoglobinuria, ii, 434
— paralysis, i, 489 *et seq.*
— scurvy, ii, 659
Infarctus, ii, 124; *see also* Embolism
— a cause of fibroid disease of heart, ii, 47
— of lung in leuchæmia, ii, 623
Infecting chancre, i, 279
Infection, i, 11; *see also* Contagion
— prevention of, i, 19
— process of, i, 17, 22
— protection from, by former attacks, i, 19
Infective diseases, i, 11, 102
Inflammation, ætiology of, i, 59
— cardinal symptoms of, i, 43, 48
— catarrhal, i, 51
— chronic, i, 60
— contagious, i, 15
— croupous, i, 52
— diphtheritic, i, 52, 257
— erysipelatous, i, 246, 249
— gangrenous, i, 53
— infective, i, 15
— membranous, i, 51, 159, 182, 190, 205, 208, 363
— — from traumatic causes, i, 263; ii, 19
— microscopical appearances in frogs, i, 44
— — in warm-blooded animals, i, 47
— of cartilage, i, 58
— of mucous membranes, i, 58
— of serous membranes, i, 58
— of skin, i, 57
— of viscera, i, 59
— a physiological process, i, 46
— purulent, i, 51
— putrid, i, 53
— serous, i, 51
Inflammatory heat, i, 49
— œdema of lungs, i, 1002
— pain, i, 49
— redness, i, 48
— swelling, i, 48
Inflation of bowel for intussusception, ii, 251
— as a means of diagnosis, ii, 265
Influenza, sporadic, i, 1123
— epidemic, i, 1124
Infusion in diabetic coma, ii, 592
Ingravescent apoplexy, i, 582
Inhalation in acute laryngitis, i, 891
— in bronchitis, i, 878
— in phthisis, i, 1113
Inheritance, *see* Heredity
Injury to the head, and abscess, i, 626
Inoculation, i, 212
— for lupus, ii, 913
— tubercle, ii, 912
Inosite in urine, ii, 409
Insanity, i, 826 *et seq.*
— ætiology of, i, 828
— alcoholic, i, 842
— certificates of, i, 848
— delusional, i, 837
— forms and varieties of, i, 826
— gouty, i, 844
— medico-legal relations of, i, 846
— moral, i, 843
— paralytic, i, 845
— paroxysmal, i, 817
Insanity, puerperal, i, 840
— relation to hypochondriasis, i, 825
— statistics of, i, 827
— syphilitic, i, 844
— from tumour of brain, i, 604
Insolatio, i, 891
Insomnia in acute alcoholism, i, 791
— in acute mania, i, 831
— in cardiac disease, ii, 62
— in delirious mania, i, 830
— in pneumonia, i, 994
— in typhus fever, i, 122
Inspection of thorax, i, 941
— in emphysema, i, 969
— in phthisis, 1071, 1073, 1090
— in thoracic aneurysm, ii, 105
Insular sclerosis, i, 541
Intercostal neuralgia, i, 949
Intermittent fever, i, 322
— pulse, ii, 8
Interstitial keratitis, i, 303
Intertrigo, ii, 767
Interval in ague, i, 325
Intestinal casts, ii, 242
— obstruction, ii, 244–275
— — from gall-stones, ii, 356
— ulceration in dysentery, ii, 241
— — in enterica, i, 155
— — in phthisis, ii, 211
— worms, ii, 276–304
Intestine, diseases of, ii, 203 *et seq.*
Intra-cardiac thrombi, ii, 49
Intra-thoracic growths, ii, 115
Intussusception or Invagination, ii, 244
Invasion of acute tuberculosis, i, 1114
— of ague, i, 323
— of cholera, i, 227
— of dengue, i, 346
— of diphtheria, i, 265
— of enteric fever, i, 136
— of erysipelas, i, 250
— of influenza, i, 1125
— of measles, i, 179
— of plague, i, 221
— of pneumonia, i, 991
— of relapsing fever, i, 125
— of rubeola, i, 197
— of scarlatina, i, 187
— of syphilis, i, 283
— of typhus, i, 104
— of varicella, i, 216
— of variola, i, 200
— of yellow fever, i, 339
Iodide of potass a cause of purpura, ii, 660
— effect on urine, ii, 415
— in aneurysm, ii, 110
— in syphilis, i, 297, 304
— in paraplegia from vertebral caries, i, 479
Iodide rash, ii, 834
Iodine in treatment of osteo-arthritis, ii, 721
— as a test for lardacein, ii, 483
— in treatment of exophthalmic goitre, ii, 131
— in treatment of vomiting, ii, 173
Ipecacuanha in asthma, i, 1148
— in bronchitis, ii, 979
— in dysentery, ii, 238
Iris or herpes iris, ii, 828

Iritis, i, 288, 304
Iron in anæmia, ii, 638
— in *Bright's* disease, ii, 510
— in cardiac disease, ii, 84
— in chlorosis, ii, 638
— in chorea, i, 745
— in convalescence from rheumatism, ii, 713
— in diphtheria, i, 272, 273, 275
— in eczema, ii, 779
— in erysipelas, i, 254
— in erythema nodosum, ii, 834
— in phthisis, i, 1111
— presence in the liver in anæmia, ii, 615
Irregular pulse, ii, 9
Irritable heart, ii, 7
Irritative fever, i, 61

J.

Jacksonian epilepsy, i, 613, 753
Jaundice, ii, 336
— from acute yellow atrophy of liver, ii, 375
— from cardiac disease, ii, 78
— from cancer of biliary ducts, ii, 388
— — of liver, ii, 386
— from cirrhosis of liver, ii, 343
— from congenital occlusion of duct, ii, 344
— epidemic, ii, 341
— febrile, ii, 342
— from gall-stones, ii, 343
— in miliary tuberculosis of liver, ii, 348
— in pneumonia, i, 993
— in pyæmia, ii, 342
— from pylephlebitis, ii, 342
— in relapsing fever, i, 126
— from stricture of bile-duct, ii, 344
— in typhus, i, 112
— in yellow fever, i, 340, 344
— and xanthelasma, ii, 834
Jejunostomy, ii, 201
Jugular veins, pulsation of, ii, 78
Jungle fever, i, 828
Juvenile ataxia, *see Friedreich's* disease

K.

Kakké, i, 426
Kaposi's disease, ii, 929
Keloid of *Addison*, ii, 888; *see* Sclerodermia
— of *Alibert*, ii, 888, 922; *see also* Cheloid
Keratitis, interstitial, i, 303
— ulcerative, 210; ii, 656
Keratolysis, ii,
Keratosis, ii, 796
— diffusa intra-uterina, ii, 888
— linguæ, ii, 883
— pilaris, ii, 796
Kerion, ii, 864
Kidney, cancer of, ii, 549
— cirrhosis of, ii, 492
— cystic disease of, ii, 504
— floating, ii, 562
— in heart disease, ii, 60
— hydatid of, ii, 553
Kidney, hypertrophy of, ii, 528
— lardaceous, ii, 487
— large red, ii, 477
— large white, ii, 478
— malformations of, ii, 560
— moveable, ii, 562
— sarcoma of, ii, 550
— small red, ii, 493
— small white, ii, 479
— tubercular, ii, 544
— waxy, ii, 487
Kleptomania, i, 844
Klopfversuch, ii, 16
Knee-jerk, i, 434
— in diabetes, ii, 571
— in general paralysis, i, 663
— in insular sclerosis, i, 544
— in locomotor ataxy, i, 587
— in spastic paraplegia, i, 489
— in tabes, i, 528
Kousso as a vermifuge, ii, 445
Kyestine in urine, ii, 431

L.

Labio-glosso-laryngeal paralysis, i, 506
Labyrinthine vertigo, i, 768
Lactic acid and acute rheumatism, ii, 696
— — in tr. of laryngeal phthisis, i, 879
— fermentation, i, 13
Lac virginum, ii, 842
Lancing the gums for laryngismus, i, 881
Landry's paralysis, i, 456
Lanoline, ii, 776
Laparotomy, *see* Abdominal section, ii, 252
Lardaceous degeneration, ii, 482–487
— of adrenals, ii, 602
— from congenital syphilis, ii, 434
— distribution of, ii, 484
— of intestine, ii, 217
— of kidneys, ii, 444, 487
— of liver, ii, 391
— in phthisis, i, 1080
— of spleen, ii, 491
— from suppuration, ii, 484
— from syphilis, ii, 484
Laryngeal insufflation, i, 899
— lupus, i, 900
— paralysis, i, 869
— perichondritis, i, 906
— phthisis, i, 894
— syphilis, i, 900
— tumours, i, 909
Laryngismus stridulus, i, 877
— — relation to rickets, i, 879
Laryngitis, i, 881, 884
— acute catarrhal, i, 890
— chronic catarrhal, i, 891
— membranous, in adults, i, 890
— œdematous, i, 903
— phlegmonous, i, 903
— stridulous, i, 877
— syphilitic, i, 900
— tuberculous, i, 894
Laryngoplegia, i, 869
Laryngoscope, i, 866

Laryngoscopic appearances—
— in carcinoma of larynx, i, 913
— in chronic laryngitis, i, 892
— in laryngeal malformations, i, 915
— in paralysis of abductor muscles, i, 871
— — of adductor muscles, i, 874
— — of recurrent laryngeal nerve, i, 869
— in tracheal obstruction, i, 921
— in tubercular disease of larynx, i, 894
Larynx, angioma of, i, 911
— carcinoma of, i, 912
— diphtheria of, i, 267, 274
— diseases of, i, 866–916
— enteric ulceration of, i, 158
— extirpation of, i, 914
— fibroma of, i, 910
— foreign bodies within, i, 915
— in glanders, i, 361
— lipoma of, i, 911
— lupus of, i, 900
— malformations of, i, 914
— mucous cyst of, i, 910
— myxoma of, i, 911
— œdema of, in *Bright's* disease, i, 903
— — in erysipelas, i, 253
— operations on, i, 911
— papilloma of, i, 909
— perichondritis of, i, 906
— sarcoma of, i, 912
— spasm of, i, 877
— syphilitic ulceration of, i, 900
— tubercular ulceration of, i, 894
Latent cancer of stomach, ii, 194
— cirrhosis, ii, 369
— diabetes, ii, 573
— gastric ulcer, ii, 186
Lateral curvature of spine, i, 471
Lateritious deposits, ii, 417
Lead-colic, ii, 205; *see also* Plumbism
Lead in treatment of dermatitis, ii, 774
— paralysis from, i, 505, 519
Lead-palsy, i, 519
Leeches, in treatment of meningitis, i, 661
— — of acute nephritis, ii, 508
— — of pericarditis, ii, 99
— — of peritonitis, ii, 315
— — of pleurisy, i, 1036
Legal capacity of the insane, i, 846
Lemon-juice in rheumatism, ii, 701
Lentigo, ii, 934
Leontiasis, ii, 918
Lepra vera, or leprosy, ii, 918
Leptomeningitis infantum, i, 635
Letters, pronunciation of, in bulbar paralysis, i, 508
— — in insular sclerosis, i, 543
Leuchæmia, i, 87; ii, 618
— lymphatica, ii, 626
— myelogenica, ii, 623
— splenica, ii, 619
Leuchæmic enlargement of liver, ii, 388
Leucin in urine, ii, 377, 380
Leucocythæmia, *see* Leuchæmia
Leucodermia, ii, 932
Leucoplakia, ii, 883
Lichen, ii, 793
— agrius, ii, 796
Lichen circumscriptus, ii, 794
— pilaris, ii, 796
— planus, ii, 798
— ruber, ii, 798
— scrofulosus, ii, 797
— syphiliticus, i, 286; ii, 898
— tropicus, ii, 795
— urticatus, ii, 796
Lientery, ii, 198
Life insurance, i, 1090, *foot-note*
— — and asthma, i, 1148
— — and phthisis, i, 1087
Lightning-pains, i, 528
Lime-juice in scurvy, ii, 658
Linear atrophy of the skin, ii, 891
Lipæmia in diabetes, ii, 577
Lipoma, i, 78
Lipomatosis musculorum luxurians, i, 514
Lithates, *see* Urates
Lithæmia, ii, 383
— and diabetes, ii, 572
Lithiasis, ii, 418
Lithic acid, ii, 416; *see also* Uric acid
Liver, abscess of, ii, 558
— acute yellow atrophy of, ii, 875
— cancer of, ii, 384
— capsulitis of, ii, 373
— cirrhosis of, ii, 367
— in diabetes, ii, 578
— fatty, ii, 388
— hydatid tumours of, ii, 392
— hypertrophy of, ii, 374
— in leuchæmia, ii, 388
— nutmeg, ii, 60
— — in chronic bronchitis, i, 961
— pulsation of, ii, 78
— in rickets, ii, 738
— simple atrophy of, ii, 374
— syphilitic disease of, ii, 374
— tuberculosis of, ii, 392
— vomica in, ii, 392
— in yellow fever, i, 341
Localisation of cerebral lesions, i, 609
— of cutaneous diseases, ii, 759
— of eczema, ii, 766
— of phthisis, i, 1055
— of cancer of stomach, ii, 190
Lockjaw, i, 720
Lock-spasm, i, 699
Locomotor ataxia, i, 524; *see also* Tabes
— diagnosis from general paralysis, i, 687
— hereditary form of, i, 535
— in disseminated sclerosis, i, 534
— relation to paraplegia, i, 527
Longitudinal sclerosis, i, 488
Lordosis of spine, ii, 736
Lotura carnea, ii, 397
Lumbago, ii, 714
— from renal calculus, ii, 517
Lumbar cushion, ii, 480
Lung, atelectasis of, i, 958
— brown induration of, ii, 60
— cancer of, ii, 120
— carnification of, i, 1021
— cirrhosis of, ii, 1007
— collapse of, i, 958
— emphysema of, interstitial, i, 962

Lung, emphysema of, vesicular, i, 962
— gangrene of, i, 113, 1014
— infarctus of, ii, 60
— inflammation of, i, 985
— iron-grey induration of, ii, 1007
— local compression of, i, 1021
— senile atrophy of, i, 966
— splenised, ii, 60
— syphilitic disease of, i, 1013
— tuberculosis of, i, 309, 1014
— tumours of, ii, 121
Lupus, ii, 902
— erythematosus, ii, 914
— — disseminatus, ii, 916
— lymphaticus, *see* Lymphangioma
— of larynx, i, 900
— of mucous membranes, ii, 904
— sebaceous, ii, 914, *foot-note*
Lymph, plastic, i, 51
Lymphadenoma, *see* Lymphoma
Lymphangioma, ii, 928
Lymph-glands (lympharia) in dengue, i, 347
— in cancer, i, 73
— in enteric fever, i, 154, 157
— in erysipelas, i, 249, 251
— in Hodgkin's disease, ii, 627
— in hydrophobia, i, 369
— in leuchæmia, ii, 622
— in rubeola, i, 197
— in scarlatina, i, 187
— in syphilis, i, 283
— tuberculosis of, ii, 633
Lymph-scrotum, ii, 556
Lymphoma, i, 87, 307; ii, 627
Lympho-sarcoma, i, 88; ii, 635
— of intestine, ii, 217
Lysis, i, 30
Lyssa, i, 368

M.

Macula, ii, 758
Maculæ hepaticæ, ii, 869
— syphiliticæ, ii, 899
Main en griffe in hemiplegia, i, 565
— in paralysis of ulnar nerve, i, 398
— in progressive muscular atrophy, i, 497
Malacosteon, ii, 746
Malaria, i, 322, 328, 331
— and cirrhosis, ii, 369
— and phthisis, i, 1105
— effect on liver, ii, 391
— relation to yellow fever, i, 343
Malarial cachexia, i, 329, 338
Male fern as an anthelmintic, ii, 286
Malformation of heart, ii, 87
— of kidney, ii, 560
— of larynx, i, 914
— of skull, i, 859; ii, 737
Malignancy, *Virchow's* characters of, i, 72
Malignant endocarditis, ii, 56
— disease, *see* Cancer
— pustule, i, 354
Malingering in hysteria, i, 802
Malleus, *see* Glanders
Mamma, inflammation in mumps, ii, 137
Mania, acute, i, 831
— — delirious, i, 829
— in chorea, i, 731
— in chronic alcoholism, i, 842
— in epilepsy, i, 754
— in general paralysis of insane, i, 683
— in hydrophobia, i, 371
— in meningitis, i, 651
— paroxysmal, i, 773
Marasmus, i, 505
— in diabetes, ii, 571
— following enteric fever, i, 149
— in hysteria, i, 811
— in phthisis, i, 1063
— in pyrexia, i, 38
Marriage, relation of syphilis to, i, 291
Marrow in leuchæmia, ii, 623
— in *Hodgkin's* disease, ii, 632
Masked ague, i, 328
Massage in marasmus, i, 505
— in peripheral paralysis, i, 407
— in pseudo-hypertrophic paralysis, i, 518
Measles, i, 177 *et seq.*
— benign, i, 181
— broncho-pneumonia in, i, 1003
— hæmorrhagic, i, 181
— and whooping-cough, i, 1140
Measly meat, ii, 281
Measurement of chest, i, 942
— of heart, ii, 33
Mediastinal abscess, i, 918
— tumours, i, 918; ii, 115–122
Medicinal eruptions, ii, 834 *et seq.*
Medico-legal relations of insanity, i, 846
Megalocytes in anæmia, ii, 612
Megalomania, i, 682
Megrim, i, 775
Melæna, ii, 175
Melanæmia, i, 330
— nature of pigment, i, 330
— relation to hæmaturia, i, 331
Melancholia, i, 833
— after chorea, i, 733
— distinction from hypochondriasis, i, 825
— in disseminated sclerosis, i, 544
— with stupor, i, 836
Melanodermia, ii, 595, 934
— in *Graves'* disease, ii, 128
Melanoma of pia mater, i, 600
Melanotic sarcoma, i, 82
Melanuria, ii, 549
Melasma suprarenale, ii, 595
— without *Addison's* disease, ii, 599
Meliceris, ii, 846
Memory, loss of, in chronic dementia, i, 839
— — in general paralysis, i, 684
Ménière's disease, i, 768, 769
Meningeal apoplexy, i, 557
— hæmorrhage, cerebral, i, 679, 746
— — spinal, i, 485
Meninges, cerebral—
— — hæmorrhage into, i, 555
— — tumours of, i, 597
— spinal—
— — gumma of, i, 474
— — hæmorrhage in, i, 483
— — tumour of, i, 473

Meningitis, acute non-tubercular, i, 647
— associated with pneumonia, i, 649
— of base, i, 638
— in *Bright's* disease, ii, 460
— epidemic cerebro-spinal, i, 653
— a cause of hydrocephalus, i, 658
— idiopathic, i, 650
— metastatic, i, 649
— spinal, i, 486
— tubercular, i, 635–646
— — diagnosis from enteric fever, i, 153
— — in phthisis, i, 1079
Meningo-ependymitis, i, 672
Mentagra, *see* Sycosis
Mental disorders, *see* Insanity
Mercurial eruptions, ii, 837
— tremor, i, 521
Mercury, externally for eczema, ii, 775
— — for impetigo capitis, ii, 786
— — for ringworm, ii, 862
— in biliary colic, ii, 354
— in *Bright's* disease, ii, 509
— in hepatic dyspepsia, ii, 164
— in hydrocephalus, i, 674
— in mitral disease, ii, 85
— in syphilis, i, 294, 304
— in tabes, i, 538
Metallic echo, i, 1048
— tinkling in pneumothorax, i, 1047
— in phthisis, i, 1074.
Metallo-thérapie, i, 814
Metamorphosing breath-sound, i, 1074
Metastasis, i, 67
— in mumps, ii, 137
Methyl violet as a test for lardacein, ii, 488
Miasmatic-contagious diseases, i, 12
Microbes, Microphytes, or Microzymes, i, 12, 13, 14, 18; *see also* Bacteria, Bacilli, Micrococci, Spirilla, Sarcinæ
— of contagia, i, 22
— of miasmata, i, 21
— physiological action of, i, 18
— pleomorphism of, i, 15
Micrococci, i, 14
— in diphtheria, i, 261
— in erysipelas, i, 248
— in pneumonia, i, 997
— in purpura, ii, 661
— of ammoniacal urine, 428
Microcytes in anæmia, ii, 612
Microsporon furfurans, ii, 870
— mentagraphytes, ii, 851
Micturition in paraplegia, i, 436
Middeldorpff's harpoon, i, 498
Migraine, i, 775
— and ague, i, 781
Miliaria, ii, 857
— in measles, i, 180
— in rheumatism, ii, 690
Miliary aneurysms, i, 559
— fever, ii, 690, *foot-note*
— sclerosis, i, 685
— tubercles, i, 306; ii, 262
— tuberculosis of lungs, i, 1114
Milium, ii, 845
Milk-diet in acute gastric catarrh, ii, 156
— in *Bright's* disease, ii, 510
Milk-diet in diabetes, ii, 588
— in enteric fever, i, 170
— in gastric ulcer, ii, 189
— in rheumatism, ii, 712
Milk as a vehicle of—
— enteric contagion, i, 165
— foot-and-mouth disease, i, 364
— scarlatinal contagion, i, 185
— of perlsucht, a source of tubercle, i, 316
Millar's asthma, *see* Laryngismus stridulus
Mineral acids in treatment of dyspepsia, ii, 165
Mineral waters in chronic bronchitis, i, 980
— — in diabetes, ii, 589
— — in dyspepsia, ii, 164, 335
— — in eczema, ii, 777
— — in gout, ii, 685
— — in jaundice ii, 348
— — in lithæmia, ii, 335
— — in pityriasis rubra, ii, 814
Miners' sarcoma of lungs, ii, 120
Mitral disease, ii, 71
— — and phthisis, i, 1105
— incompetence, ii, 75
— murmurs in *Bright's* disease, ii, 76
— stenosis, ii, 71
— regurgitation, mechanism of, ii, 75
Mixed chancre, i, 281
Molimina, i, 581
Mollities ossium, ii, 746
Molluscum contagiosum, i, 89; ii, 847
— corpuscles, ii, 848
— fibrosum, ii, 925
— meaning of word, ii, 847, *foot-note*
— sebaceum, i, 847
Moore's test for glycose, ii, 566
Moral insanity, i, 843
Morbilli, i, 178
— sine catarrho, i, 181
— sine morbillis, i, 181
Morbus maculosus, *see* Purpura
Morphœa, ii, 888
Morphia, in treatment of diabetes, ii, 591
— eruption from, ii, 836
— in treatment of heart disease, ii, 85
— injection in neuralgia, ii, 394
— — in spasmodic torticollis, i, 698
— insufflations in laryngitis, i, 891
— local use for laryngeal phthisis, i, 899
Morphia mania, i, 800
Mortification, *see* Gangrene
Mouth, diseases of, ii, 132–141
Moveable kidney, ii, 705
Movement-spasms, i, 699
Mucous patches, i, 286; ii, 884
Multilocular hydatid, ii, 548
Mumps, ii, 136
Murmur, aneurysmal, ii, 105
— respiratory or pulmonary, i, 933
Murmurs, cardiac, in chorea, i, 730
— — in cirrhosis of lung, i, 1011
— — in pleuritic effusion, i, 1031
— of anæmia, ii, 613
— of valvular disease, ii, 64, 67, 71, 75, 77, 79, 80
— vascular, over fontanelle, i, 677

Murmurs, theory of, ii, 66
Murrain, i, 363
Muscle of heart, diseases of, ii, 29
Muscles in acute atrophic paralysis, i, 490
— in cervical pachymeningitis, i, 503
— in chronic atrophic paralysis, i, 495
— in disseminated sclerosis, i, 543
— in general paralysis of the insane, i, 683
— in lateral sclerosis, i, 502
— in locomotor ataxia, i, 525, 528
— in paraplegia, i, 431, 435
— in plumbism, i, 519
— in progressive muscular atrophy, i, 497
— in pseudo-hypertrophic paralysis, i, 515
— in sciatica, i, 390
Muscular contraction from percussion, i, 931
— contracture in hysteria, i, 806
— — in spasmodic torticollis, i, 695
— in-coördination, from alcoholism, i, 797
— — in locomotor ataxy, i, 526
— rheumatism, ii, 714
— rigidity from insular sclerosis, i, 543
— — from cerebral hæmorrhage, i, 563
— — from lateral sclerosis, i, 502
— spasm, after facial paralysis, i, 411
— tremors, in disseminated sclerosis, i, 543
— — from lead, i, 520
— — from mercury, i, 521
— — in paralysis agitans, i, 715
— — in suppression of urine, ii, 525
— — in typhus, i, 108
— — in uræmia, ii, 469
Myalgia, ii, 714
Mycosis fungoides, ii, 928
Mydriasis, *see* Dilated pupils
— in enteric fever, i, 141
Myelitis, i, 439 *et seq.*
— — in chorea, i, 734
— — in specific fevers, i, 443
— chronic, i, 441; *see also* Sclerosis
Myeloid cells, i, 80
Myocarditis, ii, 45, 46
— acute, ii, 50
— fibroid, ii, 44
— from coronary thrombosis, ii, 47
— rheumatic, ii, 46
— syphilitic, ii, 46
Myoma, i, 77
— cutis, ii, 926
Myopathic atrophy, i, 514
Myotatic movements, i, 434
Myosis, *see* Contracted pupils
— in typhus, i, 108
Myotonia congenita, *see Thomsen's* disease
Myxœdema, i, 863
Myxoma, i, 79

N.

Nævi, pigmentary, ii, 935
— vascular, ii, 927
Nails, eczema of, ii, 783
— in phthisis, i, 1065
— pemphigus affecting, ii, 816
— psoriasis of, ii, 806
Nails, tinea and favus of, ii, 867
Narcotic poisoning and apoplexy, i, 585
Nasal diphtheria, i, 267, 274
Nausea from drink, i, 797
— in meguim, i, 777
Necrosis after enteric fever, i, 148
— of laryngeal cartilages, i, 906
Nematoid worms, ii, 288
Nephralgia, ii, 521, 552
Nephrectomy, ii, 542, 549, 553
— for malignant disease, ii, 553
— of moveable kidney, ii, 562
Nephritis—
— cystic form of interstitial, ii, 504
— in fever, ii, 443
— interstitial, ii, 492
— lardaceous, ii, 487
— parenchymatous, ii, 443, 476
— scarlatinal, i, 191; ii, 476, 481, *foot-note*
— suppurative, ii, 538
— tubercular, ii, 544
Nephro-lithiasis, ii, 516
Nephro-lithotomy, ii, 542
Nephro-phthisis, ii, 544
Nephrorrhaphy, ii, 562
Nephrotomy, ii, 542
"Nerve-storms," i, 758
Nerve-stretching for neuralgia, i, 394
— for tabes, i, 538
Nerves, diseases of, i, 383–426
Nervous symptoms in fever, i, 41
Nettle-rash, *see* Urticaria
Neuralgia, i, 383 *et seq.*
— from chronic alcoholism, i, 797
— cervico-brachial, i, 389
— cervico-occipital, i, 389
— cordis, *see* Angina pectoris, ii, 18
— criteria of, i, 384, 385
— from malarial poisoning, i, 328
— from syphilis, i, 284
— of rachialgia, i, 464
— reflex, i, 383
— sciatic, i, 390
— trifacial, i, 386
— after zona, ii, 939
Neurasthenia spinalis, i, 462
Neuritis, peripheral, i, 422 *et seq.*
Neuroma cutis, ii, 926
Neuroses, i, 693–825
— of skin, ii, 941
— paroxysmal, i, 748
— relation of neuralgia to, i, 385
— spasmodic, i, 693
Neuro-retinitis, *see* Optic neuritis
Neurotic affections of the skin, ii, 936
New growths, i, 70
Night-blindness in scurvy, ii, 653
Nitric acid test for albumen in urine, ii, 446
Nitrite of amyl in angina pectoris, ii, 27
— — in epilepsy, i, 765
Nitro-glycerine in angina pectoris, ii, 27
— in *Bright's* disease, ii, 510
— in cardiac disease, ii, 86
Nitro-hydrochloric acid in jaundice, ii, 348
Nodes of inherited syphilis, i, 302
— of skin, ii, 758
— of tertiary syphilis, i, 290

Nodi digitorum, ii, 725
Nodule, ii, 758
— subcutaneous, in rheumatism, ii, 692
Noma after measles, i, 182
Nomenclature, i, 381
— of cardiac valves, ii, 79
— of cutaneous diseases, ii, 754
Non-infecting chancre, i, 280
Nosology, i, 3, 98
Nummular sputa of phthisis, i, 1066
Nutmeg liver, ii, 60
Nux vomica in dyspepsia, ii, 165
Nyctalopia, ii, 653
Nystagmus in disseminated sclerosis, i, 543
— in hereditary locomotor ataxy, i, 529

O.

Obesity, ii, 390
Obstruction of the bowels, ii, 244
— acute, ii, 268
— chronic, ii, 262
— of intestine by worms, ii, 290
Ocular lesions of insular sclerosis, i, 543
— in locomotor ataxy, i, 529
— in migraine, i, 776
Odour of breath in diabetes, ii, 573
— of favus, ii, 869
— of last stage in meningitis, i, 642
— of smallpox, i, 200
— of viscera in diabetes, ii, 577
Œdema of lungs, i, 1002; ii, 481, 499
— of brain, i, 554, 601
Œdematous laryngitis, i, 903
Œnomania, i, 798
Œsophageal sound, ii, 197
Œsophagus, diseases of, ii, 147–151
— carcinoma of, ii, 148
— dilatation of, ii, 147
— diphtheria of, i, 267
— papilloma of, ii, 147
— stricture of, ii, 147
Oidium albicans, ii, 134
Oil of male fern for tapeworm, ii, 286
— — for anchylostomum, ii, 295
Oinomania (Œnomania), i, 798
Oleate of mercury in treatment of ringworm, ii, 966
Oliguria, ii, 408
Onychomycosis, ii, 868
Ophthalmia with gonorrhœal arthritis, ii, 719
— in measles, i, 182
Ophthalmoplegia externa, i, 418
Opisthotonos, i, 722
Opium and morphia in neuralgia, i, 395
— eating, effects of, i, 800
— in treatment of acute alcoholism, i, 793
— in *Bright's* disease, ii, 499
— in cardiac disease, ii, 85
— in chronic bronchitis, i, 979
— in chronic intestinal obstruction, ii, 266
— in diabetes, ii, 591
— in meningitis, i, 661
— in peritonitis, ii, 315
— in rheumatism, ii, 702, *foot-note*
— in typhlitis, ii, 222
Opium, poisoning by, i, 585, 800
Optic chiasma, course of fibres in, i, 573
Optic disc—
— atrophy of, from cerebral tumour, i, 608
— — in disseminated sclerosis, i, 544
— — in hydrocephalus, i, 668
— — in locomotor ataxy, i, 529
— condition in epilepsy, i, 757
— choked, from cerebral tumour, i, 606, 620
— — in meningitis, i, 651
— inflammation of, *see* Optic neuritis
Optic neuritis from *Bright's* disease, ii, 609
— — from cerebral tumour, i, 605, 607
— — from meningitis, i, 641, 686, 693
— — in abscess of brain, i, 630
Optic tract, course of fibres in, i, 619
Orchitis from mumps, ii, 137
— syphilitic, ii, 607
— tubercular, ii, 547
Organic and functional disease, i, 4
Orthopnœa, i, 945
—in cardiac disease, ii, 42
— in pericardial effusion, ii, 96
Orthotonus in epidemic meningitis, i, 655
Osmidrosis, ii, 856
Osseous changes in osteo-arthritis, ii, 724
— in *Charcot's* disease of the joints, ii, 731
— in mollities ossium, ii, 746
— in rickets, ii, 734
— in syphilis, i, 290
— in congenital syphilis, i, 299, 303
Osteitis deformans, ii, 749
— — relation to cancer, i, 86
Osteoid disease, malignant, i, 78
Osteo-arthritis, ii, 722–731
Osteoma, i, 78
Osteomalacia, ii, 746
Otitis in measles, i, 182
— in epidemic meningitis, i, 656
— in scarlatina, i, 192
Otorrhœa and cerebral abscess, i, 624
Ova of bilharzia, ii, 557
Ovarian affections, ii, 607
— — in hysteria, i, 810
— cyst, diagnosis from ascites, ii, 325
— — — from hydronephrosis, ii, 533
Ovary, compression of, in hysteria, i, 810
Overcrowding and phthisis, i, 1085
Over-exertion and heart disease, ii, 31
Oxalate of lime calculus, ii, 512
— in urine, ii, 421
Oxyakoia (oxbacoa),
Oxyuris vemicularis, ii, 291
Ozæna, i, 431
— from glanders, i, 361, 362

P.

Pachydermia, ii, 891; *see* Elephantiasis
— laryngis, i, 894, 909
Pachymeningitis, cerebral, i, 634
— cranii hæmorrhagica, i, 676
— cervical hypertrophic, i, 486, 503
— in relapsing fever, i, 129
Pœcilo-cytosis, ii, 645
Paget's disease of the nipple, ii, 784

Palate, paralysis of, in diphtheria, i, 270
— — in facial palsy, i, 410
— — in tonsillitis, ii, 140, *foot-note*
— ulcer of, ii, 132
Palpation in ascites, ii, 325
— in diseases of the chest, i, 941
— in cases of abdominal disease, ii, 195
— in diseases of the heart, ii, 37
Palpitation of heart, ii, 11
— — in gastric disorder, ii, 159
— — at puberty, ii, 12
Pancreas, cancer of, ii, 603
— in diabetes, ii, 578, 603
— diseases of, ii, 603
— in heart disease, ii, 60
Papilloma, i, 883; ii, 89
— of œsophagus, ii, 147
— fungoides, ii, 928
Papule, ii, 756,
Paracentesis abdominis, ii, 323, 329
— for hepatic abscess, ii, 366
— for hydatid cyst, ii, 401
— for hydronephrosis, ii, 535
— for pericardial effusion, ii, 100
— for pleuritic effusion, i, 1037
— for pneumothorax, i, 1052
— thoracis, i, 1037
Paræsthesia, i, 404
— in paraplegia, i, 430
Paraglobulin in urine, ii, 445
Paralysis, acute ascending, i, 455
— acute atrophic spinal, of adults, i, 494
— — of infants, i, 489
— agitans, i, 715
— alcoholic, i, 458
— *Bell's*, i, 407 *et seq.*
— bulbar, acute, i, 512
— — chronic progressive, i, 506
— of bladder in paraplegia, i, 436, 469
— of brachial plexus, i, 396
— after chorea, i, 733
— chronic diffused atrophic, i, 495
— of circumflex nerve, i, 396
— "crutch," i, 396
— *Cruveilhier's*, i, 497
— of diaphragm, i, 944
— after diphtheria, i, 270, 275
— in disseminated sclerosis, i, 542
— *Duchenne's*, i, 513
— following epilepsy, i, 754
— of facial nerve, i, 407
— — double, i, 410
— of fifth nerve, i, 420
— of fourth nerve, i, 416
— general, of the insane, i, 680
— general spinal, i, 489
— hemiplegic, i, 548, 561
— hydrocephalic, i, 668
— of hypoglossal nerve, i, 419
— hysterical, i, 804
— infantile, i, 489
— labio-glosso-pharyngeal, i, 506
— *Landry's*, i, 455
— of laryngeal abductors, i, 871
— — adductors, i, 874
— in lateral sclerosis, i, 502
— from lead, i, 505, 519
Paralysis in locomotor ataxy, i, 524
— in meningitis, i, 651
— of median nerve, i, 398
— in migraine, i, 778
— of musculo-cutaneous nerve, i, 398
— of musculo-spiral nerve, i, 398
— of oculo-motor nerves, i, 413
— of olfactory nerve, i, 421
— peripheral, i, 406, 422
— of posterior thoracic nerve, i, 396
— in progressive muscular atrophy, i, 497
— pseudo-hypertrophic, i, 514
— of rectum in paraplegia, i, 438, 469
— of recurrent laryngeal nerve, i, 869
— reflex, i, 396
— of sixth nerve, i, 413
— of third nerve, i, 416
— of ulnar nerve, i, 398
— of vocal cords, i, 877
Parangi, ii, 922
Paraplegia, i, 428 *et seq.*
— from alcohol, i, 458
— from anæmia of spinal cord, i, 451
— from compression of cord, i, 470
— from concussion of cord, i, 455
— in disseminated sclerosis, i, 542
— dolorosa, i, 476
— from hæmorrhage into cord, i, 449
— from hyperæmia of cord, i, 453
— hysterical, i, 462
— from chronic spinal meningitis, i, 486
— from myelitis, i, 447
— from neurasthenia spinalis, i, 463
— painful, i, 475
— partial, i, 431
— reflex, i, 460
— spastic, i, 480
— from spinal caries, i, 471
— syphilitic, i, 459
— from tumour within the cord, i, 450
— in tabes, i, 527
Parasites, animal—
— Acarus scabiei, ii, 788
— Ancylostomum duodenale, ii, 293
— Ascaris lumbricoides, ii, 288
— Bilharzia hæmatobia, ii, 304
— Bothriocephalus latus, ii, 283
— Oxyuris vermicularis, ii, 291
— Pediculus, ii, 786, 801
— Tænia mediocanellata, ii, 282
— — solium, ii, 280
— Trichina spiralis, ii, 296
Parasites, vegetable, i, 18; *see* Microphytes
— Achorion Schœnleinii, ii, 868
— Microsporon mentagraphytes, ii, 851
— — furfurans, ii, 870
— Trichophyton tonsurans, ii, 858
Parasitic anæmia, ii, 294
— hæmaturia, ii, 557
— sycosis, ii, 851
Parasiticides, vegetable, ii, 862
— animal, ii, 286
Parenchymatous nephritis, ii, 441, 476
Paresis in paralysis agitans, i, 716
Parkinson's disease, i, 715
Parotitis, ii, 137 *et seq.*
— in relapsing fever, i, 129

Parotitis after typhus, i, 113
— after yellow fever, i, 340
— after influenza, i, 1127
Parovarian cysts, ii, 326
Paroxysmal hæmoglobinuria, ii, 434
— insanity, i, 773
— neuroses, i, 748
— vertigo, i, 767
Pavy's liquid, ii, 565
Pébrine, i, 318, 367
Pectoriloquy, i, 939
Pediculi capitis, ii, 786
— corporis s. vestimentorum, ii, 801
Pelade, ii, 872; *see also* Area
Peliosis rheumatica, ii, 690
Pellagra, ii, 922
Pelvic symptoms in paraplegia, i, 439
Pelvis in osteomalacia, ii, 747
— in rickets, ii, 736
Pemphigus, ii, 815
— acutus, ii, 818
— foliaceus, ii, 818
— gangrænosus, ii, 818
— neonatorum, i, 300; ii, 817
— pruriginosus, ii, 820
— serpiginosus, ii, 819
— vegetans, ii, 819
Penicillium glaucum in urine, ii, 569
Pentastoma, ii, 405
Pepsin in treatment of dyspepsia, ii, 165
Peptones in urine, ii, 450
Percussion in ascites, ii, 324
— of chest, i, 926
— in emphysema, i, 968
— of heart, ii, 35 *et seq.*
— of liver, ii, 363
— of spleen, i, 329
Perforating ulcer of the foot, i, 530
Perforation of intestine in enteric fever, i, 146
— of stomach by gastric ulcer, ii, 180
Pericardial adhesions, ii, 97
— effusion, ii, 95
Pericarditis, ii, 92–100
— in *Bright's* disease, ii, 460
— in acute pneumonia, i, 989
— in rheumatism, ii, 691
— in scarlatina, i, 191
Perichondritis, laryngeal, i, 906
Perihepatitis, ii, 327, 373
— acute, ii, 364
Perinephric abscess, ii, 538
— diagnosis from enteric fever, i, 154
Periosteal cachexia, ii, 740
Peripheral neuritis, i, 422 *et seq.*
— paralysis, i, 396
Peripneumonia notha, i, 951
Peripneumony, i, 985, *foot-note*
Peristalsis, visible, ii, 263
Peritoneum, diseases of, ii, 305–321
Peritonitis, acute, ii, 305 *et seq.*
— in enteric fever, i, 146
— from erysipelas of abdominal wall, i, 253
— with effusion, ii, 316
— from obstruction, ii, 269
— from renal calculus, ii, 522
— cancerous, ii, 321
— circumscribed, ii, 310
Peritonitis from perforation of gastric ulcer, ii, 180, 307
— — of typhoid ulcer, i, 146; ii, 307
— — of appendix cæci, ii, 307
— puerperal, ii, 308
— chronic, ii, 316
— tubercular, ii, 318
Perityphlitis, ii, 220
Perlsucht, i, 315
Pernicious anæmia, ii, 640
— malarial fever, i, 329
Pernio, ii, 880
Peroneal atrophic paralysis, i, 514
Pertussis, i, 1136
— broncho-pneumonia in, i, 1138
Pestis, *see* Plague
Petechiæ, ii, 933
— in epilepsy, i, 782
— in erythematous rashes, ii, 832
— in measles, i, 181
— in purpura, ii, 660
— in rheumatism, ii, 690
— in scurvy, ii, 653
— in typhus, i, 107
Petit mal, i, 750
Pettenkofer's test, ii, 339
Peyer's patches in enteric fever, i, 155
Pharyngitis, granular, ii, 142
— membranous, in glanders, i, 361
— — in measles, i, 179
— — in scarlatina, i, 191
Pharynx, adenoid growths of, ii, 144
— anæsthesia of, in hysteria, i, 805
— diphtheria of, i, 265
— diseases of, ii, 142 *et seq.*
— paralysis of, in bulbar paralysis, i, 507
— — in diphtheria, i, 270
Physostigma in tetanilla, i, 713
— in general paralysis, i, 688
— in spastic paraplegia, i, 683
Phlebitis, ii, 123
— suppurative, i, 62
Phlegmonous erysipelas, i, 248
— gastritis, ii, 158
Phlorizen producing glycosuria, ii, 570, 584
Phosphatic calculus, ii, 514
— urine, ii, 424
Phosphorus in *Hodgkin's* disease, ii, 633
— in neuralgia, i, 394
— in pernicious anæmia, ii, 648
— poisoning, ii, 381
Phrenitis, i, 793, *foot-note*
Phthinoid chest, i, 1069
Phthiriasis, ii, 786
Phthisis, i, 1053–1113
— anatomy of, i, 1062
— in animals, i, 1107
— as a cause of lardaceous disease, ii, 486
— diabetic, ii, 574
— duration of, 1077
— fibroid, i, 1007
— "florida," i, 1064
— following enteric fever, i, 149
— ab hæmoptoë, i, 1067
— *Koch's* treatment by inoculation, ii, 913
— in the insane, i, 845
— relation to heart disease, i, 1105

Phthisis, relation to inflammation, i, 308
— from inhalation of dust, i, 1087
— — carbonaceous particles, i, 1094
— — cotton or flax dust, i, 1087, 1096
— — oxide of iron dust, i, 1095
— — siliceous particles, i, 1087, 1096
— relation to malaria, i, 1104
— pleurisy in, i, 1035
— pneumothorax in, i, 1079
— syphilitic, i, 1091
— traumatic, i, 1104
— relation to tubercle, i, 307, 309, 1056
— tubercular, i, 1082
— intestinal ulceration in, ii, 216
Physical examination of chest, i, 926
— signs and symptoms, i, 6
— signs of valvular disease, ii, 62
Physiognomy of congenital syphilis, i, 303
— of hydrocephalus, i, 666
Physiological albuminuria, ii, 449
Pian, ii, 922
Pica, i, 804; ii, 169
Picric acid test for albumen in urine, ii, 446
— — for sugar in urine, ii, 567
Piedra, ii, 875
Pigeon-chest, ii, 736
Pigment, biliary, ii, 338
— in melanæmia, i, 330
— urinary, ii, 414
Pigmental anomalies of the skin, ii, 932
Pigmentation of viscera in purpura, ii, 662
Pill of digitalis, squills, and mercury, ii, 85
Pilocarpin in xerostomia, ii, 136
— in *Bright's* disease, ii, 509
Pinea, tumour of, i, 619
Pituitarium, tumour of, i, 619
Pityriasis capitis, ii, 811
— maculata et circinata, ii, 811
— nigra, ii, 811
— rosea, ii, 811
— rubra, ii, 812
— tabescentium, ii, 846
— versicolor, ii, 869; *see* Tinea versicolor
Placenta, syphilis of, i, 299
Plague, i, 220 *et seq.*
Plaques of enteric fever, i, 154
— opalines in syphilis, i, 288, 290
— à surface reticulée of enteric fever, i, 168
Plastic bronchitis, i, 980 *et seq.*
Pleomorphism, i, 15
Plessimeter, i, 926
Plethora, ii, 609
Pleurisy, i, 1018 *et seq.*
— with broncho-pneumonia, i, 1004
— in *Bright's* disease, i, 1034; ii, 460
— with cirrhosis of lung, i, 1009
— dry, i, 1034
— with lobar pneumonia, i, 988
— in phthisis, i, 1035, 1060, 1065
— in rheumatism, i, 1034
— from ruptured hydatid, ii, 400
— in scarlatina, i, 191
— with effusion, i, 1020
— — diagnosis from aneurysm, i, 1031
— — — from cancerous growth, i, 1032
— — — from hydatid of liver, i, 1031
— — — from pericardial effusion, i, 1031
Pleurisy, with serous effusion, diagnosis from empyema, i, 1032
Pleuritic friction-sound, i, 1031
Pleurodynia, i, 949
Plica polonica, ii, 859
Plumbism, ii, 205–210
— and toxic anæmia, ii, 617
— gout, nephritis, and aneurysm, ii, 104
— as a cause of renal cirrhosis, ii, 494
— — of the "blue line," i, 520
— — of atrophy of brain, i, 689
— — of colic, ii, 205
— — of gout, ii, 678
— — of paralysis, i, 519
— and mental diseases, i, 845
— and peripheral neuritis, i, 425; ii, 520
Pneumonia, i, 985–1001
— acute lobar fibrinous, i, 1003
— of the apex, i, 994
— in *Bright's* disease, i, 1002
— catarrhal, i, 1003
— chronic, ii, 1002
— — interstitial, i, 1007
— — lobar, i, 1002
— pyæmic, i, 1007
— comparative pathology of, i, 995
— in croup, i, 888
— in diabetes, ii, 575
— in disease of heart, i, 1002
— epidemic, i, 995
— hypostatic, i, 144, 148
— following influenza, i, 1027
— and jaundice, ii, 342
— laryngeal ulcers, i, 898
— lobular, i, 1003, 1007
— vesicular, i, 1007
Pneumonoconiosis, i, 1092
Pneumopericardium, ii, 94
Pneumothorax, i, 1042–1059
— composition of air, i, 1046
— from emphysema, i, 1044
— from empyema, i, 1043
— from gangrene of lung, i, 1043
— from muscular effort, i, 1043
— in pertussis, i, 1043
— in phthisis, i, 1079
— traumatic, i, 1043
Pock of smallpox, i, 203
Poikilocytosis, *see* Pœcilocytosis
Point de côté of pleurisy, i, 1026
— of acute pneumonia, i, 992
Poisoning, chronic, by arsenic, i, 522
— — by lead, *see* Plumbism
— — by mercury, i, 521
— — by zinc, i, 523
— irritant, ii, 155
— narcotic, diagnosis from apoplexy, i, 585
— by phosphorus, ii, 381
Polariscope as a test for glycose, ii, 567
Polio-myelitis, anterior, i, 489
— — longa, i, 497
— in adults, i, 494
Polyarthritis, ii, 686
Polypoid form of tumour, i, 77
— growths in chronic laryngitis, i, 892
Polypus, intestinal, a cause of invagination, ii, 248

Polypus, nasal, a cause of asthma, i, 1146
— of larynx, i, 910
Polyuria, ii, 408
Pompholyx, ii, 815
— diutina, ii, 816
Pomphos, *see* Wheal
Pons, abscess of, i, 630
— hæmorrhage into, i, 583
Porrigo, ii, 796
— decalvans, ii, 872; *see also* Area
— lupinosa, ii, 868; *see also* Favus
— scutulata, ii, 858; *see also* Ringworm
Post-diastolic bruit, ii, 73, *foot-note*
Post-hemiplegic chorea, i, 569, 746
Post-peritoneal hernia, ii, 258
Post-pharyngeal abscess, i, 191, 889
Postponement of ague fit, i, 326
Potash salts in scurvy, ii, 658
— test for sugar in urine, ii, 566
Potassio-mercuric iodide test for albumen ii, 446
Potassium bromide in epilepsy, i, 765
— — in migraine, i, 785
— — in acute mania, i, 833
— — in paroxysmal vertigo, i, 772
— — in pertussis, i, 1142
— — in writers' cramp, i, 707
— ferrocyanide test for albumen, ii, 446
— iodide in chronic mercurialism, i, 523
— — in general paralysis, i, 687
— — in hemiplegia, i, 596
— — in migraine, i, 785
— — in plumbism, i, 520
— — in syphilis, i, 297, 304, 623
Pott's disease of spine, i, 471
Potters' phthisis, i, 1096
Poultices in dermatitis, ii, 774
Præsystolic bruit, ii, 72
— — of mitral stenosis, ii, 71
Pregnancy and acute atrophy of liver, ii, 380
— during chorea, i, 741
— in phthisis, i, 1090
Premonitory stage—
— of ague, i, 323
— of cholera, i, 227
— of enteric fever, i, 136
— of erysipelas, i, 250
— of measles, i, 179
— of meningitis, i, 639, 654
— — epidemic cerebro-spinal, i, 654
— of relapsing fever, i, 125
— of rubeola, i, 197
— of syphilis, i, 283
— of typhus, i, 105
— of varicella, i, 216
— of yellow fever, i, 339
Priapism in paraplegia, i, 438
Prickly heat, ii, 795
Prodroma—
— of acute ascending paralysis, i, 456
— of apoplexy, i, 581
— of epilepsy, i, 751
— of hydrophobia, i, 369
— of locomotor ataxy, i, 525
Proglottis of tænia, ii, 277, 280, 282, 284
Prognosis, i, 8
Progressive muscular atrophy of adults, i, 497
Progressive muscular atrophy of childhood, i, 513
— hereditary, i, 499
— with bulbar paralysis, i, 502
— primary form, i, 519
— of puberty, i, 514
— secondary forms, i, 502
— — in disseminated sclerosis, i, 504
— — in lateral sclerosis, i, 502
— — in locomotor ataxy, i, 504
— — with pachymeningitis cervicalis, i, 503
— — with spastic paraplegia, i, 502
— — with syringomyelus, i, 504
Prophylaxis against anthrax, i, 358
— against cholera, i, 243
— against enteric fever, i, 175
— against hydatid disease, ii, 401
— against hydrophobia, i, 376 *et seq.*
— against phthisis, i, 1107
— against scurvy, ii, 654
— against tapeworms, ii, 286
— against trichina, ii, 300
Prosopalgia, i, 886
Prurigo, ii, 800
— of *Hebra*, ii, 802
— infantilis, ii, 801
— senilis, ii, 801
— after varicella, i, 218
Pruritus, ii, 801
— from diabetes, ii, 573
— from jaundice, ii, 340
— from uræmia, ii, 470
Prussic acid, poisoning by, i, 586
Psammoma of cerebral meninges, i, 599
Psellismus mercurialis, i, 521
Pseudo-angina pectoris, ii, 24, 28, *foot-note*
Pseudo-hypertrophic paralysis, i, 514
Psoriasis, ii, 804
— and gout, ii, 807
— guttata, ii, 808
— gyrata, ii, 807
— linguæ, ii, 807; *see also* Leucoplakia
— palmar and plantar, i, 286
— and syphilis, ii, 898
Psorospermia, ii, 405, 784
Psychoses, i, 826–858
Pterygoid chest, i, 1089
Ptomaines, i, 11
Ptosis, i, 418
Ptyalism, ii, 135
Puerile respiratory murmur, i, 934
Puerperal insanity, i, 840
— peritonitis, ii, 308
Pulmonary apoplexy, ii, 59, 62
— artery, atheroma of, i, 961
— catarrh, i, 1003
— collapse, i, 958
— congestion, acute, i, 1002
— embolism, ii, 125 *et seq.*
— (dextro-sigmoid) incompetence, ii, 80
— (dextro-sigmoid) stenosis, ii, 79
Pulsating empyema, i, 1031
Pulse, alternating, ii, 10
— in anæmia, ii, 615
— in aneurysm, ii, 107
— of aortic obstruction, ii, 67
— of aortic regurgitation, ii, 68

Pulse in apoplexy, i, 579
— in *Bright's* disease, ii, 465
— disappearing on inspiration, ii, 14
— disorders of, ii, 3
— frequent, ii, 6
— hard, ii, 13
— high tension, ii, 465
— inequality on two sides, ii, 14
— infrequent, ii, 5
— intermittent, ii, 8
— irregular, ii, 9
— in jaundice, ii, 340
— of mitral regurgitation, ii, 77
— of mitral stenosis, ii, 75
— in pericardial effusion, ii, 96
— in peritonitis, ii, 306
— in phthisis, i, 1064
— in pyrexia, i, 39
— ratio to respiration in pneumonia, i, 991
— slow and infrequent, ii, 6
— tension of, ii, 13
— water-hammer, ii, 68
— wiry, ii, 14
Pulsus paradoxus, ii, 98
Pupils contracted from pressure on sympathetic, ii, 107
— — from aneurysm of transverse aorta, ii, 107
— — from thoracic growths, ii, 119
— — in typhus, i, 108
— dilated in chorea, i, 728
— — in enteric fever, i, 418
— in general paralysis of insane, i, 683
— in locomotor ataxy, i, 529
— in meningitis, i, 641
— in migraine, i, 778
— in tabes, i, 529
Purgatives in *Bright's* disease, ii, 509
— in dyspepsia, ii, 335
— in uræmia, ii, 468
— use and abuse of in constipation, ii, 211
Purpura, ii, 659
— in epidemic meningitis, i, 656
— in measles, i, 181
— in plague, i, 222
— in relapsing fever, i, 126
— in sarcomatosis, i, 85
— in scarlatina, i, 188
— in smallpox, i, 202
— in typhus, i, 106
— urticans, ii, 830
— variolosa, i, 202
Purpurine, ii, 421
Pus in urine, causes of, ii, 536
— — tests for, ii, 526
Pus-cells, i, 43
Pustular eczema, ii, 785
Pustule, ii, 756
— malignant, i, 350
Putrid urine, ii, 427
Pyæmia, i, 61
— and cerebral abscess, i, 625
— chronic, i, 69
— diagnosis from apoplexy, i, 587
— — enteric fever, i, 153
— diagnosis from rheumatism, ii, 700
— idiopathic, i, 65
Pyæmia, internal, i, 62
— as a cause of jaundice, ii, 342
Pyelitis, ii, 536
— tubercular, ii, 546
Pylephlebitis purulenta, ii, 342
— adhesiva, ii, 328
Pylorectomy, ii, 201
Pylorus, cancer of, ii, 196
Pyo-pneumothorax, i, 1046
Pyrexia, i, 27, 1027; *see also* Temperature, Fever
— asthenic, i, 40
— in cirrhosis, ii, 372
— classification of, i, 30
— clinical varieties of, i, 35
— course of, i, 37
— degrees of, i, 30
— defervescence, i, 38
— fastigium, i, 37
— hectic variety, i, 35
— idiopathic, i, 34
— initial stage, i, 36
— in leuchæmia, ii, 622
— in phthisis, i, 1063
— in pneumonia, i, 993
— in acute pulmonary tuberculosis, i, 1116
— pulse in, i, 39
— respiration in, i, 41
— stages of, i, 29, 36
— sthenic, i, 40
— symptomatic, i, 34
— theories of causation, i, 32
— wasting in, i, 38
Pyrogallic acid locally in psoriasis, ii, 809
— — and hæmoglobinuria, ii, 433
— — for ringworm, ii, 864
Pyrosis, ii, 170
Pythogenic fever, i, 136
Pyuria, ii, 536
— in cystitis, ii, 536
— in suppurative nephritis, ii, 539
— in tubercular disease of kidney, ii, 548

Q.

Quantitative estimation of albumen in urine, ii, 447
— — of sugar in urine, ii, 568
— — of uric acid in urine, ii, 419
Quarantine, i, 17
— against plague, i, 224
— against yellow fever, i, 345
Quartan ague, i, 325
Quinine in ague, i, 337
— in eczema, ii, 781
— in facial neuralgia, i, 393
— in hæmoglobinuria, ii, 436
— in pertussis, i, 1142
— in prurigo, ii, 803
— rash, ii, 836
Quinsy, ii, 139
Quotidian ague, i, 325

R.

Rabies in animals, i, 372
— in man, i, 373

Rachialgia, i, 464
Rachitic chest, ii, 736
Rachitis, ii, 733; *see also* Rickets
Radesyge, i, 277
Râles in bronchitis, i, 953
— consonating, i, 938
— in phthisis, i, 1072, 1073
— in pneumonia, i, 989
— theory of, i, 937
Rarefied air in treatment of bronchitis, i, 979
Raw, definition of, ii, 758
Raynaud's disease, ii, 439
Reaction of degeneration, i, 399
— in acute atrophic paralysis, i, 490
— in chronic atrophic paralysis, i, 495
— in disseminated sclerosis, i, 545
— in paraplegia, i, 435
— in progressive muscular atrophy, i, 498
Recovery from acute yellow atrophy, ii, 869
— from hydrophobia (?), i, 876, *foot-note*
— from phthisis, i, 1061, 1080
— from tubercular meningitis, 646
— from tetanus, i, 727
Recurrence of ague-fit, i, 325
— of cholera, i, 243
— of eczema, ii, 769
— of enteric fever, i, 151
— of erysipelas, i, 252
— of measles, i, 183
— of mumps, ii, 136
— of plastic bronchitis, i, 983
— of pneumonia, i, 995
— of relapsing fever, i, 128
— of rubeola, i, 198
— of scarlatina, i, 193
— of syphilis, i, 293
— of typhus, i, 116
— of varicella, i, 219
— of variola, i, 210
— of whooping-cough, i, 1139
Recurrent fibroid tumour, i, 76
— laryngeal nerve, paralysis of, i, 869
Red softening of cord, i, 440
Reduplicated cardiac sounds, ii, 74
— second sound, ii, 63
Reflex action in chorea, i, 728
— — abolished by shock, i, 433
— — co-ordination of, i, 432
— — in acute atrophic paralysis, i, 490
— — in chronic atrophic paralysis, i, 495
— — in hemiparaplegia, i, 469
— — in hysteria, i, 806
— — in paraplegia, i, 431
— — loss of, in Landry's paralysis, i, 457
— paraplegia, i, 460
Reflexes, deep or visceral, i, 434
— superficial, i, 433
Regurgitation, gastric, ii, 169
— œsophageal, ii, 147
— through pulmonary valve, ii, 80
— through aortic valve, ii, 67
— through mitral valve, ii, 75
— through tricuspid valve, ii, 80
Relapse of rheumatism, ii, 696
— of cholera, i, 230
— in epidemic meningitis, i, 658
— of enteric fever, i, 149
Relapse of measles, i, 183
— of relapsing fever, i, 127
— of typhus, i, 110
— of varicella, i, 218
Relapsing fever, i, 124 *et seq.*
Remittent fever, i, 328
Renal calculi, ii, 512
— casts, ii, 454
— colic, ii, 522
— cushion, ii, 457
— cyst, ii, 496
— dropsy, ii, 456, 480
— inadequacy, ii, 412
— pulse, ii, 465
Resolution of inflammation, i, 55
Resonance, amphoric, i, 930
— osteal, i, 930
— pulmonary, i, 927
— tubular, i, 930
— tympanitic, i, 929
Restraint of insane patients, i, 847
Retinal hæmorrhage in m. *Brightii*, ii, 461
Retinitis from cerebral tumour, i, 606
— from lardaceous kidneys, ii, 491
— from leuchæmia, ii, 622
— from meningitis, i, 652
— from parenchymatous nephritis, ii, 460
— from syphilis, i, 304
Retraction of head in meningitis, i, 655
Retroperitoneal hernia, ii, 258
Retropharyngeal abscess, i, 889
Rhagades, ii, 758
Rheumatic fever, ii, 686
— nodules, ii, 692
"Rheumatic gout," ii, 722
Rheumatism, acute, ii, 686–712
— chronic, ii, 713
— and chorea, i, 741
— and erythema, ii, 834
— and gout, ii, 699
— effect on cardiac muscle, ii, 42
— muscular, ii, 714
— after scarlatina, i, 191
Rheumatoid arthritis, ii, 722
Rhinoderma, ii, 796
Rhinoscleroma, ii, 917
Rhonchus, i, 953
Rib, excision in empyema, i, 1042
Rice-water stools of cholera, i, 228, 234
Rickets, ii, 733–745
— acute, ii, 739
— and hypertrophy of brain, i, 691
— and laryngismus stridulus, i, 879
— and tetany, i, 711
Right and left sides—liability to pleurisy, i, 1027, 1046
— — to pneumonia, i, 987
— — to zona, ii, 988
Rigidity, early, of hemiplegia, i, 565
— late, of hemiplegia, i, 567
— to hysteria, i, 806
— from insular sclerosis, i, 544
— from meningeal hæmorrhage, i, 578
— of neck in meningitis, i, 655
— in paralysis agitans, i, 716
— in spastic paraplegia, i, 481
Rigors, i, 36; ii, 932

Rigors, febrile, i, 125, 179, 197, 200, 250
— from abscess of brain, i, 622
— in ague, i, 324
— in pneumonia, i, 991
Ringworm of the body, ii, 865
— of the chin, ii, 851
— of the scalp, ii, 858
Risus sardonicus, i, 721
Rodent ulcer, i, 92; ii, 931
— — diagnosis from lupus, ii, 905
Roseola, ii, 825
— annulata, ii, 826
— choleraica, i, 230
— epidemica, i, 195
— furfuracea, ii, 825
— syphilitica, i, 285; ii, 897
— variolosa, i, 201
"Rotation" in hysteria, i, 819
Rötheln, i, 195
Round-worms, ii, 288
Rub in broncho-pneumonia, i, 1005
— in emphysema of lungs, i, 971
— pericardial, ii, 94
— peritoneal, i, 1019
— pleuritic, i, 1019
Rubeola, i, 195 *et seq.*
Rumination, ii, 147, 170
Rupia, ii, 899
Rupture of heart, ii, 52

S.

Saccharine in diabetes, ii, 588
Saccharometer, ii, 567, 568
Sago-grain vesicles, ii, 857
Sago-spleen, ii, 606
St Vitus's dance, i, 728
Salaam-palsy, i, 701
Salicin in rheumatism, ii, 704 *et seq.*
Salicylate of soda in rheumatism, ii, 704
Salicylic acid for corns and callosities, ii, 882
— reduction of copper in urine, ii, 566
— rash, ii, 837
Salivary glands, diseases of, ii, 135 *et seq.*
Salivation, ii, 135
— in bulbar paralysis, i, 508
— in hydrophobia, i, 371
— from mercury, i, 521; ii, 135
— from potassium iodide, ii, 135
— in smallpox, i, 208
Saltatorial spasms, i, 700
Santonin as a vermifuge, ii, 291
Sarcina ventriculi, i, 14; ii, 197
— in urine, ii, 431
Sarcoma, i, 80
— alveolar, i, 81
— of brain, i, 600
— of cerebral meninges, i, 600
— of kidney, ii, 550
— of larynx, i, 912
— of liver, ii, 387
— lymphatic, i, 87
— of mediastinum, ii, 115
— melanotic, i, 82
— multiple, i, 83
Sarcoma, round-celled, i, 80
— of skin, i, 82; ii, 931
— of spinal cord, i, 451
— spindle-celled, i, 80
— of stomach, ii, 191
Sarcomatosis, i, 83
Sarcoptes hominis, ii, 789; *see also* Acarus
Saturnine gout, ii, 678
— palsy, i, 519, *foot-note*
Satyriasis, ii, 918
Sayre's jacket in spinal caries, i, 479
Scab, ii, 757
Scabies, ii, 788 *et seq.*
Scale, ii, 758
Scarification for gutta rosea, ii, 879
— for lupus, ii, 912
Scarlatina, i, 185 *et seq.*
— anginosa, i, 190
— hæmorrhagica, i, 190
— maligna, i, 190
Scarlatinal nephritis, ii, 476, 481, *foot-note*
Scars, ii, 758
— in relation to cheloid, ii, 924
Scelotyrbe festinans, i, 717
Scherlievo of Fiume, i, 277
Schizomycetes, i, 14
Sciatica, i, 390 *et seq.*
Scirrhus, i, 90; ii, 930
Sclerema, ii, 890
Scleriasis, ii, 888
Sclerodermia, ii, 888
Sclerosis of brain, i, 690
— of cord, i, 441
— disseminated cerebro-spinal, i, 541
— insular, i, 541
— lateral, i, 502
— — after cerebral lesion, i, 568
— — ascending, i, 481
— — descending, i, 477
— — in general paralysis, i, 685
— miliary, i, 685
— posterior in general paralysis, i, 685
— — in locomotor ataxy, i, 532
Sclerostomum duodenale, ii, 293
Sclerotitis in gonorrhœal arthritis, ii, 717
Scorbutus, ii, 650
Scraping in treatment of lupus, ii, 912
Scratch mark, ii, 758
Scrivener's palsy, i, 702
Scrofula, i, 1089; ii, 908
Scrofulides, ii, 907
Scurvy, ii, 650 *et seq.*
— as a cause of pericarditis, ii, 93
Seasons, effect on bronchitis, i, 977
— relation to pneumonia, i, 996
— — to rheumatism, ii, 693
Sea voyage in phthisis, i, 1108
Sebaceous cyst, ii, 846
Seborrhœa, ii, 845
— congestiva, ii, 914
— corporis, ii, 795
— sicca, ii, 845
Secondary fever of smallpox, i, 208
Semi-tertian ague, i, 325
Sensation in disseminated sclerosis, i, 544
— in hemiplegia, i, 564
— in locomotor ataxy, i, 526

Sensation in migraine, i, 778
— in paralysis agitans, i, 716
— in paraplegia, i, 480
— in writers' cramp, i, 703
— in hysteria, i, 804
Septicæmia, i, 62
Serous inflammations in m. *Brightii*, ii, 460
Serratus magnus, paralysis of, i, 397
Sexes, proportion of, affected with—
— aneurysm, ii, 103
— exophthalmic goitre, ii, 126
— general paralysis, i, 681
— gout, ii, 676
— hæmoglobinuria, ii, 435
— thoracic tumours, ii, 120
Shaking palsy, i, 715
Shampooing, *see* Massage
Shingles, *see* Zona
Sibbens, i, 277; ii, 922
Sibilus, i, 937, 953
Sick headache, i, 775
— — and xanthelasma, ii, 894
Siderosis of lungs, i, 1095
Silver-salts in treatment of tabes, i, 537
— — of epilepsy, i, 767
Simulation of epilepsy, i, 763
Singultus, ii, 178
Sinuses of skull, thrombi of, i, 675
Skatol, ii, 415
Skin, diseases of, ii, 752 *ad finem*; *see also* Eruptions
— in diabetes, ii, 572
— in phthisis, i, 1063
Skoda's resonance in pleural effusion, i, 1025
Skull of congenital syphilis, i, 303
— of cretins, i, 859
— of hydrocephalus, i, 666
— of rickets, ii, 737
Smallpox, i, 199–214
Smell, paralysis of, i, 421
Smoking, effect on heart, ii, 41
Soda in jaundice, ii, 348
Soft chancre, i, 280
Softening of the brain, 553, 634, 675
— — relation to hæmorrhage, i, 555
— — red, i, 554, 632
— — white, i, 554, 675
— — yellow, i, 554, 601
— of spinal cord, i, 439
— — relation to hæmorrhage, i, 440
Soil and phthisis, i, 1099
Soil-water theory of cholera, i, 241
— — of enteric fever, i, 166
Soldiers, hypertrophy of heart in, ii, 39
— albuminuria in, ii, 449, 450
Solitary follicles in dysentery, ii, 225
— in enteric fever, i, 155
— in *Hodgkin's* disease, ii, 631
Solvent treatment of renal calculi, ii, 540
Somnambulism, i, 817
— in paroxysmal insanity, i, 773
Sore throat, ii, 139
Spasm of glottis in laryngismus, i, 877
— — in spurious croup, i, 885
— — in true croup, i, 887
— — in hysteria, i, 807
— of face, i, 694
Spasm of muscles after facial paralysis, i, 412
— of throat in hydrophobia, i, 870
Spasms produced by fatigue, i, 699
Spasmodic croup, i, 884
— nervous diseases, i, 693 *et seq.*
— tic, i, 694
— torticollis, i, 694
Spasmus glottidis, i, 877
Spastic paraplegia, i, 480
— — in children, i, 482
— — with atrophy, i, 502
— paresis in insular sclerosis, i, 544
Specific diseases, i, 11, 16, 96
— — characteristics of, i, 100
— — table of, i, 102
— gravity of urine, ii, 407
— — — in diabetes, ii, 569
— — — in m. *Brightii*, ii, 480, 490, 497
Spedalskhed, *see* Leprosy
Speech in bulbar paralysis, i, 507
— in chronic mercurialism, i, 521
— in disseminated sclerosis, i, 543
— in general paralysis, i, 683
— in idiocy, i, 856
— in migraine, i, 778
— in paralysis agitans, i, 717
Spermatorrhœa, i, 824
Sphacelus, *see* Gangrene
Sphygmograph, ii, 3
Sphygmographic tracings in fever, i, 40
Spina bifida and hydrocephalus, i, 666
Spinal cord, anæmia of, i, 451
— in acute atrophic paralysis, i, 492
— in chronic atrophic paralysis, i, 495
— concussion of, i, 454
— in general paralysis, i, 686
— glioma of, i, 451
— hæmorrhage into, i, 449
— hyperæmia of, i, 453
— irritation of, i, 464
— in locomotor ataxy, i, 531
— in progressive muscular atrophy, i, 500
— sarcoma of, i, 451
— sclerosis of, i, 441; *see also* Sclerosis
— segment of, i, 429, *foot-note*
— sensory and motor tracts of, i, 433
— softening of, i, 440
— syphilis of, i, 459
— tubercle of, i, 450
— tumour of, i, 473
Spinal curvature, result of wryneck, i, 697
— paralysis in adults, i, 494
— — chronic cases, i, 495
Spine, *see* Vertebræ
Spirillum, i, 14
— of relapsing fever, i, 129
Spirochæta Obermeieri, i, 130, 131
Spirometer, i, 942
Spleen, abscess of, ii, 60, 605
— in ague, i, 329
— in anthrax, i, 355
— in cardiac disease, ii, 60
— in cirrhosis of liver, ii, 371
— diseases of, ii, 604
— in enteric fever, i, 158
— in *Graves'* disease, ii, 130
— in *Hodgkin's* disease, ii, 629

Spleen, hydatid of, ii, 606
— lardaceous, ii, 606
— in leuchæmia, ii, 619
— physical diagnosis of, ii, 619
— in relapsing fever, i, 129
— in rickets, ii, 738
— in acute tuberculosis, ii, 392, 606
— in typhus, i, 111
Splenectomy, ii, 626
Splenisation of lung, ii, 60
Spontaneous origin of contagia, i, 16
Spoon, sharp, in treatment of lupus, ii, 912
Spurious croup, i, 885
— hydrocephalus, i, 645
Sputa in anthracosis, i, 1095
— in asthma, i, 1147
— in bronchiectasis, i, 974
— in bronchitis, acute, i, 956
— — chronic, i, 962
— — fœtid, i, 974
— in gangrene of lung, i, 1014
— in phthisis, i, 1066
— in pneumonia, i, 992
— in acute pulmonary tuberculosis, i, 1116
Stammering, i, 701
Staphylococcus, i, 14
Starvation in aneurysm, ii, 109
Statistics from Guy's Hospital—
— of abscess of brain, i, 627 (Pitt)
— of abscess of liver, ii, 358 (Campbell)
— of aneurysm, ii, 114 (Parkin)
— of acute yellow atrophy, ii, 382 (P. S.)
— of area, ii, 873 (P. S.)
— of cancer of stomach, ii, 190, 194 (Shaw)
— of cerebral embolism, i, 550 (C. H. F.)
— — hæmorrhage, i, 560 (C. H. F.)
— — tumours, i, 623 (W. H. White)
— of chorea, murmur in, i, 730 (Manser, Halstead, Goodall)
— — sex and age, i, 738 (Hughes and P. S.)
— of cirrhosis, hepatic, ii, 366 (Price)
— of colotomy, ii, 267 (Bryant)
— of diabetes (anatomy), ii, 578 (Campbell)
— — and phthisis, ii, 575, (Kelbe)
— of erythema, ii, 814 (P. S.)
— of gastric ulcer, ii, 182, 183 (Keiffenheim)
— of gonorrhœal synovitis, ii, 716 (Ricketts)
— of hæmoptysis in m. cordis, ii, 62, *foot-note* (Shaw)
— of bruits in rheumatism, ii, 690 (Manser)
— of hereditary diabetes, ii, 585 (Nevins)
— of intestinal obstruction, ii, 275 (Shaw and P. S.)
— of invagination, ii, 246, 250 (P. S.)
— of lardaceous disease, ii, 485, 486 (Lancaster and Campbell)
— of locality of cerebral hæmorrhage, i, 556 (C. H. F.)
— of miliary tuberculosis (age), i, 1120 (C. H. F.)
— of osteoarthritis, ii, 727 (P. S.)
— of paralysis agitans, i, 718 (Shaw)
— of pityriasis rubra, ii, 814 (P. S.)
— of primary peritonitis with effusion, ii, 317 (C. H. F.)
— of psoriasis (sex and age), ii, 808 (P. S.)
Statistics, from Guy's Hospital—
— of rheumatism, ii, 694 (Manser and Shadwell)
— — and chorea, i, 742 (Hughes and P. S.)
— — and seasons, ii, 693 (Capes)
— — natural course, ii, 703 (Gull and Sutton)
— — salicylic treatment, ii, 705 (C. H. F.)
— of ringworm, ii, 861 (P. S.)
— of sclerodermia, ii, 890 (C. H. F. and P. S.)
— of sex and age in tubercular meningitis, i, 639 (C. H. F.)
— of spinal paralysis, i, 547 (Halstead and Goodall)
— of treatment of empyema in children, i, 1042 (Goodhart)
— of tuberculous nephritis, ii, 547 (C. H. F.)
— — peritonitis, ii, 319 (C. H. F.)
— of typhlitis, ii, 222 (C. H. F. and P. S.)
— of whooping-cough, i, 1140 (Goodhart)
— of zona, ii, 940 (P. S.)
Status epilepticus, i, 797
Steatoma, ii, 846
Stenosis of aortic orifice, ii, 67
— of mitral orifice, ii, 71
— of pulmonary orifice, ii, 79
— of trachea, i, 919
— of tricuspid orifice, ii, 80
Stertor in apoplexy, i, 579
Stethometer, i, 942
Stethoscope, i, 932
Stigmata, i, 797
Stomacace, ii, 133
Stomach, carcinoma of, ii, 191
— acute catarrh of, ii, 152
— chronic, ii, 159
— cirrhosis of, ii, 202
— colloid cancer of, ii, 193
— concretions in, ii, 202
— congestion of, ii, 161
— — in cardiac disease, ii, 60
— — in chronic bronchitis, i, 961
— — in acute catarrh, ii, 154
— — in chronic catarrh, ii, 159
— — in cirrhosis of liver, ii, 371
— cough, i, 948
— dilatation of, acute, ii, 157
— — chronic, ii, 168, 197
— diseases of, ii, 152–202
— post-mortem digestion of, ii, 154
— sarcoma of, ii, 191
— ulcer of, ii, 178
— washing out, ii, 201
Stomatitis, ii, 131 *et seq.*
— in congenital syphilis, i, 301
— gangrenous, ii, 133
Stools of acute yellow atrophy, ii, 378
— of cholera, i, 228
— of dysentery, ii, 233
— of enteric fever, i, 140
— of hæmatemesis, ii, 174
— of obstructive jaundice, ii, 345
Strabismus, i, 414 *et seq.*
Stramonium in treatment of asthma, i, 1148
Strangulation of bowel, ii, 257
Streptococcus, i, 14
Striæ atrophicæ of skin, ii, 891

Stricture of bile-duct, ii, 344
— of colon, cancerous, ii, 257
— of intestine, ii, 256
— — after intussusception, ii, 245
— — after tubercular ulcer, ii, 320
— of œsophagus, ii, 147, 148
— of pylorus, ii, 196
— of ureter, ii, 531
Strobila of tænia, ii, 277
Strongylus gigas, ii, 295
Strophanthus in cardiac disease, ii, 86
Strophulus, ii, 796
— albidus, ii, 845
Struma, ii, 908
Strychnia in treatment of myelitis, i, 449
— — of paralysis of larynx, i, 873
— poisoning by, i, 724
Stupor, i, 886
Styptics in hæmatemesis, ii, 178
— in hæmaturia, ii, 521
— in hæmophilia, ii, 668
— in hæmoptysis, i, 1112
— in purpura, ii, 664
— in scurvy, ii, 658
Subintrant ague, i, 326
Subsultus tendinum in fever, i, 108
Succussion-splash, i, 1048
Sudamina, ii, 689, 857
Sudden death in cardiac disease, ii, 83
Sugar in the urine, ii, 564
Suicidal tendency in melancholia, i, 835
Sulphate of lime in urine, ii, 427
Sulphur in treatment of scabies, ii, 792
Sulphuric acid, its chemical effects, ii, 178
Sulphurous acid as a parasiticide, ii, 864
Sunstroke, i, 787
Suppression of urine, *see* Urine
Suppuration and lardaceous disease, ii, 485
Suppurative nephritis, ii, 538
— — in paraplegia, i, 437
Supra-clavicular swelling in cretins, i, 861
Suspension in treatment of tabes, i, 538
Sweat as an irritant, ii, 857
Sweat-glands, affections of, ii, 855 *et seq.*
Sweating in ague, i, 325
— local, in aneurysm, ii, 107
— in enteric fever, i, 138
— in paralysis agitans, i, 717
— in phthisis, i, 1063
— in rheumatism, ii, 689
— in rickets, ii, 739
— sickness, i, 225
— in tetanus, i, 722
Sycosis, ii, 849
— capillitii frambœsiformis, ii, 852
— parasitica, ii, 851
Symmetrical gangrene, ii, 489
Symmetry in cutaneous diseases, ii, 759
— of eczema, ii, 766
— of erythema, ii, 824
— of lupus erythematosus, ii, 915
— of phthisis, i, 1055
— of psoriasis, ii, 806
— of scabies, ii, 790
— of syphilodermia, ii, 900
Sympathetic ganglia in *Graves'* disease, ii, 129
Sympathetic ganglia in *Addison's* disease, ii, 600
— in general paralysis of the insane, i, 686
Symptoms, objective and subjective, i, 5
Syncopal ague, i, 327
Syncope, ii, 14
— fatal, in acute alcoholism, i, 792
— — in cardiac disease, ii, 82
— — in diphtheria, i, 266
— — in phthisis, i, 1079
— — in pleuritic effusion, i, 1036
Synovial rheumatism, ii, 713
Synovitis after bronchiectasis, i, 976
— in dysentery, ii, 235
— gonorrhœal, ii, 716
— tubercular, i, 1118
— in scarlatina, i, 191
Syphilis, i, 276
— a cause of aneurysm, ii, 104, 112
— of cerebral arteries, i, 551, 592
— of brain and meninges, i, 598
— congenital, i, 297
— — of bones, i, 299, 302
— — of cornea, i, 304
— — of the lung, i, 1013
— cutanea, i, 285; ii, 896
— a cause of lardaceous disease, ii, 485
— of larynx, i, 900
— of liver, ii, 374
— of lungs, i, 1011, 1092, *foot-note*
— a cause of meningitis, i, 648
— a cause of myelitis, i, 444
— of placenta, i, 299
— preceding hæmoglobinuria, ii, 435
— primary, i, 279, 283
— secondary, i, 284
— of spinal arteries, i, 459
— of spinal meninges, i, 459
— a cause of tabes, i, 536
— tertiary, i, 289
Syphilitic arteritis, i, 551
— cardiac gumma, ii, 46
— fever, i, 284
— insanity, i, 844
— phthisis, i, 1091
— stenosis of trachea and bronchi, i, 919
— ulcer, diagnosis from lupus, ii, 905
Syphilodermia, ii, 896
— of congenital syphilis, i, 301
— macular, i, 286
— papular, i, 286
— pustular, i, 287
— squamous, i, 287
— tertiary, i, 285, 289
Syringomyelus, i, 451, 504
Systemic diseases of the cord, i, 489

T.

"Tabby" degeneration of heart, ii, 51
Tabes, i, 1053, *foot-note*
— and insanity, i, 846
— dorsalis, i, 524 *et seq.*
— mesenterica, ii, 319
Tabid arthritis, ii, 731
Tache cérébrale, i, 641
Tachycardia, ii, 6

Tactile vocal fremitus in pneumonia, i, 990
Tænia mediocanellata, ii, 282
— solium, ii, 280
Tapeworms, ii, 277
Tar acne, ii, 843
— locally in treatment of eczema, ii, 775
— — of lichen ruber, ii, 800
— — of psoriasis, ii, 809
Tarantulism, i, 818
Taraxacum in treatment of jaundice, ii, 348
Teeth ground down in lithæmia, ii, 334
— of inherited syphilis, i, 303
— in rickets, ii, 736
Teigne pelade, ii, 872; *see also* Area
Temperature, i, 30
— in abscess of brain, i, 629
— in acute atrophic paralysis, i, 490
— in acute miliary tuberculosis, i, 1116
— in acute yellow atrophy of liver, ii, 378
— in ague, i, 324
— in anæmia generally, ii, 615
— in grave idiopathic anæmia, ii, 644
— in apoplexy, i, 579
— in broncho-pneumonia, i, 1005
— in cirrhosis of liver, ii, 372
— in cholera, i, 229, 230, 232
— in chronic atrophic paralysis, i, 495
— in dengue, i, 347
— in diabetes, ii, 572
— in diphtheria, i, 265
— in enteric fever, i, 137, 141, 142, 150
— in epilepsy, i, 754
— in erysipelas, i, 250
— in glanders, i, 361
— in jaundice, ii, 343
— in leuchæmia, ii, 632
— local, in inflammation, i, 49
— — in muscular atrophy, i, 498
— in measles, i, 179, 180
— in meningitis, i, 643
— — epidemic cerebro-spinal, i, 656
— in myelitis, i, 445
— paradoxical, i, 31
— in peritonitis, ii, 306
— in phthisis, i, 1063
— in plague, i, 221
— in pleurisy, i, 1027
— in pneumonia, i, 993
— post-mortem rise in tetanus, i, 722
— in relapsing fever, i, 126, 127
— in renal tuberculosis, ii, 548
— in rheumatism, ii, 689
— in rubeola, i, 197
— in scarlatina, i, 187
— in smallpox, i, 200, 207, 209
— subnormal, i, 42
— in syphilis, i, 284
— in tetanus, i, 722
— in typhus, i, 105, 106
— in varicella, i, 218
— in yellow fever, i, 339
Tender points in neuralgia, i, 384, 387
— in rachialgia, i, 465
Tendon-reflex in paraplegia, i, 434
— in lateral sclerosis, i, 481
— absence in locomotor ataxy, i, 528
Tertian ague, i, 325
Tertiana soporosa, i, 327
Testis in *Hodgkin's* disease, ii, 632
— syphilitic, i, 291
— tubercle of, ii, 547; *see also* ii, 607
Tetanilla, *see* Tetany
Tetanus, i, 720
— acute, i, 726
— chronic, i, 726
— hydrophobicus, i, 376
— idiopathic, i, 723
— neonatorum, i, 922
— traumatic, i, 723
Tetany, i, 708
Thalamus, tumour of, i, 619
Therapeutics generally, i, 9
Thermometer, clinical, i, 27
Thermometric charts, i, 29
Thirst in diabetes insipidus, ii, 409
— — mellitus, ii, 571
Thomsen's disease, i, 713
Thoracocentesis, i, 1037
Thorax of cirrhosis of lung, i, 1010
— of empyema, i, 1030
— of phthisis, i, 1074
— of pleural effusion, i, 1023
— examination of, i, 926
— of emphysema, i, 969
— of phthisis, i, 1074
— of rickets, ii, 735
Thorn-apple crystals of lithic acid, ii, 418
Thread-worms, ii, 291
Thrill, tactile, ii, 65; *see also* Fremitus
— — in aneurysm, ii, 105
Throat-cough, i, 947
Thrombosis, ii, 123
— cardiac, ii, 46
— of cerebral arteries, i, 550
— — sinuses, i, 674
— of femoral vein in enteric fever, i, 149
— of portal vein, ii, 386
Thrush, ii, 134
Thymic asthma, i, 877
Thymol as a parasiticide, ii, 865
Thymus, suppuration in congenital syphilis, i, 300
Thyroid body, absence of in cretins, i, 860
— in exophthalmic goitre, ii, 127
— removal of, a cause of cretinism, i, 865
Thyrotomy, i, 911
Tic-douloureux, i, 386
— spasmodic, i, 694
Tichopsia, i, 776
Tinea, ii, 858 *et seq.*
— circinata, ii, 866
— erythrasma, ii, 868
— favosa, ii, 868
— imbricata, ii, 866
— marginata, ii, 866
— rosea, ii, 870; *see also* Pityriasis rosea
— scutulata, ii, 858
— sycosis, ii, 849
— tonsurans, ii, 858
— versicolor, ii, 869
Tongue in bronchitis, i, 957
— in chronic dysentery, ii, 236
— in diabetes, ii, 571
— in enterica, i, 138

Tongue in gastritis, ii, 153
— ichthyosis of, ii, 883
— paralysis of, i, 420
— in pneumonia, i, 993
— in rheumatism, ii, 688
— in scarlatina, i, 187
— in tertiary syphilis, i, 290
— tremulous in general paralysis, i, 683
— ulcer of, in pertussis, i, 1138
— ulcers of, ii, 132
Tonic spasm, i, 752
— wryneck, i, 695
Tonsillitis, acute, ii, 139
— chronic, ii, 141
— in rheumatism, ii, 692
— in scarlatina, i, 187
Tooth-cough, i, 948
Tophi, ii, 672
Torticollis, i, 695
Torula cerevisiæ, i, 14
— — in vomit, ii, 197
— in diabetic urine, iii, 569
— in gastric dilatation, ii, 197
Toxic albuminuria, ii, 453
— eruptions of the skin, ii, 834
— hæmoglobinuria, ii, 433
— paraplegia, i, 528
Trachea, obstruction of, i, 916, 919
— — from abscess of mediastinum, i, 918
— — from aneurysm, i, 917
— — from dilated left auricle, i, 919
— — from goitre, i, 917
— — from primary carcinoma, i, 919
— — from scrofulous glands, i, 918
— — from syphilitic ulcerations, i, 919
— — from tumour of mediastinum, i, 918
— — from ulceration in glanders, i, 919
— tubercular disease of, i, 894
Tracheotomy for removal of foreign body, i, 916
— for chronic laryngitis, i, 892
— for diphtheria, i, 274
— for paralysis of abductor muscles, i, 874
— for perichondritis of larynx, i, 909
— as a cause of pneumothorax, i, 1044
Trance, i, 817
Transfusion of blood in anæmia, ii, 648
— of saline solution in cholera, i, 245
Traube's plugs in fœtid bronchitis, i, 974
Traumatic diphtheria, i, 882
— hepatitis, ii, 361
Tremors, fibrillary, in general paralysis of the insane, i, 683
— — in muscular atrophy, i, 498
— from alcohol, i, 797
— in disseminated sclerosis, i, 543
— in hemiplegia, i, 569
— in insular sclerosis, i, 543
— mercurial, i, 521
— in paralysis agitans, i, 715
— in plumbism, i, 520
— in suppression of urine, ii, 525
— in typhus, i, 108
— in uræmia, ii, 469
— from zinc, i, 523
Trephining in epilepsy, i, 767
Trichina spiralis, ii, 296
Trichiniasis, ii, 298
— diagnosis from enteric fever, i, 153
Trichocephalus dispar, ii, 293
Trichoclasia, ii, 874
Trichophyton tonsurans, ii, 850, 858
Trichoptilosæ, ii, 874
Trichorrhexis nodosa, ii, 874
Tricuspid regurgitation, ii, 80
— — in emphysema, i, 971
— stenosis, ii, 80
Triethylamine in treatment of rheumatism, ii, 823
Triple phosphate, ii, 426
Trismus, i, 720
— neonatorum, i, 711
— in uræmia, ii, 469
Tropæolin in diagnosis of gastric disease, ii, 199
Trophic changes in hemiplegia, i, 565
— — in paraplegia, ii, 488
— — from peripheral neuritis, i, 404, 941
— — from tabes, i, 530
Tubal nephritis, ii, 443, 476
Tube-casts, ii, 454; *see also* Casts
Tubercle and scrofula, ii, 758
— of adrenals, i, 320; ii, 602
— articular, i, 320
— artificial production of, i, 313
— of brain, i, 597
— of cerebral meninges, i, 635
— of choroid, i, 644
— of cord, i, 450
— and cyanosis, ii, 88
— its general pathology, i, 306
— of genito-urinary tract, i, 320, ii, 546
— and gout, ii, 681
— infiltrated, i, 310
— its relation to inflammation, i, 308, 309
— of intestine, ii, 216
— of kidney, i, 320; ii, 544
— of larynx, i, 894
— of liver, ii, 392
— of lung, i, 306, 1057–8
— of lymph-glands, i, 320; ii, 633
— microscopic structure of, i, 307
— obsolescence of, i, 1121
— of pericardium, i, 320, 942; ii, 93
— of peritoneum, i, 94; ii, 318
— its relation to phthisis, i, 309, 1089
— of pleura, i, 320, 1035
— of spinal cord, i, 450
— of testis, ii, 547
— in rickets, ii, 742
Tuberculin, treatment by inoculation with it, ii, 913
Tuberculosis, i, 305
— bovine, *see* Perlsucht
— a sequela of enteric fever, i, 149
— its diagnosis from enteric fever, i, 152
— forms of, i, 319
— acute miliary, of the lungs, i, 1114
— verrucosa cutis, ii, 914
Tubular breathing, i, 934
Tumours, i, 70–97
— adenoma, i, 89
— angioma, i, 77
— cerebral, i, 597, 624

Tumours, carcinoma, i, 89
— cheloid, ii, 921
— cholesteatoma, i, 600; ii, 846
— classification of, i, 76
— of cord, i, 450
— cylindroma, i, 92
— enchondroma, i, 78
— epithelioma, i, 89
— fibroma, i, 77
— — molluscum, ii, 925
— glioma, i, 78
— — of brain, i, 600
— — of spinal cord, i, 451
— granuloma, i, 88
— of kidney, ii, 551
— of larynx, i, 909
— of liver, ii, 884
— of lung, ii, 121
— lipoma, i, 78
— lymphadenoma, i, 87
— lymphangioma, ii, 928
— lymphoma, i, 87
— lympho-sarcoma, i, 88
— malignant osteoid, i, 78
— of mediastinum, ii, 119
— melanoma of meninges, i, 600
— melanotic sarcoma, i, 82
— myoma, i, 77; ii, 926
— myxoma, i, 600; ii, 79
— neuroma, i, 77; ii, 926
— osteoma, i, 78
— papilloma, i, 89
— psammoma, i, 599
— sarcoma, i, 80, 599
— of the skin, ii, 925
— of spinal cord, i, 473
— of spinal meninges, i, 473
— tuberculous, of brain, i, 597
— — of spinal cord, i, 450
— vascular, ii, 927
— villous, of bladder, i, 89; ii, 520
— xanthoma, ii, 893, *foot-note*
Turpentine causing strangury, ii, 521
— in treatment of purpura, ii, 663
Tyloma, ii, 883
Tylosis linguæ, ii, 881
Tympanites in hysteria, i, 808
— in peritonitis, ii, 307
— in typhoid fever, i, 140
Tympanitic percussion-note, i, 930, 931
Typhlitis, ii, 220
— diagnosis from enteric fever, i, 154
— — from obstruction, ii, 269
Typhoid fever, *see* Enteric fever
— state in scarlatina, i, 190
— in typhus, i, 105
Typho-malarial fever, i, 329
Typhus, i, 103 *et seq.*
— siderans, i, 111
Typus inversus, i, 1116
Tyrosin in urine, ii, 377, 380

U.

Ulcer, ii, 758
— crateriform, ii, 931
Ulcer of duodenum, ii, 180, 185
— of intestine in dysentery, ii, 241
— — in enteric fever, i, 155
— of intestine in erysipelas, i, 253
— lupous, ii, 903
— of mouth, ii, 178
— of œsophagus, ii, 148
— of palate, ii, 132
— rodent, i, 92; ii, 931
— of stomach, ii, 178
— tertiary, ii, 900, 905
— of tongue in pertussis, i, 1128
— — in syphilis, i, 289; ii, 899
Ulceration of the colon, ii, 241
Ulcerative endocarditis, ii, 56
Ulcers, tubercular, ii, 914
Ulnar paralysis, i, 398
Unconsciousness in apoplexy, i, 578
— in hysteria, i, 809
Uræmia, acute, ii, 468
— in *Bright's* disease, ii, 468
— in cholera, i, 282
— chronic, ii, 470
— diagnosis from apoplexy, i, 587
— — from meningitis, i, 645
— theory of, ii, 471
Urate or lithate of soda in gout, ii, 675
Urates or lithates, amorphous, ii, 417
— crystalline, ii, 416
Urea, excretion of, in fever, i, 89
— — in diabetes, ii, 571
— in blood of relapsing fever, i, 129
— in urine of acute yellow atrophy, ii, 385
— — of cirrhosis of kidneys, ii, 497
— — of consecutive *Bright's* disease, ii, 503
— — of diabetes insipidus, ii, 409
— — of diabetes mellitus, ii, 571
— — of parenchymatous nephritis, ii, 480
Ureter obstructed by calculus, ii, 525, 530
— — by malformation, ii, 531
— — by peritoneal band, ii, 531
— — by rachitic pelvis, ii, 530
— — by traumatic stricture, ii, 531
Uric-acid as a calculus, ii, 512
— in diabetes, ii, 571
— estimation of, ii, 419
— in gout, ii, 673
— theory of its formation, ii, 420
— as a deposit in urine, ii, 416
Uridrosis, ii, 857
Urinary calculi, ii, 512
— deposits, ii, 416
— pigments, ii, 414
— organs, diseases of, ii, 407–562
Urine—
— in acute alcoholism, i, 795
— in acute yellow atrophy of liver, ii, 380
— in ague, i, 324, 325, 327
— ammoniacal, in paraplegia, i, 437
— in grave anæmia, ii, 644
— bilious, ii, 338
— bloody, ii, 517; *see also* Hæmaturia
— in cholera, i, 231
— density of, ii, 407
— — in chorea, i, 730
— — in m. *Brightii*, ii, 480, 490, 497, 503
— — in diabetes mellitus, ii, 569

Urine, density of, in hysteria, i, 809
— — in obstructive suppression, ii, 524
— in diabetes insipidus, ii, 408
— in diabetes mellitus, ii, 567
— in diphtheria, i, 269
— in enteric fever, i, 140
— in epidemic meningitis, i, 656
— in erysipelas, i, 252
— in glanders, i, 361
— in jaundice, ii, 338
— in melanotic sarcoma, i, 83
— in paraplegia, i, 437 *et seq.*
— in pneumonia, i, 998
— in acute pulmonary tuberculosis, i, 1116
— quantity of, ii, 407
— — in *Bright's* disease, ii, 480, 480, 497
— — in diabetes insipidus, ii, 408
— — in diabetes mellitus, ii, 569
— — in intestinal obstruction, ii, 268
— reaction of, ii, 412
— retention of—
— — in hemiparaplegia, i, 469
— — in hysteria, i, 805
— — in paraplegia, i, 435
— in rheumatism, ii, 689
— in scurvy, ii, 658
— specific gravity of, *see* density
— suppression of, in cholera, i, 229, 231, 235
— — in acute nephritis, ii, 480
— — in hysteria, ii, 524
— — in acute intestinal obstruction, ii, 268
— — non-obstructive, ii, 524
— — obstructive, ii, 524
— — in parenchymatous nephritis, ii, 480
— in suppurative nephritis, ii, 539
— in tubercular disease of kidney, ii, 547
— tests for alcohol in, i, 586, *foot-note*
— — albumen in, ii, 445
— — biliary acids in, ii, 339
— — biliary pigment in, ii, 338
— — blood in, 518
— — glycose in, ii, 564
— — tyrosin in, ii, 377
— in variola, i, 202
— in yellow fever, i, 340
Urobilin, ii, 414
Uroerythrin, ii, 415
Urostealith, ii, 514
Urticaria, ii, 830
— pigmentosa, ii, 832
Uterine affections in hysteria, i, 810
Uterus in heart disease, ii, 60

V.

Vaccination, i, 212
Vacuolation of brain, i, 685
Valvular disease of the heart, ii, 53
Varicella, i, 215
Varicella-prurigo, i, 218
Varicose veins producing eczema, ii, 768
Variola, i, 199 *et seq.*; *see also* Smallpox
— hæmorrhagica pustulosa, i, 209
— nigra, i, 202
— sine variolis, i, 203
Variolois, i, 206
Venesection, *see* Blood-letting
Ventilation, defective, a cause of phthisis, i, 1085
Veratrum viride in treatment of cardiac hypertrophy, ii, 44
Verruca, i, 883
— senilis, ii, 884
Verrugas, ii, 922
Vertebræ, cancer of, i, 472
— caries of, i, 471
— erosion by aneurysm and hydatid, i, 473
Vertigo, i, 767
— from cerebral tumour, i, 604
Vesicles of the skin, ii, 756
Vesicular murmur, i, 952
Vibex, ii, 758
Vibrio of cholera, i, 238
Vicarious epistaxis, i, 1132
— hæmoptysis, i, 1076
Villous tumour of bladder, i, 89
Vis medicatrix naturæ, i, 2
Vitiligo, ii, 932
Vitiligoidea, ii, 893; *see also* Xanthelasma
Vocal fremitus, i, 938
— resonance, i, 938
Voice, functional disorders of, i, 875
— as affected by obstruction of nares, ii, 145
Volvulus, ii, 171, 259
Vomica in liver, ii, 392
— in lung, i, 1058–9, 1061
Vomit, black, i, 339
— "coffee-ground," ii, 194
Vomiting, ii, 171
— in *Addison's* disease, ii, 597
— in *Bright's* disease, ii, 498
— from cerebral abscess, i, 629
— from cerebral tumour, i, 605
— in cholera, i, 228
— from gall-stone, ii, 351
— from gastric cancer, ii, 194
— — catarrh, ii, 152
— — ulcer, ii, 178
— in hydrocephalus, i, 668
— from invagination of intestine, ii, 248
— in meningitis, i, 640
— in migraine, i, 777
— from obstruction of intestine, ii, 262
— in peritonitis, ii, 307
— in phthisis, i, 1064; ii, 172
— from renal calculus, ii, 522
— from salicylates, ii, 707
— in scarlatina, i, 187
— in smallpox, i, 201, 208
— stercoraceous, ii, 262
— in uræmia, ii, 470
— in whooping-cough, i, 1137
— in yellow fever, i, 340
Vox choleraica, i, 229

W.

Warts, ii, 884
Washing out stomach, ii, 201
Wasting palsy, i, 497
Water in spread of ague, i, 334
— — of cholera, i, 239
— — of enteric fever, i, 162
— its effect in dermatitis, ii, 773

Watering-places, *see* Baths
Waxy liver, ii, 391
Weak-mindedness, i, 839
Weber's nasal douche, i, 274
Weil's disease, ii, 356
Werlhoff's disease, *see* Purpura
Wet packing, i, 172
Wheal, ii, 758
Whooping-cough, i, 1136
Wolff's treatment of writer's cramp, i, 707
Woolsorters' disease, i, 353
Worms, intestinal, ii, 276–304
Wrist-drop, i, 519
Writers' cramp, i, 702
Wryneck, i, 695

X.

Xanthelasma, ii, 893
Xanthelasmoidea, ii, 832
Xanthine, ii, 513
Xanthoma, ii, 893
— diabeticorum, ii, 895
Xerodermia (of *Wilson*), ii, 845, 886
— pigmentosa maligna (of *Kaposi*), ii, 929
Xerostomia, ii, 135

Y.

Yaws, ii, 922
Yeast plant, i, 14
Yellow fever, i, 339 *et seq.*

Z.

Zinc in treatment of epilepsy, i, 767
— poisoning by, i, 523
— in treatment of chorea, i, 745
Zona, ii, 936
Zoogloea, i, 14
Zymotic diseases, i, 12

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

$14 [illegible]

HC 32L1 D

6.G.1891.1
Text-book of the principles and1891
Countway Library AGQ7401
3 2044 045 057 023